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MEDICAL

LECTURES AND ESSAYS

BY

GEORGE JOHNSON, M.D., F.R.C.P., F.R.S.

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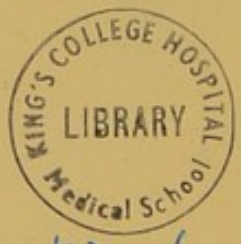

Presented by
Dr. Willoughby Lyle.

TO

SIR JOSEPH LISTER, BART.

M.B. LOND.; M.D. (HONORIS CAUSA) DUBLIN; LL.D. EDIN., GLASG., AND CANTAB.; D.C.L. OXON.; F.R.S. LOND. AND EDIN.; PROFESSOR OF CLINICAL SURGERY IN KING'S COLLEGE, LONDON; SURGEON EXTRAORDINARY TO THE QUEEN; ETC., ETC.,

WHO BY THE INTRODUCTION OF THE ANTISEPTIC PRINCIPLE AND METHODS INTO THE PRACTICE OF SURGERY HAS CONFERRED AN INCALCULABLE BENEFIT UPON MANKIND,

THIS VOLUME IS DEDICATED BY HIS FRIEND AND COLLEAGUE,

THE AUTHOR

PREFACE.

This selection of medical lectures and essays, while containing some recently written chapters and sections, is, for the most part, a reprint of papers which have been published in various forms and at different times, during the last thirty years or more. In bringing together and carefully revising some of my published writings relating to subjects which I have been led to investigate with more than ordinary care and diligence, my main object has been to communicate to the profession my latest and most matured opinions on such interesting and much debated questions as, for instance, the pathology and treatment of cholera and of the various forms of Bright's disease of the kidneys, the relation of membranous croup to diphtheria, the proximate cause of epileptiform convulsions, &c.

I am not without the hope of convincing every unprejudiced reader that the key to the solution of some of the most important pathological problems is to be found in a correct appreciation of the power possessed by the muscular-walled arterioles to regulate and, under certain conditions, to entirely arrest the circulation of the blood. That a disregard or misinterpretation of the now well-established physiological functions of these powerful agents in controlling the movement and distribution of the blood has been the source of grave pathological errors has, it is believed, been conclusively proved in the following pages.

My endeavour to demonstrate the correlation of various

pathological phenomena has led to the repetition of some statements of fact and doctrine—a repetition which is neither undesigned nor, it is hoped, without practical advantage.

I have to express my especial obligation to two friends who have rendered me much assistance in the preparation of this work. To my colleague Dr. Tirard I am indebted, not only for the labour of reading and correcting the proof-sheets, but also for many valuable suggestions; and another friend, who prefers to remain anonymous, has done me and my readers the great service of preparing the very full and complete index.

Since two of the authors whom I have had frequent occasion to quote have received from Her Majesty the well-deserved honour of knighthood while these pages were passing through the press, it may be well, though perhaps scarcely necessary, to mention that Dr. Garrod and Mr. John Simon, who are quoted in the earlier chapters, are identical with Sir Alfred B. Garrod and Sir John Simon, whose names appear later.

11 Savile Row: *September*, 1887.

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CHAPTER I.

INTRODUCTORY ADDRESS ON MEDICAL WORK AND MEDICAL DUTY.[1]

That my colleagues should, for the second time within a period of six years, have assigned to me the task of opening the medical session by distributing the prizes and delivering an address, is, I feel, to confer a great honour, and also to impose a grave responsibility—a responsibility which I should not have sought, but which I felt that I should not have been justified in declining.

It is probable that most men who have lived beyond middle age have sometimes indulged the wish that they could begin life again with the aid and guidance of such experience as they have acquired during their past career. The wish is obviously vain, and impossible of fulfilment; but it may be permitted, and, on such an occasion as this, it may perhaps be expected, that one who has reached the autumn of life should endeavour to draw from his stores of knowledge and experience some materials which may be of service to those who are yet in the spring time of existence. And that is the object which I shall endeavour to keep steadily in view, while I address myself to those who are just commencing, or who have already made some progress in, the study of subjects connected with the science and practice of medicine and surgery.

Let me beg of you not to suppose that I assume to be a living exemplification of the precepts which I am about to

[1] Delivered, after the distribution of prizes, at the opening of the winter session of King's College Medical School, October 1, 1886.

submit to you. Rather I desire to perform the humble but useful part of a whetstone, in accordance with the familiar passage: 'Fungar vice cotis, acutum reddere quæ ferrum valet, exsors ipsa secandi;' which I will venture to paraphrase by saying that, while making no claim to special acuteness myself, I desire, if it may be, to contribute to the sharpening of your intellectual and moral faculties.

My object is to offer you some advice, which may be of use to you during your student career, and also to make some suggestions for your guidance when, hereafter, you assume the duties and responsibilities of medical and surgical practice.

You will scarcely need to be told that very weighty, though, at present, unforeseen and incalculable results, to yourselves and to others, depend upon the manner in which you spend the four or five years during which you have to qualify yourselves for the practice of your profession. If, unhappily, any of you should neglect the opportunities which will now be afforded you for laying a solid basis upon which to erect the superstructure of professional knowledge and skill, it is scarcely possible that, by any future effort, you will be able to retrieve the lost time.

In pursuing your studies, you will do well to follow exactly the course prescribed for you by authority. You will find hereafter, if you do not already see, that there is good reason for the order in which the various subjects are brought before you.

The three great subjects to which your attention will first be directed are anatomy, physiology, and chemistry; and, unless you have mistaken your vocation, you will find in these studies an abundant source of interest. It scarcely need be insisted upon, that a knowledge of the structure and functions of the human body is an essential foundation for the practice of medicine and surgery.

It may not appear so obvious, but it is not less true, that some knowledge of chemistry is absolutely necessary for every practitioner. Apart altogether from its practical utility, a diligent study of the facts and laws of chemistry will prove an invaluable means of mental training and discipline. Observation, memory, and the reasoning faculty are each and all

exercised and strengthened by a thorough study of that delightful science. The subject is so extensive that a lifetime scarcely suffices for the thorough mastery of all its details and principles. But, without aspiring to be profound chemists, it will be your duty to acquire such a theoretical and practical knowledge of the subject as will be required in the daily practice of your calling. A man ignorant of chemistry cannot write a prescription without the risk of combining ingredients which are incompatible, and so bringing about most unexpected and undesirable results. A knowledge of chemistry is essential for understanding the operation, and therefore for the successful administration, of antidotes for poisons. It is equally essential for the testing of the various secretions, by which the most valuable information as to the presence and progress of disease is obtained.

The time will come when circumstances or your own deliberate choice will decide for each of you what department of practice you shall adopt; whether you will become consulting physicians or surgeons or so-called general practitioners, or whether you will select some such special department of practice as obstetrics, lunacy, or ophthalmic, aural, or dental surgery. Now, I desire to impress upon you that the only sure and safe foundation for specialism is a thorough knowledge of disease in general. It should be said with truth of the specialist, not that he knows less of general disease, but that he has more knowledge of the nature and treatment of some special class of diseases. Let me now give you some proofs and illustrations of this statement.

There are few, if any, special departments of practice which have so indisputable a *raison d'être* as ophthalmic surgery, for the successful practice of which a special manual dexterity, and constant practice in the performance of delicate operations, are essential. But again, there are few diseases which afford so many, and such striking, illustrations of the close relationship between various tissues and organs, and the dependence of apparently local structural changes upon remote and often general constitutional conditions, as those which affect the eye. Thus, in one case, defect of vision may, by the aid of the ophthalmoscope, be seen to be caused by a degeneration of the

retina, the result of Bright's disease of the kidney; in another case, blindness may be seen to result from obstruction of a retinal artery by a portion of blood-clot which has been detached from a diseased valve of the heart; while, in yet another, loss of vision may be traced to degenerative changes in the optic nerve, consequent on the growth of a tumour in the brain. Now, from these facts, it is manifest that an ophthalmic surgeon, who had no knowledge of diseases of the kidney and the heart and the brain, would often fail to correctly interpret the ocular defects with which he has to deal; while, on the other hand, a physician who undertakes the study and treatment of renal or cardiac or cerebral disease, without having acquired the power of inspecting the interior of the eye by the aid of the ophthalmoscope, is at a manifest disadvantage.

The same principle, namely, the dependence of apparently local disease upon remote pathological changes, may be illustrated by affections of the larynx. The use of that simple instrument, the laryngoscope, enables us to demonstrate that while, in one case, loss or impairment of voice is caused by structural change within the larynx, in another case, the vocal defect is a result of an aneurysm or other tumour in the neck or within the chest, pressing upon one or other of the nerves which supply the organ of voice; while, in yet another class of cases, the laryngeal trouble is traceable to disease within the brain or spinal cord. Obviously, then, a mere laryngoscopist, that is, one whose vision is limited to the larynx, would be very incompetent to deal successfully with the diseases of that organ.

My object in referring to the facts which I have here briefly set forth has been twofold. First, I wish to impress upon you, that between the various organs and functions of the body there exist, both in health and in disease, a correlation, and, to use a modern phrase, a solidarity, the recognition of which is of the highest practical importance. There is a strictly scientific truth in the familiar Apostolic doctrine, 'whether one member suffer, all the members suffer with it.'

The universe is governed by all-pervading immutable laws.

As the astronomers, relying upon the constancy of the laws of gravitation and of planetary motion, have discovered a previously unknown planet by observing the perturbed orbit of one before known; as the biologists, in reliance upon the constancy of organic types, have been enabled, from a fragment of a skeleton, or from a tooth, to reconstruct the whole framework of an extinct reptile, bird or mammal, so the pathologist and the practising physician, familiar with the laws of physiology, which, although modified, are not suspended by disease, have learnt that a disease commencing in one organ may involve, as a necessary consequence, widespread structural changes in other parts.

For a man ignorant of general pathology to claim to have special knowledge and skill in the treatment of some particular class of diseases would be as baseless an assumption as that of a would-be astronomer who, while ignorant of the elementary fact that the sun is the centre of our planetary system, pretended to have acquired a special knowledge of the orbit and phases of the moon, and the flux and reflux of the tides. Such a pretender would be rightly looked upon as the type and embodiment of lunacy.

It should be clearly understood that the main reason and justification for certain forms of specialism consist in the convenience, and even the need, of a division of labour, and not in the possibility of separating the body into different parts, and then making one of them the exclusive object of study and practice.

The second object which I have had in view, has been to insist upon the value of all physical means of diagnosis, and especially of two of the most recently introduced, the ophthalmoscope and the laryngoscope. No student, whatever his future line of practice may be, can be considered to have completed his medical training until he has learnt to use these simple but invaluable instruments, which have, in a very real and literal sense, shed an entirely new light upon a large and important class of diseases, and by the aid of which our knowledge of many facts tending to establish the correlation and interdependence of pathological conditions has been greatly extended.

I exhort you earnestly to avail yourselves to the utmost of the special opportunities which will be afforded you for obtaining a practical knowledge of these and other physical methods of diagnosis. And, in connection with this subject, I venture to protest against the rapid increase of small and uncalled-for special hospitals. Most of these institutions, whatever personal advantage they may bring to those who promote them, are certainly not, on the whole, beneficial to the public at large. There are two ways in which their existence is a public detriment.

1. They divert funds from the general hospitals, most of which are in sore need of more liberal support.

2. By attracting special classes of patients, they lessen the materials for teaching which, in all the hospitals connected with medical schools, are utilised for the practical instruction of their students, and so obviously for the public benefit.

If there should be any amongst you who feel a painful shudder on first entering the dissecting room or the operating theatre, you may be assured that, without any diminution of that reverence for the dead, or that sympathy with suffering, the loss of which would be indeed deplorable, you will soon cease to be troubled by those distressing emotions, the continuance of which, in their original intensity, would be a serious bar to your usefulness. The explanation of the fact that while painful passive impressions grow weaker by being repeated, the practical habits which they are designed to promote are formed and strengthened by repeated acts, is admirably given by Bishop Butler in the fifth chapter of the 'Analogy.' The whole passage is too long to quote, but the substance of it is the following: 'Perception of distress in others is a natural excitement, passively to pity, and actively to relieve it; but let a man set himself to attend to, inquire out, and relieve distressed persons, and he cannot but become less and less sensibly affected by the various miseries of life with which he must become acquainted; when yet, at the same time, benevolence, considered not as a passion but as a practical principle of action, will strengthen; and whilst he passively compassionates the distressed less, he will acquire a greater aptitude actively to assist and befriend them.'

Herein lies the explanation of the fact that a surgeon, while retaining all his active kindliness of heart, becomes a bold and skilful operator, never losing his presence of mind, even in the most trying circumstances. A conspicuous example of the combination of these high qualities was afforded by the great surgeon who was the friend and teacher of many generations of students in this college. I scarcely need say that I refer to the late Sir William Fergusson.

Lord Tennyson, in a poem entitled 'In the Children's Hospital,' says of a surgeon, whom he introduces into the narrative, 'He was happier using the knife than in trying to save the limb.' I venture to express my conviction that the Laureate never met with such a surgeon, and it is to be regretted that a great poet who has written so much to delight and instruct mankind should have given publicity to a fictitious character, the idea of which must surely have had its origin in a troubled dream. My experience of surgical practice differs essentially from that of the poet. All the surgeons with whom I have the honour and the happiness to be acquainted, whatever may be their political opinions, are, without exception, pre-eminently conservative in the practice of their profession; and, although they often have reason to be proud of the radical cures which they accomplish, they are staunch unionists. To bring about and to maintain union is always the first intention of the surgeon; and when, in an extreme case, he is reluctantly compelled to have recourse to dismemberment, he acknowledges with regret that the resources of civilisation and of surgery have failed to accomplish their primary aim and object.

When you come to the study of disease, you will learn that one of the most important duties of the practitioner is the correct interpretation of symptoms, the object being to get behind the symptoms, so as to ascertain their cause. And this great subject has a twofold aspect: first, we endeavour to ascertain the seat and nature of the functional derangements, or the structural changes within the body which have given rise to certain symptoms, such symptoms as, for instance, pains or other perverted sensations, loss or impairment of motor power, difficult breathing, cough, palpitation, dropsical

swelling, &c. Secondly, a most important subject of inquiry is what, in the habits or the surroundings of the patient, has caused his illness. In illustration of this, I need only refer to the numerous instances in which contaminated water or milk, or air-pollution by defective drainage or overcrowding, has been the cause of serious and often widespread disease.

During about ten days in the month of September 1854, 600 people died of cholera within a very limited district in the neighbourhood of Golden Square; and this terrible outbreak was traced to the contaminated water of a well in Broad Street, to which the whole neighbourhood was in the habit of resorting.

In the prompt recognition and demonstration of such like sources of disease our profession is daily conferring incalculable benefits upon mankind. Who shall estimate the amount of suffering and of mortality daily and hourly prevented by the now general use of antiseptics in the practice of surgery?

But there are yet other fruitful causes of disease. Here I quote a sentence from Sir Thomas Watson. 'It is ours to know in how many instances, forming, indeed, a vast majority of the whole, bodily suffering and sickness are the natural fruits of evil courses, of the sins of our fathers, of our own unbridled passions, of the malevolent spirit of others.'

I have before insisted on the value and importance of physical methods of diagnosis; and now let me impress upon you that as man is something more than a cunningly devised machine, so he is the subject of disorders which escape all physical means of research. If you cultivate that power of sympathy with which all are more or less endowed, and the importance of which was so ably and eloquently insisted upon by the Lord Bishop of London in the address which he delivered here last year, you will learn that there are mental pains and disorders which exceed in their distressing results any merely bodily ailment. The student of physiognomy, as every practitioner of medicine ought to be, will often see in the anxious and troubled expression of the patient the mental source of his suffering, and so will not seldom find the way to afford relief.

The question will often arise in substance, though not in words, identical with that addressed to the doctor by Macbeth :

> Canst thou not minister to a mind diseased ;
> Pluck from the memory a rooted sorrow ;
> Raze out the written troubles of the brain ;
> And, with some sweet, oblivious antidote,
> Cleanse the stuff'd bosom of that perilous stuff
> Which weighs upon the heart ?

It is not surprising that the unsatisfactory reply of the doctor should have driven the indignant inquirer to speak disdainfully of physic.

It may be that in some cases, as the doctor said of Lady Macbeth, the patient 'more needs the divine than the physician ;' but judicious medical treatment will often do much for the relief of this class of sufferers; and a method of treatment which has often proved successful is indicated by the physician who was ministering to the troubled mind of King Lear :

> Our foster nurse of nature is repose,
> The which he lacks ; that to provoke in him
> Are many simples operative, whose power
> Will close the eye of anguish.

The successful result of the physician's treatment is shown in a later scene. The old king had slept so soundly that they had been enabled to change his garments without awaking him. When at length he is awakened in the presence of his daughter Cordelia, although his words and manner indicate the doubt and confusion of one just aroused from a terrible dream, yet the physician's encouraging language is fully justified :

> Be comforted, good madam, the great rage
> You see is killed in him.

There are few results of medical treatment more entirely satisfactory than the calm which often comes over a delirious brain under the timely operation of an opiate ; and one of the most important practical lessons which every medical man has to learn is the true use, while he avoids the abuse, of narcotics.

You will find hereafter, when you enter upon the practice

of your profession, that, under the heading of Medical Ethics, many rules have been laid down for the guidance of the medical man in his conduct towards his patients and his brother practitioners.[1] If we could all learn and earnestly endeavour to act upon the comprehensive maxim to love our neighbour as ourselves, to do to others as we would wish others in like circumstances to do to us, few additional rules would be required for our guidance. The very nature of our studies, and of our daily work, should teach us the duty of absolute truthfulness in all our dealings both with our professional brethren and with our patients.

Upon this point I may quote the advice of the sagacious though rather garrulous old gentleman, Polonius :

> To thine own self be true,
> And it must follow as the night the day,
> Thou canst not then be false to any man.

Perhaps it may not be without advantage if I forewarn you that to proclaim a truth is not seldom attended with unpleasant consequences. Thus, after due consideration, you deem it your painful duty to announce to a patient or, it may be, to some member of his family, that he has an incurable disease. The result often is that you at once lose your patient, not by his death, but by his going to one consultant after another, in the hope that he will find some one to reverse your verdict, and to give a more hopeful opinion. The late Dr. P. M. Latham, referring to cases of this kind, said : 'He consults an infinite number of medical men, and it is remarkable that he gets no comfort or satisfaction from those who understand his disease the best, and the greatest comfort and satisfaction from those who understand nothing about it.'

Before giving utterance to an opinion as to the existence of an incurable disease, it is obviously the practitioner's duty to make sure of the evidence upon which his opinion is based. A hopeless prognosis deduced from insufficient evidence inflicts unnecessary pain upon others, and brings discredit upon the erring practitioner. But, having satisfied himself of the

[1] Mr. Jukes Styrap has published an admirable *Code of Medical Ethics* with the very appropriate motto 'Bear and Forbear' (J. and A. Churchill).

existence of a disease which is beyond human aid, the possibility of losing his patient in the manner which I have before indicated, should not deter the medical attendant from the timely intimation of a state of things with which, for various reasons, it may be of the highest importance that the patient and his family should be made acquainted.

Again, it may be that, after the expenditure of much labour and time, you discover some new fact, or you arrive at some conclusion from previously known facts which you believe to be both new and true, and of sufficient importance to be made public. Now, you will probably be disappointed if you expect that the publication of your discovery will be received with general acclamation. To borrow a sentence or two from 'The Autocrat of the Breakfast Table,' 'Every real thought on every real subject knocks the wind out of somebody or other. As soon as his breath comes back he very probably begins to expend it in hard words. These are the best evidence a man can have that he has said something it was time to say.'

Although I have given you this warning, I trust that neither the anticipation nor the actual experience of factious opposition will hereafter deter any of you from earnestly contending for that which you believe to be true. Without doubt, the general reception of truth is often retarded by senseless and selfish opposition and prejudice; but truth is all-powerful, and in the end it will prevail.

It is sometimes painful to witness local and temporary rebellion against truths which all sane and sensible men have long accepted as indisputable. As an instance of this, I may refer to the notorious town of Leicester, a large proportion of whose inhabitants refuse to obey the law which wisely decrees that every child shall have the benefit of the proved protection of vaccination. Now this is a subject respecting which it is quite safe to prophesy. It is as certain as that the sun will rise to-morrow that at no distant period small-pox will invade the town of Leicester, and that the result will be such a massacre of the innocents — innocent victims of parental ignorance and prejudice—as will probably suffice to carry conviction to that eccentric member of the House of Commons

who lately had the assurance to ask for the appointment of a Committee to inquire into the practice of vaccination.

On the subject of consultations, I venture to offer a few words of advice. Some practitioners appear to think that the suggestion of a consultation implies a mistrust of their knowledge and skill, which they resent as a personal affront. Now this is surely a very shortsighted and selfish view of the matter, and one which takes no account of the natural anxiety of the patient's family and friends. The most skilful and experienced practitioners, clearly perceiving the limits of their own resources, and sympathising with the anxieties and fears of the patient's friends as to the result of a dangerous illness, are, as a rule, the most willing and anxious to agree to, and indeed are often the first to suggest, a consultation. On the other hand, the medical attendant has a right to expect that his feelings and susceptibilities shall meet with due consideration from the patient and his friends. When they desire a consultation, it is their duty at once to make their wish known to the gentleman in attendance. The practitioner has good reason for displeasure if, without adequate reason, he is taken by surprise, and finds that a consultant has been sprung upon him without notice. It scarcely needs to be insisted on that a practitioner who declines to join in a consultation when requested to do so by his patient's family incurs a grave responsibility.

It goes without saying that the welfare of the patient is the main and primary object of every consultation; that being duly safeguarded, care should be taken that the credit and reputation of the medical attendant suffer no detriment.

You will not, I trust, fall into the error of supposing that when you have completed the usual course of lectures and examinations, and have obtained your legal qualification to practise, your student days are at an end. The conscientious practitioner of medicine must continue to be a diligent student to the end of his active life. When a man is too old to learn he is too old to practise. The late Sir Thomas Watson, soon after the publication of the last edition of his lectures, when he had entered upon his eightieth year, was reproached by a

medical friend a few years his junior for having introduced into his work so many new doctrines, or as his friend expressed it, 'for having abandoned so many of his earlier principles.' The reply of Sir Thomas to this reproof was characteristic. He said, 'Although advanced in years, I hope I am not too old to learn.'

In these days of active research, continual additions are being made to our knowledge of subjects more or less directly bearing upon the science and practice of medicine. The science of Bacteriology is of comparatively recent origin, but it has already been productive of a copious literature, and has been fruitful of lively controversy. Amidst much that is as yet open to doubt and discussion, enough has been firmly established to entitle the workers in this new and difficult field of research to the gratitude of mankind. Although those who possess the requisite qualifications and opportunities for successfully conducting researches of this kind are comparatively few, on the other hand there are few who cannot avail themselves of the practical results arrived at by others.

As in general terms knowledge is power, so every addition to medical knowledge brings with it an increased power of doing good to our fellow-men, and the conviction of this fact should act as a continual stimulus to exertion.

That which Lord Bacon has eloquently said of all knowledge is in an especial manner and degree applicable to the knowledge which it will be the chief business of your lives to acquire, and to use for the benefit of mankind. 'It is not a couch whereupon to rest a searching and restless spirit, or a terrace for a wandering and variable mind to walk up and down with a fair prospect, or a tower of state for a proud mind to raise itself upon, or a fort or commanding ground for strife and contention, or a shop for profit or sale, but a rich storehouse for the glory of the Creator and the relief of man's estate.'

CHAPTER II.

THE PHYSIOLOGY OF THE CIRCULATION.[1]

The Structure of the Arteries, Arterioles, and Capillaries—The Vaso-Motor Nervous System—The Physiology of the Circulation—Influence of the Heart, the Large Elastic Arteries, and the Muscular Arterioles—The Phenomena of Asphyxia or Apnœa illustrated by Post Mortem Appearances and by Experiments—Nitrous Oxide Anæsthesia: the Symptoms and their Physiological Explanation.

MR. PRESIDENT AND GENTLEMEN,—In this course of Lumleian Lectures, which, by the favour of yourself, Sir, and the Censors, I am to have the honour to deliver, I propose to discuss certain questions relating to the structure of the minute blood-vessels and the forces concerned in carrying on and regulating the circulation of the blood. Upon this subject modern researches have thrown an entirely new light; and I shall endeavour to show that the increased knowledge of the physiology of the circulation which has been acquired within the last quarter of a century has rendered necessary a revision and correction of some pathological doctrines which had gained more or less general acceptance.

The chief anatomical discovery relating to the organs of circulation made during the period to which I refer was Henle's demonstration of the muscular elements in the middle coat of the arteries. John Hunter and others, it is true, had on theoretical grounds assumed that the middle coat of the arteries contains muscular tissue; but it was Henle[2] who first described the fusiform muscular elements encircling the arterial tube between the outer and the inner coats, and who

[1] This lecture was the first of the Lumleian Lectures 'On the Muscular Arterioles, their Structure and Function in Health and in certain Morbid States,' delivered at the Royal College of Physicians in 1877, and is now revised and reprinted from the *British Medical Journal*.

[2] *Wochenschrift für die gesammte Heilkunde*, 1840, No. 21, p. 329.

showed that these have the same characters as the unstriped tissue of involuntary muscle.

There are obvious structural differences, corresponding with important diversities of physiological function, between the large and the small arteries. The chief anatomical distinction between the large and the small arteries is to be found in their middle coat. The middle coat of the largest arteries is composed almost entirely of elastic tissue, with a very slight admixture of muscular fibres. As the arteries diminish in size, the proportion of muscular tissue increases, until, in the smallest arteries, the middle coat is composed entirely of muscular tissue. These smallest arteries are commonly designated 'muscular arterioles,' to distinguish them from the large elastic arteries. The muscular arterioles, varying in diameter from the one-hundredth to the one-three-thousandth of an inch, have their middle coat composed of muscular fibre-cells without the slightest admixture of connective or elastic tissue. The muscular fibre-cells which, when separated, are seen to be elongated and spindle-shaped, with an oblong nucleus in the centre, are arranged in a circular or spiral manner round the arteries, forming contractile muscular lamellæ. The circular muscular coat in arteries between about the one-hundredth and the one-three-hundredth of an inch in diameter possesses two or three layers of muscular fibres. In the smaller arteries, the muscular coat consists of only a single layer of fibres, whose elements become shorter and shorter until, in the smallest arteries approaching the capillaries, the muscular elements separate from each other and at length completely disappear.

The muscular coat has on its inner surface the tunica intima, and on its outer the tunica adventitia. The tunica intima consists of three layers: an inner endothelial layer, an outer elastic layer in contact with the muscular coat, and an intermediate layer of delicate connective tissue. The tunica adventitia consists of connective tissue and fine elastic fibres with elongated nuclei, having their long diameter parallel with the axis of the vessel. The tunica adventitia is generally as thick as, and often thicker than, the muscular coat; and it is readily made to swell up under the influence of certain reagents. My colleague Dr. Beale and other microscopic

observers have demonstrated the presence of minute nervous ganglia and extremely delicate nervous fibres ramifying upon the minute arteries and the capillaries.

During the last quarter of a century, the physiology of the vasomotor system and the relation between the nervous and the vascular apparatus have been the subject of laborious research by numerous and very able investigators; and the result has been a large addition to our positive knowledge of the forces which are concerned in regulating the movement of the blood through the minutest subdivisions of the vascular system. M. Vulpian[1] has given a very lucid and complete history of these investigations. An able summary of the physiology of the vaso-motor system appeared in the 'British and Foreign Medico-Chirurgical Review' for October 1876; and the whole subject of the vascular mechanism has been treated with great ability by Dr. Michael Foster in his 'Handbook of Physiology.'

We have already seen that in the year 1840 Henle had demonstrated the muscular tissue of the middle arterial coat. About the same time Stilling[2] was led to the conclusion that there are certain nerves which influence the movements of the blood-vessels. For these nerves he invented the term *vaso-motor*, and he looked upon them as analogous to the *musculo-motor* nerves. But the starting-point of our present positive knowledge of the vaso-motor nerves was the year 1851, when M. Claude Bernard published his first conclusive experiments.[3] In his first memoir, Bernard showed that after division of the cervical sympathetic, but more especially after removal of the superior cervical ganglion, in the horse, the dog, or the rabbit, there is an increased afflux of blood to the ear and the whole of that side of the face, and with this an elevation of temperature and an increased sensibility. In a second communication, made this time to l'Académie des Sciences,[4] he described in more detail the facts recorded in his first paper. It was not until towards

[1] *Leçons sur l'Appareil Vasomoteur*, Paris, 1875.

[2] *Recherches Pathologiques et Médico-Pratiques sur l'Irritation Spinale*, Leipzig, 1840.

[3] *Comptes Rendus de la Société de Biologie*, 1851, p. 163.

[4] *Comptes Rendus de l'Acad. des Sciences*, 29 Mars 1852.

the end of the year 1852 that Bernard published his explanation of the phenomena which he had discovered. Meanwhile, public attention having been directed to these researches, in the interval between the publication of Bernard's second and third memoirs, Dr. Brown-Séquard had published in America [1] the interesting results at which he had arrived. This able experimenter confirmed Bernard's observation of the dilatation of the blood-vessels and the elevation of temperature resulting from division of the cervical sympathetic. He then went on to show that the galvanic stimulus applied to the cut end of the peripheral portion of the nerve caused a constriction of the blood-vessels and a lowering of the temperature. He thus proved that the elevation of temperature resulting from division of the sympathetic is directly due to the increased afflux of blood consequent on paralysis of the arterioles. In Bernard's third memoir, published in November 1852,[2] he also records the observation that the increased blood-supply which results from the paralysing influence of dividing the sympathetic is at once arrested by galvanising the divided end of the nerve, when the parts which were previously red and congested become pale and comparatively bloodless.

Since this great field of research was opened up by Claude Bernard and Brown-Séquard, numerous experimenters have laboriously entered upon it, and the result has been the accumulation of many interesting facts and the construction of a tolerably consistent though not as yet an entirely complete theory of the vaso-motor system.

In a paper 'On the Minute Structure of Involuntary Muscular Fibre,' [3] Sir Joseph Lister demonstrated more clearly than had been previously done the beautiful spiral arrangement of the muscular elements surrounding the arterioles; while in another paper 'On the Parts of the Nervous System which regulate the Contractions of the Arteries,' [4] he described a most interesting series of observations and experiments,

[1] *Philadelphia Medical Examiner*, August 1852.
[2] *Comptes-Rendus de la Société de Biologie*, Nov. 1852, p. 168.
[3] *Transactions of the Royal Society of Edinburgh*, vol. xxi. part iv. 1857.
[4] *Philosophical Transactions*, part ii. for 1858.

which contributed greatly to the clear understanding of the functions of the arterioles.

Time would not permit me now, even if it were necessary or desirable, to enter into the minute details of this extensive subject. I need only refer to such ascertained facts and principles as have relation to some pathological phenomena which we shall presently have to discuss. The vaso-motor nerves may be said, in a general way, to belong to the great sympathetic ; but, by means of communicating branches, they are also connected with the spinal nerves and with the spinal cord. In fact, there is reason to believe that all the vaso-motor fibres are derived from the cerebro-spinal axis, from which they pass out chiefly by the anterior roots of the spinal nerves ; and that the chief centre of vaso-motor nerve action is the medulla oblongata, near the floor of the fourth ventricle. Injury to this part of the nervous centre or division of the cord in the upper cervical region, cutting off the communication between the centre above and the vaso-motor nerves, causes general relaxation of the arterioles and a fall of blood-pressure throughout the body. On the other hand, electrical stimulation of the centre excites general contraction of the arterioles and an increase of blood-pressure.

The nerves which, when divided, cause arterial paralysis, and when stimulated excite arterial contraction, have been designated *vaso-constrictor* nerves. There are other nerves having a different and, in some respects, an antagonistic function: these are designated *vaso-dilators*. Of this class of nerves, the *chorda tympani* is a conspicuous type.

The chorda tympani is a branch of the facial nerve, which joins the lingual branch of the fifth nerve, and is distributed to the tongue and the submaxillary gland. Bernard discovered that electrical stimulation of the peripheral end of the divided nerve causes great dilatation of the blood-vessels of the submaxillary gland, and a rapid and profuse secretion of saliva.

Many experiments of various kinds have proved that the vessels may be made to contract or to dilate by an influence conveyed through incident nerves to the centre, and thence reflected through other fibres to the arterioles. Thus when a

sensitive nerve, such as the fifth, or a mixed nerve like the sciatic, has its central end stimulated, a reflex contraction of the arterioles occurs throughout the body, and the blood-pressure rises. On the other hand, Ludwig and Cyon discovered that one branch of the pneumogastric, when its central end is stimulated, has a reflex influence on the vaso-motor nerves, which causes a general relaxation of the arterioles and a consequent fall of the blood-pressure. This nerve, therefore, is called *the depressor nerve*.

There is now a very general agreement amongst physiologists with respect to the influence which the heart, the large elastic arteries, and the muscular arterioles respectively exert upon the circulation. The force which propels the blood through the systemic arteries is derived entirely from the contraction of the muscular walls of the left ventricle of the heart. The elastic walls of the large arteries, distended by the injecting force of the ventricle, react upon and force the blood onwards during the diastole of the ventricle. This forcible resiliency in the walls of the arteries is as obviously derived from the muscular contraction of the heart as the elastic power of an archer's bow has its source in the contracting muscles of the arm which bends the bow. The resiliency of the arterial wall, reacting upon the blood during the diastole of the ventricle, gradually converts the interrupted jet of blood from the heart into a continuous current in the minute arteries and capillaries. The muscular arterioles, under the influence of the vaso-motor system of nerves, regulate the blood-supply to the various organs and tissues. The action of the muscular arterioles is, as I have ventured to suggest,[1] that of stopcocks. By the contraction of the muscular walls, their canals are narrowed, the blood-stream is in a corresponding degree lessened, and the pressure of blood in the larger arteries is increased. On the contrary, relaxation of the walls of the arterioles enlarges their canals, permits a fuller stream of blood to pass, and lowers the blood-pressure in the arterial trunks. The minute muscular arteries, therefore, through their stopcock action, exert a regulating but not a propelling influence upon the blood-current.

[1] *Medico-Chirurgical Transactions*, li. 60.

The influence of the heart, the larger elastic arteries, and the muscular arterioles respectively upon the circulation, may be demonstrated by the very simple apparatus which I have here.[1] A pump is made of a hollow india-rubber ball, with two orifices, to one of which is attached a tube six inches long, and to the other an elastic india-rubber tube about four feet long, at the distal end of which is attached a metallic stopcock. The central orifice of each tube is guarded by a bullet valve. The end of the short tube is dipped in a basin of water, while the elastic ball is alternately compressed and relaxed by the hand. The intermitting jet of water from the hollow ball, representing the heart, is gradually converted into a continuous stream by the tube acting thus like the large elastic arteries, and the size of the continuous jet from the metallic orifice is regulated by turning the stopcock. If, now, I substitute for the elastic tube one with rigid walls, the stream of water from the orifice of the stopcock is no longer continuous, but an interrupted pulsating jet; so, if the opening in the stopcock be large enough to allow the water to escape as fast as the pump drives it into the tube, the flow will be interrupted. This wide-open state of the stopcock represents a greatly dilated condition of the muscular arterioles, when the pulse may extend through the capillaries even into the veins. For the conversion of the intermitting jet from the pump into a continuous stream from the stopcock, it is requisite that the orifice in the latter should be so small as to allow the fluid to accumulate in and distend the elastic tube, the resiliency of which continues to drive on the fluid, while the pump, representing the heart, is dilating to receive a fresh supply.

It is evident then that, while the resiliency of the large arteries, which are mainly elastic but partly muscular, aids the heart in propelling the blood onwards towards the capillaries, the contraction of the arterioles, whose middle coat is entirely muscular, antagonises the heart and the larger arteries; and their stopcock action, under the guidance of the nervous system, regulates the blood-supply to the various

[1] This apparatus was designed by Dr. Rutherford (*Lancet*, Oct. 12, 1872).

tissues and organs in accordance with their physiological requirements.

There is no evidence of a *peristaltic* muscular contraction of the arteries, as some writers—amongst others, MM. Legros and Onimus—have supposed. Anyone who has carefully watched the circulation in the web of the frog's foot, or in other transparent parts of a living animal, must have observed that, so long as the circulation is active, the blood-stream in the terminal arterioles is as continuous and uniform as it is in the capillaries, and there is no appearance of an alternating contraction and relaxation of the arterioles.

The true capillaries have no muscular fibre in their walls, and there is reason to believe that they have no power of active contraction. They become distended and dilated when the muscular arterioles are relaxed, and they return to their original size when the arterioles contract and lessen the blood-stream; but this contraction of the capillaries is probably the result of simple elastic resiliency after distension, and not of an active vital contraction. The capillary obstruction which occurs during the progress of inflammation is of course quite different from a normal physiological impediment.

I now pass on to the consideration of some pathological phenomena which, while on the one hand they receive a more or less complete explanation by the aid of the physiological principles to which I have referred, on the other hand tend to confirm the generally received physiological doctrines relating to the circulation.

The phenomena of what is commonly called *asphyxia*—death, that is, by suffocation or suspension of the respiration—could not be completely and satisfactorily explained before the structure and functions of the muscular arterioles had been revealed. I propose now to devote some time to the consideration of these phenomena, and I shall afterwards endeavour to show that the disturbance of the circulation which results from the suspension of the respiration is strictly analogous to the hindrances and disorders of the circulation which occur in other abnormal states, and especially

in connection with choleraic collapse and certain forms of renal disease.

The immediate cause of death from suffocation is the arrest of the circulation through the lungs. The obvious and indisputable evidence of this is found in the fact that, when the chest is opened immediately after death, the right cavities of the heart and the large branches of the pulmonary artery are found to be greatly distended with blood, while the left cavities are flaccid and comparatively empty. This elementary fact in the pathology of *apnœa* [1] (a term which I use in preference to *asphyxia*, which literally means pulselessness) was first demonstrated by our own illustrious Harvey. Harvey says: 'I have several times opened the breast and pericardium of a man within two hours after his execution by hanging, and before the colour had totally left the face, and in presence of many witnesses have demonstrated the right auricle of the heart and the lungs distended with blood—the auricle in particular being of the size of a large man's fist, and so full of blood that it looked as if it would burst. This great distension, however, had disappeared the next day, the body having stiffened and become cold, and the blood having made its way through various channels.' [2]

Two circumstances may prevent the recognition of the distension of the right side of the heart—first, the inspection may be delayed until, as Harvey said, 'the blood has made its way through various channels;' secondly, the large veins may be cut before the heart is exposed, and as a result the distended right cavities rapidly empty themselves.

The great distension of the right cavities of the heart with the relative emptiness of the left cavities, so clearly and accurately described by Harvey, has sometimes been denied by recent writers on apnœa; it may, therefore, be well to

[1] Physiologists apply the term *apnœa* to the suspended respiration which results from hyperoxygenation of the blood. The term *asphyxia* as usually applied to death by suffocation is, as Sir Thomas Watson says (*Lectures*, vol. i. p. 69), peculiarly inappropriate. 'It might express any kind of death whatever; or, if applied to any particular mode of dying, it would seem to belong to death beginning at the heart.' The pulselessness of syncope is literally *asphyxia*, as is also the pulselessness of choleraic collapse.

[2] *Second Disquisition on the Circulation of the Blood*, Sydenham Society's Translation, p. 127.

give some additional evidence of this fundamental anatomical condition.

Dr. Massey of Nottingham has published the following report of the appearances found in the chest of a man four hours after his execution by hanging:[1]—'On removing the sternum and cartilages of the ribs, the lungs were not to be seen, but were found to occupy a very small space at the back part of the chest, resembling the contents of a fœtal thorax, the pericardial sac alone being seen. The colour of the lungs was of a darker hue than natural, especially at the bases. On cutting out the lungs, a quantity of black liquid flowed. The structure was natural, but there was loss of crepitancy, and but very little air was contained in them. The right auricle of the heart was gorged at the greatest state of distension with blood, and the inferior cava was in the like condition. On opening the auricle, a great quantity of black fluid blood gushed out. The right ventricle also contained a large amount of blood. The left auricle and ventricle were completely empty.'

In October 1867, a dog weighing fourteen pounds and a quarter was killed in my presence by a ligature on the trachea. The animal continued to struggle convulsively for five minutes. *As soon as these movements had ceased* the chest was opened. The pericardium was so filled and stretched by the distended heart that it was at first supposed that the pericardium had been opened so as to lay bare the heart. The right cavities of the heart were full and tense, the left comparatively empty and flaccid. In particular, the two auricles presented a marked contrast: the right auricle stood out in a globular form and had a tense and elastic feel like an india-rubber ball distended with air, while the left auricle was flaccid and its surface wrinkled. A ligature having been placed round the large vessels, the heart was removed and its cavities opened, when two ounces of blood gushed out of the distended right cavities, while two drachms and a half only flowed slowly from the left side. After division of the large vessels, twelve ounces of blood escaped into the cavity of the chest, chiefly from the venæ cavæ and the pulmonary artery. The lungs collapsed to an extreme degree; they were pale and non-crepitant.

[1] *Lancet*, November 9, 1867.

It will be seen that the condition of the heart's cavities, and in particular the great distension of the right auricle, as described by Harvey and by Dr. Massey in men executed by hanging, is identical with that which I observed in the dog killed by a ligature on the trachea.

The great distension of the right cavities of the heart, with comparative emptiness of the left, is very generally admitted, and believed to result from some impediment to the passage of the blood through the lungs, consequent on the suspension of respiration. The question then arises, What is the mechanism of the process by which the flow of blood through the lungs is impeded and finally arrested? It was formerly supposed that the arrest of the circulation through the lungs might be explained by the cessation of the respiratory movements. It is, of course, indisputable that the movements of the ribs greatly influence the amount of blood within the chest, and especially in the large veins. In inspiration, the blood is sucked into the venæ cavæ and the superficial veins in the neck may be seen to collapse; while, in expiration, the intrathoracic trunks are compressed, and the jugulars and other affluent veins are distended; but the hypothesis that the arrest of the blood-stream through the lungs is due mainly to the immobility of the chest is completely disproved by the fact that when an animal is made to breathe nitrogen gas unmixed with oxygen or nitrous oxide gas, although the movements of the chest continue as in ordinary respiration, the passage of the blood through the lungs is arrested as speedily and completely as when the chest is motionless. It is evident, then, that the arrest of the pulmonary circulation is in some way caused by the suspension of the chemical changes in the blood and the respired air which are affected by the inhalation of oxygen.

One great step towards the elucidation of this problem was made many years ago by Dr. John Reid.[1] He experi-

[1] See his paper, 'On the Order in which the Vital Actions are arrested in Asphyxia,' which was first published in 1841, and republished in his collected *Physiological, Anatomical, and Pathological Researches*, 1848. In this paper Dr. Reid gives a complete history of the attempts which had been made by previous observers to explain the phenomena of so-called asphyxia.

mented in the following manner upon dogs. A tube with a stopcock was placed in an opening in the trachea, and a mercurial dynamometer was introduced into the femoral artery for the purpose of measuring the blood-pressure within the arterial system. Dr. Reid expected to find that when air was excluded from the lungs by turning the stopcock, and when, consequently, unaërated black blood began to pass into the systemic arteries, there would be a steady decrease of the blood-pressure there. He found, however, to his great surprise, that for a period of about two minutes after the animal had ceased to struggle, the mercury stood higher in the dynamometer, and the arteries were more tense, than when the animal was breathing freely. After this high pressure had continued for about two minutes, it began to decline rapidly, in consequence of the increasing impediment to the flow of blood through the lungs.

Referring to the temporary increase of pressure in the systemic arteries, Dr. Reid says: 'This was so unlooked for, at first sight was so inexplicable and so much at variance with my preconceived notions on the subject, that I was strongly inclined to believe that there must be some source of fallacy; but, after repeating the experiment more than twenty times, and invariably with the same result, I was at last compelled to admit its accuracy. I then began to surmise that this arose from an impediment to the passage of the venous blood through the capillaries of the systemic circulation.' We shall presently return to this suggested explanation of the phenomena; meanwhile, it is to be observed that, in experiments performed as Dr. Reid performed his, the observation of the blood-pressure is liable to be more or less interfered with by the struggles of the animal, which, while they continue, have the effect of increasing the arterial pressure.

In some experiments afterwards performed by Mr. Erichsen,[1] the struggles were prevented by the animal being pithed; and, this disturbing element being thus removed, Mr. Erichsen obtained results which were entirely in accordance with those of Dr. Reid—namely, that with the suspension of the respiration and the consequent passage of black blood into the

[1] *Edinburgh Medical and Surgical Journal*, January 1845.

systemic arteries, there is, for a time, an increased blood-pressure within those arteries, the result of some resistance in the terminal vessels ; then, after a period of two or three minutes, there is a rapid decrease of pressure, in consequence of the impeded and finally arrested passage of the blood through the lungs.

But the most complete and entirely satisfactory experiments tending to throw light upon the phenomena of apnœa are those which have been performed upon dogs under the paralysing influence of curara. I am indebted to my friend and former colleague Dr. Rutherford, now the distinguished Professor of the Institutes of Medicine in the University of Edinburgh, for the opportunity of witnessing some experiments performed in 1873, the results of which I will endeavour as briefly as possible to describe. I may state at once that the results, although in some respects more complete and conclusive than those obtained by Dr. John Reid and Mr. Erichsen, are entirely in accordance with their observations.

Into the trachea of a dog a tube was introduced and connected with a bellows for the performance of artificial respiration. The voluntary muscles were then paralysed by the injection of curara, and the animal was kept alive by artificial respiration. The sternum and portions of the ribs were removed and the pericardium was opened, so as to expose the whole of the anterior surface of the heart. One common carotid artery was divided, and a dynamometer-tube connected with a kymograph was introduced into the proximal end. In making all these preparations, much time and labour and great skill were required. Artificial respiration was now suspended, and immediately the colour of the left auricle changed from crimson to purple, and the kymograph indicated a continuous increase of pressure in the systemic arteries. After the increase of pressure had continued for about a minute, the *left* cavities of the heart became much distended ; the auricle, in particular, became expanded into a tense globular ball with a smooth surface (fig. 1). In the next period, the pressure in the arteries began to fall, and, about the same time, the right cavities of the heart, which had hitherto remained of the normal size and form, began to

expand, while the distension of the left began rapidly to subside. Meanwhile, the right cavities became more and more

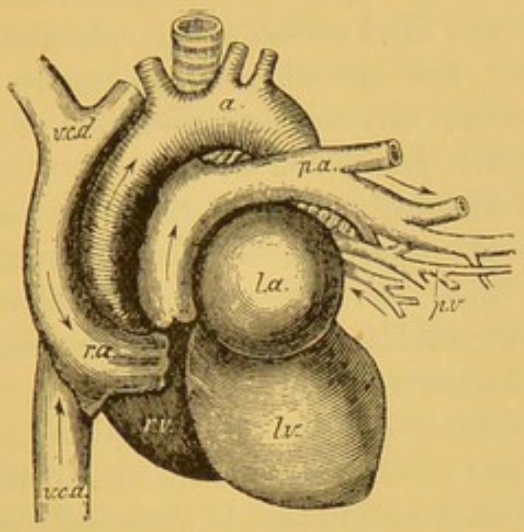

FIG. 1.—Represents the distension of the left cavities of the heart and aorta in the first stage of apnœa (asphyxia). *l.a.* left auricle. *l.v.* left ventricle. Both greatly distended, the former like a smooth india-rubber ball. *a.* aorta distended. *p.a.* pulmonary artery. *p.v.* pulmonary vein. *r.a.* right auricle. *r.v.* right ventricle. *v.c.d.* descending vena cava. *v.c.a.* ascending vena cava. The right cavities of the heart, the pulmonary artery, and the systemic veins are in a state of normal fulness.

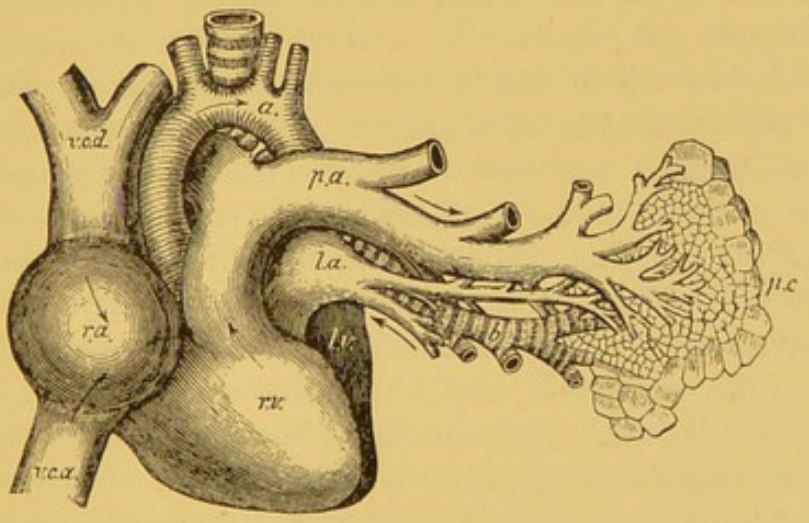

FIG. 2.—Represents the distension of the right cavities of the heart, of the pulmonary artery, and the large systemic veins in the final stage of apnœa (asphyxia). The letters have the same significance as in fig. 1. In addition, *p.c.* indicates the anæmic condition of the pulmonary capillaries. *b.* left bronchus. The right auricle and ventricle and the pulmonary artery are fully distended, the auricle having the form and smoothness of a distended ball, while the left cavities of the heart and the aorta are lapsed and nearly empty.

distended, and now the *right* auricle assumed the appearance of a round tense ball, while the left auricle had become nearly

empty and flaccid. The right ventricle also became so distended that it projected above the level of the left (fig. 2).

This was the condition of the heart's cavities when the animal died by the final arrest of the circulation; but, more than once when the circulation was nearly at a standstill, artificial respiration was resumed, and then all the phenomena rapidly changed. The blood at once passed freely through the lungs, the distension of the right cavities of the heart subsided, and the systemic arterial pressure became first excessive and then normal, when the blood had become thoroughly oxygenised and its passage through the terminal vessels was no longer resisted.

We now return to the consideration of the minute mechanism of the process by which, first, the systemic and then the pulmonary circulation is impeded when the respiration is suspended.

Dr. Reid maintained that the obstruction occurs in the *capillaries*, in accordance with Dr. Alison's doctrine. He says: 'He (Dr. Alison) has shown that this phenomenon is to be referred to an interesting general law of physiology, that has hitherto not received the attention which its importance demands, by which the movement of nutritious juices is influenced by the chemical changes, or, as he terms them, the vital attractions connected with the chemical changes constantly going on in the capillary vessels between those juices and the surrounding tissues by which nutrition and secretion are effected. That such a moving power exists, regulating the movement of the blood that flows through each individual organ, independent of any impulse from the living solids, cannot be doubted. Before arterial blood can be transmitted freely through any tissue or organ, it is not only necessary that the contractions of the heart be performed with a certain amount of force, but that the actions of nutrition and secretion be also in operation; so, in the same manner, before the blood can be transmitted through the lungs, it is not only necessary that the right side of the heart should retain its contractility, but that the chemical changes between the blood and the atmospheric air should proceed. This doctrine is still further illustrated by the fact which we have ascertained, that when

the blood in the systemic circulation becomes decidedly venous and unfit for carrying on the process of nutrition, it passes less freely through the capillary arteries into the veins.'

In another part of his essay, Dr. Reid expresses his conviction that the irregular afflux of blood to different organs and tissues 'cannot be explained by contractile movements of the smaller arteries or capillaries,' and he appears to consider that the best explanation of the phenomena has been afforded by Professor Draper, who shows, he says, 'in an apparently satisfactory manner, how the arterial blood should be drawn into the systemic capillaries, so long as the chemical changes between it and the surrounding tissues proceed, and how the venous blood, which has no chemical affinities for these tissues, should be driven onwards along the veins to the heart. If this occur in the systemic, the reverse will occur in the pulmonic circulation; for the venous blood has a strong affinity for the oxygen of the atmospheric air that occupies the air-cells upon which the pulmonic capillaries are ramified, while the arterial blood has none, and the venous blood is drawn into these capillaries and drives the arterial blood before it towards the heart.'

I have quoted this explanation of the phenomena at some length, because it was the best that could be suggested before the discovery of the structure and functions of the muscular arterioles and of the vaso-motor system of nerves. We are now in a position to substitute for the hypothesis of *vis à fronte*, drawing the blood onwards or retarding its progress, a simple physical explanation of all Dr. Reid's facts, which he himself would have been amongst the first to accept as conclusive. We substitute for mysterious hypothetical attractions and repulsions or suspended attractions the simple demonstrable phenomena of arterial contraction and relaxation under the influence of nervous agency, and we see how completely this explains the obstruction first in the systemic and then in the pulmonic circulation during the progress of apnœa.

The respiration being suspended, unoxygenised black blood at first passes freely to the left side of the heart and the systemic arteries and capillaries. Arrived there, either by its

direct stimulation of the muscular arterioles or, more probably, by a reflex influence through the vaso-motor nerves and centre, the arterioles are excited to contract, and by this action of the arterial stopcocks the blood-pressure in the arterial trunks is increased and the left cavities of the heart become distended and dilated, as seen in the exposed heart of the living dog (fig. 1, p. 27). The circulation through the systemic arterioles is thus impeded, but not arrested; some black blood passes through the capillaries, and this black blood not being capable of effecting the usual chemical changes between itself and the tissues, and becoming more and more entirely deoxygenised, in this abnormal state arrives through the veins at the right side of the heart and the pulmonary vessels. Reaching the pulmonary arterioles and capillaries, it excites there the same arterial contraction and resistance as had before occurred in the systemic vessels. The resistance offered by the contracting pulmonary arterioles, while on the one hand it tends to empty the left side of the heart and so to lessen the blood-pressure in the systemic arteries, on the other it causes that great distension and dilatation of the right cavities, more especially of the auricle, which are invariably found to exist when the chest is opened soon after death from apnœa, and which, in Dr. Rutherford's experiment, was plainly seen to occur during the lifetime of the animal (fig. 2, p. 27). It would appear that, while the systemic arterioles *immediately* resist the passage of imperfectly aërated blood, the resistance offered by the pulmonary arterioles does not commence until the deoxidation of the blood has passed beyond a certain stage, and this resistance is rapidly overcome by the readmission of atmospheric air to the lungs.

If the pulmonary arterioles began to contract and so to resist the onward movement of the blood as early as do the systemic arterioles, death from suspended respiration and circulation would be much more rapid than it actually is.

The instantaneous restoration of the pulmonary circulation by the readmission of air to the lungs is inconsistent with the theory which attributes the arrest of the circulation to the direct influence of unaërated blood on the vaso-motor centre. It is obvious that until the pulmonary circulation has been set

free the blood in contact with the nervous centres must still be black and unaërated.

Some additional facts which were observed during the progress of this experiment are worthy of remark. It was noted that the increased arterial pressure, which commenced as soon as black blood began to pass into the systemic vessels, had existed for some seconds before the left auricle and ventricle began to dilate, and continued for some time after the dilatation of those cavities had reached its height; then, while the distension of these cavities persisted, the arterial pressure began to fall, and it was just at this time that the right cavities, which had heretofore retained their normal size and form, began to be distended and dilated.

The question arises, What was the immediate cause of the diminished arterial pressure which began while the left cavities were still distended? It might possibly be due to diminished contraction of the terminal arterioles, but this is not a probable explanation. It was more probably a result of diminished power of the left side of the heart consequent on over-distension of its cavities. It is not unlikely that the heart's contraction may be in some degree enfeebled by the circulation of black blood through its nutrient vessels, but this obviously does not explain the dilatation first of the left cavities and subsequently of the right: a phenomenon which can be accounted for only by excessive contraction, first of the systemic and then of the pulmonary arterioles. We have additional evidence that the weakening of the left side of the heart, and the consequent diminution of the arterial pressure, are due to over-distension of the cavities, in the fact that, when, in consequence of the increasing resistance to the circulation through the pulmonary vessels, the blood-supply to the left side of the heart is diminished, the contraction of their muscular walls speedily restores the left cavities to their normal size.

It is manifest from the phenomena which we have been considering—the great distension, first, of the left cavities of the heart, then of the right, and the final rapid arrest of the circulation through the lungs, notwithstanding the forcible contraction of the right ventricle—that the active contraction of the systemic arterioles throughout the body is more than

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equal to the contractile power of the left ventricle, and the force of contraction in the pulmonary arterioles is more than equivalent to that of the right ventricle.[1]

It is evident that the immediate cause of death from apnœa is the arrest of the current of blood in the lungs, and this is confirmed and illustrated by the curious fact, which was first observed by Buffon, that the young of certain warm-blooded animals—for example, the dog, the cat, and the rabbit—may, within a few days after their birth, be immersed in water of moderate temperature for a period of sometimes half an hour before life is extinct. The explanation of this interesting phenomenon is without doubt to be found in the fact that, in these animals, the foramen ovale and the ductus arteriosus remain patulous for a few days after birth, so that, when, in consequence of the exclusion of air from the lungs, the pulmonary circulation is impeded, the blood passes directly from the right to the left side of the heart and to the aorta, the same as during fœtal life, and the circulation consequently continues much longer than in older animals, where, the foramen ovale and the ductus arteriosus being closed, all the blood has to pass through the pulmonary vessels and is thus subjected to their regulating and retarding influence.

With reference to the exact seat of the impediment which arrests the flow of blood through the lungs, I may remark that the extreme anæmia of the minute tissue of the lungs, when examined immediately after death in cases of acute apnœa, is evidence that the stoppage occurs before the blood has reached the capillaries. If, in accordance with the hypothesis of Alison and Reid, the blood were attracted into the capillaries and retained there, in consequence of its not having undergone the normal chemical changes, the capillaries would be in a state of engorgement, and not in that nearly bloodless state in which they are actually found to be.

[1] Some physiologists maintain that the comparative emptiness of the left side of the heart is due to the expulsion of the blood by *rigor mortis*. The error of this view is shown by the fact that when the chest is opened *immediately* after death the left cavities are found nearly empty, and in Dr. Rutherford's experiment the emptying of these cavities was seen to occur gradually while the animal was still alive.

Nitrous oxide anæsthesia.—In the phenomena of apnœa with the resulting rapid arrest of the circulation which occurs when nitrous oxide gas is inhaled as an anæsthetic, we have a very interesting confirmation of the results obtained by excluding atmospheric air from the lungs of animals; and, on the other hand, the records of physiological experiments enable us more completely to understand and interpret the facts of nitrous oxide anæsthesia. On several occasions I have availed myself of the opportunity afforded me by the courtesy of the staff of the Dental Hospital to watch the phenomena which attend the inhalation of the gas, and I will now briefly describe them.

In most cases, during the first few seconds the pulse and the breathing are quickened, as a result probably of emotional excitement. In the next stage, the breathing becomes slow and shallow and the pulse full and firm. Then, after a period which varies in different cases from forty to eighty or ninety seconds, the pulse suddenly becomes almost, or even quite, imperceptible, the features become livid, the pupils are widely dilated, there is a state of general muscular rigidity; in short, all the phenomena of the first stage of an epileptic fit are present. The mouth-piece being removed, the morbid phenomena quickly pass away, the features regain their normal colour, the pulse returns, and for a few seconds has again a full and throbbing character, but quickly regains its normal condition.

The explanation of the phenomena appears to be sufficiently obvious. It is admitted on all hands that, at the temperature of the body, the nitrous oxide gives up no oxygen to the blood or the tissues. The gas becoming rapidly diffused and replacing the oxygen in the lungs and in the blood, black unoxygenised blood passes into the systemic arteries, and excites, through the vaso-motor nerves and centre, contraction of the muscular arterioles. The resistance thus offered to the passage of unaërated blood through the terminal arteries explains the temporary fulness and tension of the radial pulse.[1] The par-

[1] Some writers have asserted that the full pulse during the early stage of nitrous oxide inhalation is evidence of the direct stimulant action of the gas upon the heart; but since the exclusion of atmospheric air has precisely the same effect upon the pulse, that explanation of the arterial fulness and tension is evidently untenable.

tially unoxygenised blood, passing through the systemic capillaries without the usual interchange of materials between it and the tissues, soon becomes so entirely deoxygenised as, when reaching the lungs, to excite contraction of the pulmonary arterioles. The resistance thus offered to the passage of blood through the lungs explains, on the one side, the systemic arterial emptiness with feebleness or even complete disappearance of the pulse, and, on the other, the systemic venous fulness with lividity of the skin. The epileptiform condition is explained by the sudden and extreme diminution of the blood-supply to the brain, the blood at the same time being unaërated. (See the chapter on the *Pathology of Epilepsy*.)

If the inhalation were continued, death would occur from the complete arrest of the pulmonary circulation and consequent over-distension of the right side of the heart, and this is the mode in which death occurs when an animal is killed by the continued inhalation of the gas. A year ago, my friend and colleague Mr. Hamilton Cartwright assisted me to kill two rabbits with the gas. In both animals convulsions preceded death; and, the chest being opened immediately after death, the heart was found still beating. The right cavities and the systemic veins were greatly distended with blood, while the left cavities and the aorta were comparatively empty and flaccid, the blood on both sides of the heart being equally black. The lungs were anæmic and collapsed to an extreme degree.

It will be seen that the phenomena observed during life and the appearances after death from the inhalation of the nitrous oxide gas are precisely similar to those which result from suspension of the respiration in the human being and in the lower animals. (See fig. 2, p. 27.)

It is evident, from the many thousands of cases in which the gas has been given and the extreme rarity of a fatal accident from its use, that, in the hands of a skilled and careful operator, no great risk attends the employment of this anæsthetic;[1] but it is also obvious that, to a patient with a feeble

[1] 'The late Mr. Clover, in a letter to the *Lancet* (vol. i. 1876), stated that he had put to sleep more than eleven thousand persons with the gas without one fatal result.

fat heart, the distension of the right cavities which accompanies the disappearance of the radial pulse and the general lividity of the features must be attended with some degree of risk, and the danger must be increased when the muscles of the trunk and limbs being convulsed, the pressure of the contracting muscles upon the veins drives the blood forcibly towards the right cavities of the heart, and so adds to their distension.

CHAPTER III.

ON CERTAIN PHYSICAL PHENOMENA CONNECTED WITH THE CIRCULATION, RESPIRATION, SECRETION, AND ABSORPTION.[1]

Facts proving rapidity of Absorption and Elimination—Effect of Overfulness and of Depletion of Blood-vessels—Venesection lowers the Density of the Blood—Action of a Hydragogue in removing Dropsy—Mere Diarrhœa does not thicken the Blood—Influence of Venous Engorgement in causing Blood-thickening—Explanation of the Blood-thickening during the Collapse Stage of Cholera.

In the body of a living animal there is an incessant movement of liquids. Not only is the blood constantly propelled through the vessels, but there is a continual passage of fluids from the blood to the tissues, and from the tissues into the blood. The phenomena of the circulation, respiration, secretion, and absorption, are most intimately correlated with each other and with an unceasing movement of the interstitial fluids. Life implies motion; stagnation involves death, either local or general. The movement of the blood is dependent on obvious mechanical processes. The contractions of the heart, the elastic resiliency of the large arteries, the regulating power of the muscular walled arterioles, the pressure of contracting muscles on the veins, and the movements of the chest in respiration, these forces, regulated and controlled by the nervous system, are the main influences concerned in maintaining the circulation of the blood. The proofs and the explanation of the constant and rapid interchange of fluids between the blood and the tissues are less obvious, but the phenomena will well repay an attentive study.

The quick and powerful action of morphine and other substances, when injected beneath the skin, affords a familiar

[1] *British Medical Journal*, Jan. 1 and 8, 1876.

illustration of the fact that a current of liquid is constantly passing from the tissues into the blood; the blood being, of course, the vehicle by which the absorbed morphine reaches and acts upon the nervous centres.

The late Dr. Bence Jones made a number of interesting experiments to demonstrate the rapid passage of crystalloids into and out of the vascular and non-vascular textures of the body.[1] The experiments were made with the salts of lithium, which are readily detected by spectrum analysis. It was found that in guinea-pigs, within a quarter of an hour after three grains of chloride of lithium had been taken by the stomach, the lithium was present not only in all the vascular textures, but even in the cartilage of the hip-joint and in the humours of the eye. In half an hour it was detected in the crystalline lens. When the same quantity was injected beneath the skin, even in four minutes it might be detected everywhere except in the lens, and in ten minutes it had passed into the lens.

In the human subject, lithium may be detected in the urine in from five to ten minutes after a ten-grain dose of the carbonate has been swallowed; and it continues to pass out by the urine for six or seven days. Carbonate of lithium was administered to patients at variable intervals before the operation for cataract. After a twenty-grain dose, it was found in small quantity in the lens in two hours and a half; and it was found in every part of the lens in three and a half, five, and seven hours. No trace was detected in the lens when twenty grains of the carbonate of lithium had been taken seven days before the extraction of the cataract. Within that period the whole of the lithium had passed out of the tissue of the lens, and had ceased to be excreted by the kidneys. This rapid passage of substances into and out of the tissues belongs to the class of phenomena which come under the designation of diffusion of liquids.

Most liquids exert a molecular attraction upon each other, as a result of which they mingle with and diffuse into each other. This diffusion, by a process of endosmosis and exos-

[1] *Proceedings of the Royal Society*, June 15, 1865; and *Croonian Lectures on Matter and Force*, Appendix III.

mosis, takes place through membranes; but the interposition of an animal membrane has this important influence, that, while it allows the ready passage of liquids containing in solution substances which the late Professor Graham has designated 'crystalloids,' it arrests, or greatly retards, the passage of 'colloids' or gum-like substances. A membranous septum, therefore, serves as a means by which crystalloids may be separated from colloids. Place albuminous urine in a dialyser floated on distilled water, and in the course of a few hours the crystalloids of the urine—the urea and the salts—will have passed out of the dialyser, while the greater part of the colloid albumen is retained within. The molecular attraction between the dissolved crystalloids and the membranous septum acts as a motor force, which gradually carries the materials of the crystalloid into the liquid outside the dialyser. The fact that some albumen always diffuses through a dead membrane proves that such a membrane differs from the walls of healthy Malpighian capillaries, which, in the normal state, permit no escape of albumen. The passage of albumen through the Malpighian capillaries is a result of an abnormal condition of the blood or of the walls of the vessels, or, it may be, of abnormal blood-pressure.

The same physical forces are concerned in the production of *Renal Dropsy*. When, in consequence of acute inflammatory disease of the kidney, the urinary crystalloids and water accumulate in the blood, they pass thence, by an exosmotic process, through the walls of the capillaries into the subcutaneous areolar tissue and into one or more of the serous cavities. On the other hand, when the kidney has reverted to its normal condition and recovered its functional activity, the blood being first freed from excess of urinary solids and water, a current of dissolved urinary crystalloids sets in from the tissues into the blood, and out by the kidneys. Thus, by a copious flow of urine, the dropsical accumulation is soon swept away.

We have an illustration of the result of an excess of a crystalloid in the blood when the kidneys are healthy in the phenomena of *diabetes*. The dissolved crystalloid sugar finds a ready outlet through the kidneys, and carries with it so

much water that, notwithstanding the copious imbibition of liquid which is prompted by the urgent thirst, the tissues of the body often become more or less dehydrated.

The diffusion of a soluble crystalloid through the blood and tissues, and its final elimination, occur probably in this manner. Whether introduced by the stomach or beneath the skin, the crystalloid enters the blood, and, mingling with that fluid, is rapidly and equally diffused into all the tissues. In a few minutes, as we have seen, the salts of lithium begin to pass off by the kidneys. The elimination by the kidneys lessens the amount in the blood, and the distribution through the system is equalised by the return of a portion of the crystalloid from the tissues to the blood. This sequence of events continues: escape by the kidneys; passage back of a portion of the salt from tissues to blood; the blood and the tissues at each round of the circulation retaining less and less of the unassimilated salt, until, in the course of a few days, the whole of the crystalloid has passed out, mainly through the kidneys, but partly, perhaps, by some other channels.

As overfulness of the blood-vessels results often in a transudation of water from the blood into the tissues, so depletion of the blood-vessels dertermines the rapid passage of water from the tissues into the blood. The soft tissues, namely the muscles, the viscera, and connective tissues, be it remembered, contain four-fifths by weight of easily separable water. Dr. Polli, an Italian physician, in the course of an elaborate series of experiments on human blood, ascertained that the specific gravity of the blood was rapidly lowered, not only by successive bleedings, but by a single bleeding. He found, for instance, that at the end of a single bleeding, taking the mean of forty experiments, the blood had a lower density than that drawn at the commencement of the venesection, in the proportion of 5·96 to 6·127.[1] This result is in entire agreement with the experiments of Zimmerman on the blood of animals. For instance, Zimmerman found that the blood which first flowed from the crural artery of a dog contained 199·3 parts of solids to 800·7 of water. After the loss of about ten ounces of blood the proportion of solids in 1,000 parts was reduced to 187,

[1] *Medico-Chirurgical Review*, 1847, p. 306.

while the water had increased to 813. A horse suffering from tetanus had his carotid artery opened: 1,000 parts of the serum of the blood which first flowed contained 106 parts of solids; while, after fifteen pounds of blood had flowed, the proportion of solids in the serum was reduced to 100·5 in 1,000.[1]

The explanation of the rapid passage of water from the tissues into the blood appears sufficiently obvious. Normally, there is an equilibrium of pressure within the blood-vessels and in the surrounding tissues. A partial emptying of the vessels by an escape of blood tends to lessen the pressure within them, and the equilibrium is restored by water being pressed from the tissues through the capillary walls. Thus the fulness of the blood-vessels is maintained, while the blood is diluted with water, and its proportion of solids is lessened.

We have illustrations of the same hydraulic principles in some morbid phenomena and in the practice of therapeutics. Thus, a dropsical patient has his blood-vessels overfull, and his tissues flooded with excess of water. A dose of elaterium excites copious watery discharges from the bowel; this escape of liquid tends directly to deprive the blood of its water. Then the drain of water from the blood, and the consequent diminution of pressure within the vessels, cause an absorption of the dropsical fluid, which is now pressed from the tissues through the capillary walls; and thus, by a copious purging, a dropsical accumulation may be removed. Sir Thomas Watson mentions the case of a man in whom a profuse purging excited by intoxicating doses of rack punch, led to the disappearance of a large hydrocele in the course of one night. Here it is clear, as Sir Thomas remarks, 'that the expenditure of serous fluid from one part led to its absorption into the blood from another.' In other words, the pressure within the blood-vessels having been lessened by a copious drain of liquid, the equilibrium was restored by water from the hydrocele being pressed through the walls of the capillaries into the blood.

Now let us consider the physical effects of copious watery purging upon a subject not dropsical, but previously in a normal condition. Mere diarrhœa, however copious, has no appreciable influence in thickening the blood or increasing its

[1] Heller's *Archiv für Chimie und Microskopie*, 1847, p. 385.

specific gravity, and the explanation of this is sufficiently obvious. As venesection causes a rapid dilution of the blood by water passing from the tissues through the walls of the vessels, and as the operation of a hydragogue purgative often results in the removal of a dropsical accumulation, so profuse choleraic purging first tends to lessen the contents of the blood-vessels, and then, to fill the vacuum and restore the equilibrium, water passes from the soft tissues into the blood, which thus maintains its fluidity notwithstanding the copious discharge of liquid through the bowels. When the watery discharges from the bowels have ceased, the hydraulic currents are reversed. During the stage of diarrhœa, absorption of water through the mucous membrane of the alimentary canal is prevented by the outward flux of liquid; the current is entirely outwards; but, when the discharges have ceased, water taken into the stomach rapidly enters the blood-vessels, and is thence transferred to the tissues, which have before given up a large portion of their water to the blood. So great is now the demand for water by the tissues, and so strong is the current towards them, that for a period, varying in different cases from a few hours to several days, little or no urine is secreted, the main stream of water being into the dehydrated tissues: a direct reversal of the tide of liquid which sets from the tissues through the blood-vessels, towards the alimentary canal, during the diarrhœal stage. The stream of liquid from the blood into the tissues may carry with it urea and other excreta, and thus may contaminate and poison the tissues. This tissue-poisoning is probably in part the cause of the consecutive fever of choleraic attacks. On the other hand, we have evidence that, when in consequence of degeneration of the kidneys the blood and the tissues are contaminated with urine, a hydragogue purgative removes not only water, but also some urinary excreta from the blood, and thus counteracts the tendency to uræmic poisoning. We know from experience that the surest way to restore the secretion of urine after a copious watery diarrhœa is to supply an abundance of water to the blood and the tissues through the stomach and through the skin by an occasional immersion in a tepid bath. If frequent vomiting interfere with the intro-

duction of liquid through the stomach, tepid water may be injected into the rectum.

I wish now to direct attention to some of the physical results of an impeded return of blood through the systemic veins. In order that the blood may maintain its normal composition and its physical properties, it must move freely through the vessels. Dr. Polli,[1] by a series of experiments, clearly proved that the blood is rendered more dense by inducing an artificial stagnation or engorgement by means of the fillet applied for some time previously to opening the vein; and his conclusion is, that 'venous blood, when it is prevented by any obstacle from circulating freely in the veins, becomes more dense, or loses a certain portion of its watery principle.' And he asks: 'Does not this fact constitute a principal pivot of the doctrine which explains œdemas, cellular dropsies, and other infiltrations, by considering them as exudations induced by obstructed circulation?' Without doubt, the œdematous swelling of a limb, whose principal veins are obstructed, is the counterpart of the blood-thickening which results from the transudation of the watery constituents. The force which drives the water through the walls of the vessels is derived from the left ventricle of the heart. The blood is driven through the capillaries into the veins, and, its passage through the venous channels being impeded, a portion of its water is forced through the capillary walls into the tissues outside. The result is an œdematous dropsical swelling of the limb, and an increased thickness and consistency of the blood within the veins. This simple experiment explains some forms of blood-thickening which have perplexed pathologists.

It explains the blood-thickening of apnœa. It is a well-known fact that a great impediment to the aëration of the blood is always associated, as one of its consequences, with an impeded circulation through the lungs, and a consequent engorgement of the right side of the heart and of the whole systemic venous system.[2] One result of this impeded venous circulation, when long-continued, is a puffy, œdematous con-

[1] *Op. cit.*, p. 306.

[2] See the *Lecture on the Physiology of the Circulation*, p. 14.

dition of the subcutaneous tissue, especially of the face and neck; and another result is that the blood becomes dark, thick, and treacly, and coagula often form in the right cavities of the heart and in the pulmonary artery.

Dr. George Buchanan, in one of his papers on tracheotomy in croup and diphtheria, read before the Medico-Chirurgical Society of Glasgow, in giving the history of a case in which he opened the windpipe in an advanced stage of laryngeal diphtheria, states that, 'as soon as the wound was made, a quantity of dark, tarry-looking blood flowed from the edges, and was with difficulty cleared away by firm and repeated applications of a moist sponge.' Dr. Buchanan, again referring to the state of the blood, says 'it was quite evident that that fluid was in a dark and viscid state. The darkness might be accounted for,' he says, 'by the impeded respiration; but the tarry consistency struck me as a condition which might be one of the pathological elements of the disease (diphtheria) in an advanced stage.' The tarry consistency of the blood is, in fact, one of the usual results of extreme and prolonged apnœa. Not long since, during the operation for tracheotomy on account of laryngeal obstruction of long standing, I saw a decolourised fibrinous coagulum coughed out of one of the cut veins in the neck. When I say it was coughed out, I mean that it was thrust out of the vein during the expiratory effort of coughing, which, as we continually see, always forcibly drives the blood into the superficial cervical veins.

There is also chemical evidence of the blood-thickening which results from apnœal conditions. Thus, the late Dr. Dundas Thomson, who published an elaborate paper on the 'Chemistry of the Blood in Cholera,'[1] states that one specimen of blood from a patient who laboured under an affection of the mucous membrane of the air-tubes contained a greater excess of solids in proportion to water than he had found even in cholera blood.

I now come to one of the main objects of this communication, which is to suggest a more complete explanation than has hitherto been given of the increased density and viscidity of the blood during the collapse stage of cholera. In order to

[1] *Medical-Chirurgical Transactions*, xxxiii. 74.

explain the phenomena, we must have the facts clearly set forth.

The main facts, then, are these. The blood-thickening is a phenomenon which is present only during the stage of collapse; it does not occur during the diarrhœal stage, and it quickly passes away when reaction occurs. We have already seen that mere diarrhœa, however profuse, does not thicken the blood; the reason being that, so long as the circulation of the blood is unimpeded, water passes from the tissues into the blood as rapidly as it escapes from the blood into the bowels. Dr. Garrod, many years ago, made careful analyses of the blood in cases of cholera, and he states that 'the amount of intestinal evacuations in any case is by no means an indication of the extent to which the blood has become altered.'[1]

The blood-thickening, as a rule, bears a direct relation to the degree and the duration of the collapse. Now, there is one condition which is common to cases of apnœa and of choleraic collapse; and that is an impeded flow of blood through the lungs. This impeded circulation, with the consequent engorgement of the systemic venous system, is the main cause of the blood-thickening, which is also common to the two pathological states. The blood is thickened partly in consequence of its slow movement and partial stasis within the veins; but mainly, perhaps, by the passage of its watery portion through the walls of the capillaries. In the choleraic cases, it is probable that the passage of water from the distended venous system into the tissues is facilitated and much increased by the previous partial dehydration of the tissues during the diarrhœal stage. The less water the tissues contain, the more readily would water pass into them through the walls of the over-distended capillaries.

There is another condition, common to cases of apnœa and choleraic collapse, which contributes to the blood-thickening. In consequence of the defective supply of oxygen to the system, resulting from the partially arrested pulmonary circulation, those solids of the blood, especially the coloured corpuscles, which normally, under the influence of oxygen, are disintegrated and converted into the correlated products,

[1] *London Journal of Medicine*, i. 409.

carbonic acid and biliary and urinary constituents, accumulate in the blood and so increase its density.

On the other hand, during the stage of reaction when the full current of arterial blood brings with it a free supply of oxygen, there is a rapid disintegration of the accumulated blood-corpuscles, with a resulting abundant secretion of bile and urine, and consequently a rapid restoration of the normal proportion of solids and liquids in the blood, and this notwithstanding the continuance of the intestinal discharges in greater or less abundance.

It is evident that during the collapse stage, in consequence of the partially arrested pulmonary circulation with resulting distension of the systemic venous system, the loss of water by the intestinal discharges can no longer be compensated by the absorption of water from the soft tissues. The liquid currents are now reversed, and water passes from the distended systemic veins into the partly dehydrated tissues. So long as the state of collapse continues, therefore, the discharges from the alimentary canal tend to increase the blood-thickening.

There is yet another co-operating cause of the blood-thickening during the stage of collapse. The greatly contracted pulmonary arterioles probably act as a filter, allowing the more liquid parts of the blood to pass on into the systemic arteries, while the more solid parts are kept back and accumulate in the systemic venous system.

This filtering action of the contracting arterioles upon the blood will be more fully explained hereafter in connection with the pathology of choleraic collapse.

CHAPTER IV.

ON SOME RESULTS OF A RETROGRADE ENGORGEMENT OF THE BLOOD-VESSELS.[1]

The Lung contains two sets of Blood-vessels: Pulmonary for Respiration, Bronchial for the Nutrition of the Lung and Pleura—State of Pulmonary and Bronchial Vessels in the Collapse Stage of Cholera—Comparison of Spasmodic Asthma with Spasmodic Cholera—Acute and Chronic Apnœa—Hæmoptysis of Asthma—Case of Tricuspid Regurgitation—Condition of the Sound Lung in cases of One-sided Pneumonia or Pleurisy—Effect of Vesicular Emphysema on the Bronchial Vessels—Results of Impeded Circulation through the Liver—The Circulation through the Liver, the Kidney, and the Lung, compared and contrasted—The Results of Impeded Circulation through each of these Organs.

My object in the present communication is to direct attention to certain results of a retrograde engorgement of the blood-vessels, consequent upon an impediment to the onward movement of the blood. Most of the facts to which I am about to refer are well-known and familiar facts; I believe, however, that they have not hitherto been collated and analysed with the care and the accuracy which their pathological interest and their practical importance demand.

I have to speak first of certain phenomena which result from an impeded circulation through the *lungs*. I shall afterwards refer briefly to some analogous phenomena which follow an interrupted circulation through the *liver* and through the *kidney* respectively.

Let me first, then, direct attention to the well-known peculiarities of the blood-supply to the lungs. The lung, as a respiratory organ, is supplied by the pulmonary artery, the blood from which passes through the pulmonary capillaries and veins from the right to the left side of the heart. The

[1] *Lancet*, June 20, 1868.

lung, as an organ whose tissues have to be nourished, is supplied with blood from the aorta by the bronchial arteries, and this blood, having been distributed to all the proper tissues of the lung, is returned by the bronchial veins (one opening into the left superior intercostal, and the other into the vena azygos) into the superior cava. In the lung, therefore, there is the pulmonary circulation proper, and, in addition to this, there is the bronchial circulation, by means of which its own tissues are nourished, this bronchial circulation constituting a part of the general systemic circulation. 'The bronchial arteries and veins carry blood for the nutrition of the lung, and are doubtless also the principal source of the mucous secretion found in the interior of the air-tubes.'[1] It is generally believed, and it probably is a fact, that where these two different sets of vessels come into contact, where the bronchi and intercellular passages merge into air-cells, there is a communication between the two sets of vessels—between the pulmonary and the bronchial systems of vessels.

I have elsewhere sufficiently proved that during the collapse stage of cholera there exists a great impediment in the minute branches of the pulmonary artery. The result is that those parts of the vascular system which lie in front of the smallest branches of the pulmonary artery are comparatively empty of blood, while the parts behind are distended. The whole of the systemic venous system is gorged with blood; hence the lividity of the face, and hence congestion of the bronchial veins and capillaries, which may impart to the lung a dark colour and an appearance of congestion, while yet the lung, as a whole, contains very little blood, and is much reduced in weight. Even the *pulmonary* capillaries may become partially injected in consequence of an impeded return of blood through the bronchial veins. There is a communication between the two sets of vessels about the terminal bronchi, and a part of the blood carried by the bronchial arteries is returned by the pulmonary veins. An impeded return by the bronchial veins would therefore divert more bronchial blood into the pulmonary capillaries and veins.

Some pathologists have supposed that the impeded transit

[1] *Quain's Anatomy*, edited by Dr. Sharpey, 7th edition, p. 903.

of blood through the lungs in the collapse stage of cholera is a result of its becoming too thick and viscid to pass through the capillaries. This view is proved to be erroneous by a number of facts, and amongst others by the anatomical fact that the mass of blood is arrested *before* it reaches the pulmonary capillaries, while the blood is obviously not too thick to enter the bronchial capillaries, which, although of smaller size than the pulmonary capillaries, are as over-full as the pulmonary capillaries are empty of blood. In fact, there is usually a direct relation between the anæmia of the pulmonary capillaries and the engorgement of the bronchial; and the contraction of the minute pulmonary arteries, presenting an abrupt bar to the onward movement of the blood, is the probable cause of both phenomena. One result of the engorgement of the bronchial veins and capillaries during the collapse stage of cholera is a tendency to passive serous transudation through the mucous membrane into the bronchial tubes. The consequence is that after prolonged collapse the minute bronchi and air-cells become more or less filled with serum; and when during reaction the blood again passes freely through the pulmonary artery, the respiratory changes are interfered with by the œdematous condition of the lung, and there often occurs a fatal apnœa.

Now let us compare the phenomena of cholera collapse with those of spasmodic asthma. The general appearance of a patient during a severe paroxysm of asthma is strikingly like that of one in the collapse of cholera. Dr. Hyde Salter, in his masterly treatise on Asthma, says (p. 71): 'If the bronchial spasm is protracted and intense, the heat of the body falls; the oxygenation of the blood is so imperfectly performed, from the sparing supply of air, that it is inadequate to the maintenance of the normal temperature; the extremities especially get cold and blue and shrunk; I have known the whole body deathly cold and resist all efforts to warm it for four hours. But while the temperature is thus depressed, the perspiration produced by the violent respiratory efforts may be profuse, so that the sufferer is at the same time cold and sweating. It is this union of coldness and sweat, combined with the duskiness and pallor of the skin, that gives to the

asthmatic so much the appearance of a dying man. The pulse during severe asthma is always small, and small in proportion to the intensity of the dyspnœa; it is so feeble sometimes that it can hardly be felt.'

The resemblance between some of the most striking features of the asthmatic paroxysm and the collapse of cholera is obvious. What, then, is common to these two forms of collapse? Obviously not a drain of liquid from the blood, which was so long and so erroneously supposed to be the main cause of cholera collapse, but a partial arrest of the pulmonary circulation. In the lecture on the physiology of the circulation I have shown that in cases of acute apnœa there is the same evidence of an arrest of blood in the minute branches of the pulmonary artery as exists in the case of cholera collapse. In both pathological conditions the pulmonary capillaries contain very little blood, while the right cavities of the heart and the pulmonary artery are distended, the left cavities of the heart being comparatively empty. In both asthma and cholera the circulation is impeded by the muscular contraction of the pulmonary arterioles; and hence the symptoms of collapse in both diseases. In cholera the contraction of the arterioles is a result of the poisoned blood in the vessels; in asthma it is a consequence of the partial apnœa occasioned by spasm of the bronchi. In cholera there is a primary asphyxia and a secondary apnœa,[1] consequent upon the blood-stasis; in asthma there is a primary apnœa, the result of bronchial spasm, and a secondary asphyxia. In both forms of disease the symptoms of collapse may speedily be removed for a time by measures which overcome the primary spasm: in asthma by the inhalation of chloroform, which relaxes the bronchial spasm; in cholera by the injection of a hot liquid into the veins, which, reaching the lungs, overcomes the arterial spasm. In asthmatic apnœa we have evidence of congestion of the bronchial system of vessels in the occasional occurrence of bronchial hæmoptysis, and in the more constant occurrence of bronchial mucous expectoration, which is copious and prolonged in

[1] I use the terms 'asphyxia' and 'apnœa' in their strictly literal sense of pulseless and breathless.

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proportion to the intensity and duration of the previous paroxysm.[1]

I have dwelt at some length upon the phenomena of asthma as a type of partial acute apnœa. The causes of acute apnœa are very various; but in all essentials the phenomena are alike. Whether the apnœa be the result of hanging, drowning, suffocation, inflammatory croup, diphtheria, epileptic or tetanic spasm implicating the respiratory muscles, division of the vagi nerves as in Dr. John Reid's experiments, apoplectic coma, narcotic poisoning, or injury to the upper part of the spinal cord,—in each and all of these cases, in proportion to the suddenness and completeness of the apnœa, the pulmonary capillaries are anæmic, while the vessels containing black blood which lead up to those capillaries are gorged. In all these cases, too, the retrograde passive engorgement of the bronchial veins and capillaries results in a serous and mucous, and sometimes sanguineous, exudation into the bronchial tubes.

We are now in a position to understand the condition of the lungs which is found in cases of *chronic* apnœa. In acute apnœa we have seen that the lungs are anæmic, and we have explained this by the stop-cock action of the minute pulmonary arteries consequent upon the suspension of the respiratory changes. When death occurs after a state of partial apnœa has continued for many hours, or for several days, the lungs are always found more or less gorged with blood and serum. The explanation of this is simple. The apnœal blood-stasis in the pulmonary artery throws back the blood upon the bronchial veins and capillaries, from which there is poured a serous dropsical exudation into the bronchial tubes. This fluid,

[1] Dr. Hyde Salter, in the first edition of his book, having attributed the hæmoptysis of asthma to rupture of the pulmonary capillaries, in the second edition, with characteristic candour, adopts the explanation which I had given him and attributes the blood-spitting to a retrograde engorgement of the bronchial veins and capillaries. After explaining my theory he says (p. 89, note): 'I believe he is perfectly right; I believe he has solved the difficulty, and his solution satisfactorily explains to my mind, not only the source of apnœal hæmoptysis, but, what I never could well understand before, the invariable sequence of bronchial mucous exudation upon any form of protracted partial apnœa; for that which would produce bronchial hyperæmia, even though passive, would necessarily produce an increase of the bronchial secretion.'

sometimes blood-tinged, reaches the extremities of the bronchial tubes and the air-cells, where it compresses the network of pulmonary capillaries, and impedes the flow of blood through them. Thus the lung becomes more and more gorged with blood and serum. An analogous phenomenon may often be witnessed in anasarcous legs. The accumulation of liquid in the areolar tissue sometimes compresses the vessels so as to cause passive congestion, and not unfrequently inflammation and gangrene, of the skin and tissues beneath the skin.

A case of tricuspid regurgitation, which occurred under my care in the hospital some years ago, affords an instructive illustration of the effects of an impediment originating in the right side of the heart, first upon the bronchial, and secondly upon the pulmonary, circulation. I gave the particulars of this case in a clinical lecture which was published in the *Medical Times and Gazette* of February 13, 1858. A woman, aged fifty-two, was admitted with general dropsy, albuminuria, a systolic bellows-sound at the bottom of the sternum, distended and pulsating jugulars, and the physical signs of bronchitis. A single large dose of elaterium acting very freely upon the bowels cleared away the dropsy, the albuminuria, and the bronchial secretion. After a short time, however, *all* the symptoms returned, and she died. We found, as we had expected, great dilatation of the tricuspid orifice, while the valves on the left side of the heart were normal. The lungs were much engorged. In this case it is clear that the primary cause of all the suffering was incompetence of the tricuspid valve. There was, consequently, a reflux of blood into the systemic veins; renal congestion and albuminuria, anasarca, bronchial congestion and exudation, and then a secondary obstruction of the pulmonary capillaries. It is interesting to note that before her admission she had spat some blood, the source of which was in all probability the over-gorged bronchial capillaries. We are familiar enough with the occurrence of hæmoptysis as a consequence of mitral or aortic valve disease; the source of such hæmoptysis being the pulmonary capillaries, while disease on the right side of the heart throws back the strain and pressure primarily upon the bronchial vessels. If there were no bronchial vessels, if the sole blood-supply to

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the lungs were through the pulmonary artery, the tendency of obstruction at the right side of the heart would obviously be to render the lungs anæmic, and not to overfill them with blood.

Another interesting phenomenon, allied to those to which I have already referred, is the œdema of the sound lung, which not unfrequently occurs when one lung has been suddenly consolidated by pneumonia, or compressed by a pleuritic effusion. In such a case we sometimes hear crepitating sounds in the bronchi of the healthy lung; and we look upon this condition of things with anxiety, because we know that an increase of the œdematous effusion into the bronchi may cause fatal apnœa. Until quite recently I have always considered that the pulmonary capillaries were the source of the serous exudation in these cases. I have now no doubt that it is from the bronchial capillaries mainly that the liquid escapes. In consequence of the impervious condition of the lung on the diseased side there is a state of partial apnœa; more blood is sent to the sound lung than can readily be aërated; its progress is checked and regulated by the stop-cock action of the minute pulmonary arteries; there is more or less engorgement of the right cavities of the heart and the systemic veins; and with this there is bronchial congestion, and a consequent serous exudation into the bronchi.

Once more, it is notorious that patients who have general vesicular emphysema of the lungs are very liable to bronchial catarrh. Why is this? In an emphysematous lung there is dilatation of air-cells, rupture of their walls, and obliteration of capillaries. The vascular network of the lung is much diminished; there is consequently an impeded pulmonary circulation, fulness of the right side of the heart and systemic veins, bronchial capillary congestion, and so a liability to catarrh and bronchitis. This passive congestion of the bronchial mucous membrane predisposes to catarrh and bronchitis, just as varicose veins in the leg predispose to inflammation of the integuments. Another result of the passive congestion of the bronchial capillaries, which is a consequence of emphysema, is the occasional occurrence of bronchial hæmoptysis. Of this I have seen a considerable number of instances, and not a few

in which the occurrence of blood-spitting has led to an erroneous diagnosis of tubercular disease of the lung.

The *liver* resembles the lung in this respect, that, as an excretory organ, it is supplied with venous blood. Like the lung, too, it receives a separate supply of arterial blood, through the hepatic artery, for the nutrition of its own tissues. It differs from the lung in this important particular, that the blood retains its venous character as it emerges from the liver. There is, therefore, no need for a companion vein to the hepatic artery corresponding with the bronchial veins ; but the blood from the hepatic artery, after supplying the liver tissues, mingles with the portal blood, and so passes on into the vena cava. In cases of cirrhosis of the liver there is an impeded circulation through the gland : the whole of the portal venous system is consequently engorged ; the hæmorrhoidal veins are often enlarged ; there is sometimes hæmorrhage from the mucous membrane of the alimentary canal, and still more frequently a serous dropsical effusion into the cavity of the peritoneum.

The *kidney* differs from both the lung and the liver in this respect, that it has but one source of blood-supply. The blood of the renal artery serves at once to nourish the gland and to supply the materials for its secretion. With respect, however, to the distribution of blood within its substance, the kidney presents some points of analogy with the lung on the one hand, and with the liver on the other. It resembles the lung in the fact that it has two sets of capillary vessels—the Malpighian capillaries and the intertubular capillaries ; but it differs from the lung in the fact that the same blood passes in succession through both sets of capillaries. Then there are some points of analogy between the capillary arrangement within the kidney and that within the liver. Sir Wm. Bowman, in his original paper 'On the Structure of the Malpighian Bodies of the Kidney,'[1] pointed out that each efferent vein may be looked upon as a portal vein in miniature, the resemblance between the portal and the efferent vein consisting in the fact that each vein intervenes between two sets of capillary vessels. And thus we find that as an impediment to the circulation

[1] *Philosophical Transactions.*

through the liver causes a serous exudation into the cavity of the peritoneum from the capillary vessels in which the portal vein takes its rise, so an impeded circulation through the kidney causes a serous exudation from the capillary vessels in which these miniature portal veins—the efferent veins—originate, and this serous exudation from the Malpighian capillaries passes into the uriniferous tubes. Dr. George Robinson, Frerichs, and others have shown that a ligature on the renal vein of a rabbit causes a retrograde engorgement of the Malpighian capillaries, and a consequent escape of blood constituents into the uriniferous tubes, which, mingling with the urine, render it albuminous. In cases of Bright's disease there is an impeded circulation through the intertubular capillaries, which is, in part at least, occasioned by the pressure of the swollen tubes upon the vessels which lie between them; there is, consequently, an engorgement of the Malpighian capillaries, a serous exudation into the uriniferous tubes, and thus an albuminous condition of the urine.

We see, then, that while an impeded circulation through the pulmonary vessels throws back the blood upon the bronchial veins and capillaries, and causes a serous, mucous, or sanguineous exudation into the bronchial tubes, an impeded circulation through the liver distends the portal veins and capillaries, and causes hæmorrhage from the mucous membrane of the alimentary canal, or serous effusion into the cavity of the peritoneum; and, lastly, an impeded circulation through the kidney distends the Malpighian capillaries, and causes a serous exudation, and sometimes hæmorrhage, into the uriniferous tubes. The results in each case are readily explained by a reference to the peculiarities of the circulation in each of these important organs—the lung, the liver, and the kidney. Without such a reference the facts are utterly unintelligible.

If there be anyone who doubts whether the retrograde action of a block to the circulation can be so far-reaching as I have described it to be, he has only to be reminded of the undoubted fact that an obstructive valvular disease on the left side of the heart may affect the most remote parts of the circulatory system. Thus the impediment resulting from a

defective mitral valve may extend backwards through the pulmonary capillaries to the systemic veins and capillaries, causing anasarca of the feet, &c.; it may extend through the capillaries within the liver to the portal vein and its capillary origin, causing enlargement of the liver and ascites; and through the intertubular renal capillaries to the Malpighian capillaries, causing albuminuria. It is manifest that neither extreme distance from the seat of obstruction, nor the intervention of two successive sets of capillaries, will prevent that retrograde passive engorgement of vessels which results from a block in the course of the circulating current.

In a future chapter I shall endeavour to show that a careful study of the hydraulics of the circulation will lead us to the establishment of some simple, yet most useful, guiding principles for the employment of blood-letting, general and local, and for the use of hot and cold applications and counter-irritants in the treatment of inflammation.

CHAPTER V.

THE ACTION OF BLOOD-LETTING, HEAT, COLD, AND IRRITANTS, IN THE TREATMENT OF DISEASE.[1]

Venesection—Changes of Fashion in regard to it—The main use of it is to relieve Over-distension of the Venous System—Typical Case—Dr. John Reid's Experiments on Dogs—The Cases in which Venesection is most beneficial are those in which there is Impeded Circulation through the Lungs, including the Collapse Stage of Cholera—A Feeble Pulse does not contraindicate it—Local Bleeding, its Action in Relieving the Pain of Pleurisy, &c.—Hot Fomentations, Poultices, &c. act by diverting Blood to the Surface—Marvellous Effect of Hot Saline Injection into the Veins during the Collapse Stage of Cholera—Harvey's Experiment with Chilled Blood—Hot and Cold Applications for different forms of Headache—Dry Cupping—Cold Applications—Counter-irritants—Summary.

I PURPOSE in this chapter to direct attention to certain general principles which may serve as guides for the employment of remedies which act by altering the amount and the distribution of blood in the vessels—I mean blood-letting, general and local; hot and cold applications; and counter-irritants.

And, first, I will speak of venesection. This powerful remedy, which in past times has been much employed, and in many cases, apparently, with excellent results, has, during the last forty years, fallen into disrepute. It is not now my intention to discuss the question whether this disuse of a once popular, and always a powerful, method of cure, has been the result of a change of type in inflammatory disease. This question has been very ably argued, and answered in the negative, by Dr. Hughes Bennett[2] and by Dr. Markham.[3] Sir Thomas Watson, who, in the fourth edition of his 'Lectures,' had expressed his opinion 'that there are waves of time through

[1] *British Medical Journal*, Nov. 7, 1868.
[2] *The Principles and Practice of Medicine.*
[3] *Bleeding and Change of Type in Disease.*

which the sthenic and the asthenic characters of disease prevail in succession, and that we are now living amid one of its adynamic phases,' has, with characteristic candour, in a published letter addressed to Dr. Markham, and in the last edition of his 'Lectures,' expressed his suspicion that, in the sentence above quoted, he ought to have spoken of successive waves of *opinion* rather than of *time*.

It appears, then, that, while medical opinions and medical practice have fluctuated, the diseased state which we call inflammation, and the constitutions of our patients, have remained essentially unchanged. Great errors have been committed in past times by excess in bleeding, and now the tendency is to the opposite extreme—to neglect venesection entirely, even in cases which admit of unquestionable relief from this practice. We are now suffering from an epidemic of *hæmatophobia*. A fashionable and very eminent physician said to me lately, 'It is so long since I have prescribed leeches, that I have forgotten the Latin name for a leech!'

This irrational rushing from one extreme to another under the influence of a fashionable impulse, this drifting upon the waves of opinion without rudder or compass, is equally discreditable to us as a scientific profession and disadvantageous to our clients. It is well worth an effort to avoid the scandal for the future.

It may be laid down as a general principle that the object of blood-letting, whether general or local, is to abstract blood, either directly or indirectly, from vessels which are overfull, and to promote its more equal diffusion through the system. And, more particularly, the main object of venesection is, to lessen over-distension of the venous system, under which term I include all the vessels that contain dark blood, the systemic veins, the right cavities of the heart, and the pulmonary artery. It will be found, on a careful inquiry, that all those cases in which the most striking relief has been afforded by venesection are characterised by an impeded circulation through the lungs, and a consequent distension of the venous system. This proposition has been advocated and illustrated with much ability by Dr. Markham in the treatise already referred to.

I will relate, in a few words, a typical case. At the end of

September 1866, during my visit to the hospital, one of the sisters came to tell me that a woman who had just been admitted into Twining Ward was dying. The patient had chronic renal disease, considerable anasarca, with the physical signs of engorgement of the lungs and of liquid in the left pleura. She was gasping for breath in great agony; the face was blue; the eyeballs prominent and watery; the jugulars much distended. She was apparently dying from over-distension of the right side of the heart. I asked Dr. Fenn, the house-physician, to open the jugular vein. About ten ounces of blood escaped. As the blood flowed, the distension of the veins was seen to subside; the breathing became easier before the blood had ceased to flow; in ten minutes, the dyspnœa had passed away, and it did not return. She died three weeks afterwards, gradually worn out by the results of advanced renal degeneration.

Dr. John Reid [1] proved, by experiments on dogs which were killed by various methods, but all involving apnœa and a consequent blood-stasis in the lungs, that opening the jugular vein, by lessening the distension of the right side of the heart, has the effect of increasing its contractile power, and so assisting the circulation.

We shall find that this principle is applicable to nearly all those cases in which pure empiricism has led to the practice of venesection, with apparently the most satisfactory results. These cases include some forms of inflammatory disease, and others of a non-inflammatory nature. They comprise cases of pneumonia, pleurisy, and bronchitis, in which the respiratory capacity of the lung has been suddenly and greatly reduced below the requirements of the blood to be aërated, when, consequently, there is a continual tendency to a serous effusion into the bronchi of the healthy lung-tissue, and cases of apnœa, the secondary result of brain disorder—in other words, some cases of apoplectic and of epileptic coma, in which the true object of venesection is not to arrest cerebral hæmorrhage or to lessen cerebral congestion, but to relieve the oppressed right

[1] 'On the Effects of Venesection in Renewing and Increasing the Heart's Action under Certain Circumstances.' (*Physiological, Anatomical, and Pathological Researches.*)

heart, and so to help on the circulation, first through the lungs, and then through the systemic arteries.

And here it is important to observe that a full and a strong pulse, which, in past times, has been looked upon as an indication for the practice of venesection, is rarely present in those cases in which the practice is known to have been most successful. Obviously, the true indication for venesection is not fulness of the *arteries*, but overfulness of the *veins*; and, inasmuch as this overfulness of the veins is a result of an impeded transit of blood from the venous into the arterial system, it is manifest that the arterial pulse will be the smallest and the feeblest in some of those cases in which venesection is most needed to relieve the over-distension of the venous system; while the effect of venesection will be to increase the volume and the power of the arterial pulse. In no class of cases has this been more strikingly shown than in some well-authenticated cases of cholera collapse, in which venesection has been attended with the happiest results. In illustration of this, I may quote the following striking case, which has been recorded by Sir Ranald Martin :[1]—

'On visiting my hospital in the morning, the European farrier-major was reported to be dying of cholera. His appearance was surprisingly altered; his respiration was oppressed; the countenance sunk and livid; the circulation flagging in the extremities. I opened a vein in each arm, but it was long ere I could obtain anything but trickling of dark treacly matter. At length the blood flowed, and by degrees its darkness was exchanged for more of the hue of nature. The farrier was not of robust health; but I bled him largely, when he who, but a moment before, I thought a dying man, stood up and exclaimed, "Sir, you have made a new man of me." He is still alive and well.'

The reports of the Indian practitioners abound in cases like this, showing, as the immediate results of venesection, a relief from dyspnœa and cardiac distress, and an increase in the volume and force of the arterial pulse, while the blood, which had nearly stagnated in the veins, and had consequently

[1] *The Influence of Tropical Climates on European Constitutions*, 6th ed., p. 349.

become dark and viscid, quickly assumed its natural colour and consistency, when the disengorgement of the oppressed right heart had increased the freedom of the circulation.

Bell makes the following statement :[1] 'The effect of blood-letting would, indeed, sometimes appear almost miraculous. A patient will be brought in a cot, unable to move a limb, and, but that he can speak and breathe, having the character, both to touch and sight, of a corpse ; yet will he, by free venesection alone, be rendered, in the course of half an hour, able to walk home with his friends.'

Rogers gives the following description of the effects of venesection by a medical man who was himself the patient : 'There was a sensation, which I am at a loss to describe, as if my heart was ceasing to beat, and a dread of suffocation ; this sensation was instantly relieved by bleeding, and I recovered immediately.'[2]

When we come to the subject of cholera, I shall prove to demonstration that, during the collapse stage of that disease, the right cavities of the heart and the systemic veins are over-distended in consequence of an impediment to the flow of blood through the lungs. Here I would say emphatically that I look upon venesection, not as a cure for pneumonia, or pleurisy, or bronchitis, or for apoplectic or epileptic coma, or for any of the various forms of apnœa, or for the collapse of cholera, but only as a means of mitigating one of the accidents of those various morbid states—namely, a paralysing over-distension of the right heart and the whole systemic venous system. If we can satisfy ourselves that this state of the venous system exists to a degree that perilously interferes with the onward flow of blood, then we need not be deterred from having recourse to venesection by the general feebleness of the patient or by the special weakness of the arterial pulse. The object of venesection in all these cases is, by abstracting a certain quantity of nearly stagnant venous blood, to promote a more rapid and a more abundant supply of arterial blood to the tissues ; and, amongst other tissues, to the muscular walls

[1] *Treatise on Cholera Asphyxia*, p. 118.

[2] *Reports on Asiatic Cholera in Regiments of the Madras Army*, by Samuel Rogers, p. 259.

of the heart itself. For it should be borne in mind that an impeded circulation through the lungs necessarily involves a defective blood-supply to the coronary arteries, and a consequent loss of muscular power in the heart's walls, quite apart from the paralysing over-distension of the right cavities.

When an obstruction in the larynx threatens fatal apnœa, in considering the question of tracheotomy, we take little note of what may have been the patient's previous condition of health and strength. The main question for consideration is, whether an opening in the trachea will remove the state of apnœa and the consequent asphyxia; and we act accordingly. So, with regard to venesection, while moving blood is the life of the body as a whole, and of each particular part, stagnant blood is death, whether general or local. If, then, by the withdrawal of blood from overgorged vessels, we can promote the movement of that which remains in the system, we effect a renewal of life, and we afford a physical illustration of the great doctrine that life may be lost by saving it, and saved by losing it.

These being, as I conceive, the true principles which should guide us in the practice of venesection, we see plainly the mischievous absurdity of the practice, which formerly prevailed, of bleeding a patient before a surgical operation, in order to prevent subsequent inflammation, and of bleeding persons in health periodically at certain seasons, to obviate a possible tendency to disease.

Local Bleeding.—I have long been convinced, by observation and experience, that local bleeding, by leeches or by cupping, is of great use in cases of painful acute pleurisy, pericarditis, and peritonitis, in some acute inflammatory cases of Bright's disease, and in some inflammations of the eye and of the brain. The relief from pain and distress, especially in the early stages of acute inflammation of the serous membranes, is so manifest that it can scarcely be disputed, even by the most sceptical. The relief thus afforded has received different explanations. Some practitioners maintain that leeching and cupping are only forms of counter-irritation, and that the abstraction of blood is unnecessary and useless, if not actually injurious. Others affirm that local bleeding is useful only where there is a *capillary connection* between the skin and the

tissues which are inflamed. But this explanation is obviously not applicable to some of the cases in which local bleeding is most indisputably useful; for instance, to inflammation of the eyeball, of the pericardium, and of the kidney. What, then, is the *modus operandi* of local bleeding in these cases? It appears to me to admit of a most satisfactory explanation through the *arterial connection* between the skin and the parts beneath. Take, for instance, the case of pericarditis. The internal mammary artery supplies the pericardium and the integuments over the heart. By the application of leeches over the heart, we abstract blood from the integumentary branches of the internal mammary artery, and in the same proportion we divert blood from the deeper pericardial branches. So, when leeches are applied near an inflamed eye, blood is drawn from the superficial branches of the ophthalmic artery, and diverted from the deeper ocular branches.

Let me illustrate this principle by some very simple and obvious analogies. When my opposite neighbour, Mr. Poole, exhibits his well-known illumination, he so depletes the main gas artery of the street, that the chandelier in my dining-room burns with a faint and feeble light. Mr. Poole obviously takes no gas directly from my pipes—there is no immediate 'capillary communication' between my pipes and his—yet, by his rapid consumption, he lessens the pressure in my main pipe and so diverts the gas from my burners. My chandelier, therefore, is the analogue of the inflamed pericardium when leeches are drawing blood through the skin. This is literally a case of inflammation being mitigated by local depletion.

I was giving this illustration to an intelligent non-medical friend, and he said: 'I can give you another instance. I have a long pipe from a cistern for watering my garden; at the end of the pipe there is a tap for watering the ferns; nearer the cistern, there is another tap for supplying the general surface of the garden. Sometimes, when I am watering the ferns, I find a sudden diminution of the stream, and I know that my sister has turned on the other tap for watering the garden.' Here, again, the ferns are in the position of the inflamed pericardium.

I am indebted to another friend for the following illustra-

tion. A spring on a neighbouring height supplies a house with water, and on its way there a branch pipe goes off to a fountain in the garden. When water is being drawn off through a large tap in the kitchen, the fountain-jet is much reduced in height. Here, again, the fountain represents the inflamed pericardium.

The same explanation applies to all the cases in which experience has shown that local bleeding is beneficial. It has been denied that cupping or leeching the loins can help an inflamed kidney more 'than if the blood had been taken from the arm or from the nape of the neck.' But this surely is a mistake. The lumbar arteries, which supply the integuments of the loins, arise from the abdominal aorta close by the renal arteries; and, when leeches or cupping-glasses draw blood through the skin of the back, it is certain that the diminished pressure within the lumbar arteries will divert a certain quantity of blood from the neighbouring renal arteries. The principle holds here precisely as in the case of the mammary artery and the pericardium. The blood will as surely take the course indicated by diminished pressure within the vessels, as the water in a pump will follow the rising piston, and the diminution of pressure in a main artery will be greatest near the point where the blood is being rapidly withdrawn through one of the branches. It may be thought that the quantity of blood thus diverted is very small; and so, in the case of venesection in the arm or neck, how slender is the stream of blood which escapes from the opening in the vein compared with the torrents which are rushing through the great venæ cavæ into the right side of the heart; and yet how decidedly and promptly does this diverted current lessen the distension of the whole venous system. The effect is to remove that proverbial last straw which is threatening to break the camel's back.

If the explanation which I have given of the influence of venesection and of local bleeding be the true one, then it will be seen that, although venesection would, by lessening the amount of blood in the body, and by enfeebling the heart's action, tend to diminish the flow of blood to an inflamed pleura, or pericardium, or kidney, yet any relief thus obtained

would be purchased by a much greater expenditure of blood than when the blood is drawn from vessels near the seat of inflammation.

Hot fomentations and poultices, and coverings of *cotton-wool and oil-silk*, have a soothing and beneficial influence in many cases of inflamed serous membranes, pericarditis, pleurisy, peritonitis. What is the operation of these remedies? Their action is very similar to that of local bleeding. The warmth relaxes the subcutaneous arteries. The skin, therefore, receives a larger supply of blood; and in the same degree blood is diverted from the vessels of the inflamed serous membrane. Then, too, there is a sort of depletion from the full cutaneous capillaries by the free local sweating which the warmth occasions.

The most marvellous illustration of the relaxing effect of warmth upon the arteries is afforded by the results of a hot injection into the veins of a patient in cholera collapse. The hot liquid (usually at 110° Fahr.), passing into the pulmonary artery, is directly applied to the inner surface of the contracted arterioles, whose spasm being thus relaxed, the blood passes freely on, and the state of collapse is for the time removed. The injection is a form of hot fomentation, but with this important difference, that the fomentation is applied directly to the inner surface of each small artery, and not through the integuments to their outer surface.

The faintness which is not unfrequently induced by a hot bath is a result of the relaxing effect of the warmth upon the subcutaneous vessels. There is, consequently, a copious afflux of blood to the skin, and a correspondingly diminished supply to the deeper parts, including the brain. Now, it is remarkable that the pulse of a patient in cholera collapse is often rendered fuller and stronger by a hot bath. The venous blood returning to the heart has its temperature raised; and, in proportion to this elevation of temperature, it will tend to relax the arterial contraction in the lungs, and thus give freedom to the whole circulation. The action of the warmed blood will be similar to that of the hot injection into the veins, but less powerful in proportion as the temperature is less elevated. When bleeding was a common practice in India, it was not unfrequently

found that while, during extreme collapse, the circulation was so much obstructed that no blood flowed from a cut vein, immersion in a hot bath so helped on the circulation, that the blood soon flowed from the vein with comparative freedom, and with speedy relief to the patient. It is very noteworthy that the simultaneous application of two methods of treatment, the hot bath and venesection, both of which singly, and still more when combined, tend in ordinary circumstances to cause faintness, should give increased volume and force to the arterial pulse of a patient in cholera collapse. A convincing proof is thereby afforded of the essential difference between the collapse of cholera and the collapse of ordinary exhaustion, whether the result of cardiac weakness or loss of blood-constituents. The influence of the warmed blood in promoting the circulation is the exact counterpart of an observation of Harvey's, to the effect that, when the fillet is removed from the arm after venesection, the blood, which had stagnated in the veins and become chilled, as it returns to the heart, carries with it a sensation of cold; and 'fainting frequently supervenes even in robust subjects.'[1] Harvey also noticed similar results from putting a bandage on the arm, so as to make the veins swell; immersing the arm in very cold water or snow; then suddenly removing the bandage, and allowing the chilled blood to flow back to the heart.[2] We now believe that the temporary check to the circulation after such an experiment is due to contraction of the minute pulmonary arteries excited by the chilled blood.

And now to return to the action and uses of hot fomentations. We find that, in some cases of severe throbbing headache, no remedy affords so much relief as the continuous application of a sponge wrung out of hot water to the forehead and temples while the head is kept somewhat raised. The warmth relaxes the superficial arteries; there is, consequently, a partial diversion of blood from the internal carotids through the external carotids to their branches beneath the skin. In some cases, headache is relieved by immersing the

[1] *On the Motion of the Heart and Blood*, Sydenham Society Translation, p. 58.

[2] *Ibid.*, p. 138.

hands or the feet in warm water. The arteries are relaxed, and blood is thus diverted from the head. On the other hand, the effect of putting the feet and legs in cold water is to cause 'a determination of blood to the head;' and we find that the headache of anæmia and exhaustion is best relieved by keeping the head low while cold is applied to the forehead and temples. Thus, the superficial vessels being contracted by the cold, more blood is diverted through the intracranial arteries to the anæmic brain.

Dry cupping acts in a somewhat similar way to hot fomentations. It draws an abundance of blood through the arteries into the subcutaneous capillaries, which, when the cups are removed, returns through the veins to the heart. In order that dry cupping may be most effectual, each cup should be removed as soon as the vessels beneath are well filled, and then it should be reapplied. The object is first to draw the blood through the arteries into the capillaries; then to allow it quickly to return by the veins; and not to keep it stagnating in the capillaries, which will happen if the glass be retained long on one spot. Another point is not to draw the blood into the skin with sufficient force to cause extravasation, the effect of which will be to impede the circulation through the skin, and so to divert more blood into the inflamed tissues beneath. The sole object of dry cupping, be it remembered, is not to *irritate* the skin, but to draw blood rapidly from the arteries, and as rapidly to transmit it through the capillaries to the veins, in its backward course to the heart.

Cold applications are employed not merely to subdue inflammation of the skin, where they act by contracting the vessels, and so lessening the afflux of blood to the inflamed tissues; but I believe that it is now a common practice in Germany to apply ice to the chest in cases of pericarditis. What is to be said of this practice? In my judgment, the practice is attended with much risk. The continuous application of cold to the surface of course causes contraction of the subcutaneous arteries. The secondary results of this may be diverse in different cases. Through the nervous system there may be a sympathetic contraction of distant arteries. The arteries of the left hand may be thus excited to contract while

the right hand is immersed in iced water. Thus pulmonary hæmorrhage may sometimes be arrested through the sympathetic contraction of the minute arteries of the lung, excited by the application of cold to the surface of the chest; and a vulgar remedy for epistaxis is a cold iron key applied to the skin of the back. On the other hand, the contraction of the superficial vessels under the influence of cold may drive more blood into deeper and distant parts. Thus, when the inmate of a Turkish bath is rendered faint in consequence of the large afflux of blood to the overheated skin, and a correspondingly deficient supply to the brain, a cold douche contracts the cutaneous vessels; then instantly the brain gets a larger supply of blood, and the feeling of faintness is removed. A temporary removal to the cooling-room has the same restorative influence; the cutaneous vessels contract, and more blood is diverted to the brain. So the six lights of a gas chandelier may have been burning steadily, without noise or smoke; but if now three burners are suddenly turned off, the other three, receiving an increased pressure of gas, will often begin to whistle and smoke. The same hydraulic and pneumatic principles are applicable to all these phenomena; and a consideration of these facts suggests that the application of cold to the surface, in cases of inflammation of the deeper tissues, is a remedy of doubtful and possibly of dangerous tendency.

Counter-irritants.—In my judgment, a very common mistake is that of applying strong counter-irritants, so as to inflame the skin, in the early stage of acute inflammation of the viscera or their investing membranes. The result is, that another inflammation, with its attendant pain and febrile excitement, is added to the primary disease. The discomfort resulting from this may be so great that the patient will think less of his original malady, which may, nevertheless, have been aggravated by the cutaneous inflammation. It should be borne in mind that the circulation through the vessels of an inflamed part is more or less impeded, more or less stagnant. If, then, in a case of acute pleurisy or pericarditis, we excite inflammation in the skin over the chest, the result is the opposite to that which is brought about by local bleeding, by dry cupping, and by hot fomentations; for, whereas by these means we

divert blood from the deeper tissues to the surface, the effect of an inflamed patch of skin is, through the arterial connexions, to throw back the blood upon the parts beneath. In applying irritants to the skin in the early stage of acute internal inflammation, care should be taken not to allow the effect to pass beyond the stage of active engorgement of the vessels; it should stop short of capillary stasis and inflammation. I have already said that local bleeding has been supposed to operate only as a form of counter-irritation. It would be nearer the truth to affirm that the so-called counter-irritants act after the manner of local bleeding, by diverting blood from the inflamed parts beneath. Vesication by means of cantharides may sometimes be useful, by the depletory serous effusion from the vessels.

Let me now gather up into a few sentences the general principles which I have endeavoured to establish.

The object of blood-letting is to lessen hyperæmia of certain parts of the vascular system.

Venesection is adapted for lessening engorgement of the venous system, which is usually a result of an impeded circulation through the lungs and left heart. When there are manifest signs of engorgement of the veins and obstruction in the lungs, a feeble arterial pulse does not contraindicate venesection; and there is no inconsistency in combining the practice of venesection with the administration of stimulants.

Local bleeding, by leeches or by cupping, is useful in many cases of inflammation. The bleeding acts by diverting blood through the superficial arteries from the deeper arteries which supply the inflamed parts.

Warm baths, fomentations, poultices, and dry cupping, act in an analogous way to local bleeding, but without actually removing the blood from the system.

Cold contracts the vessels to which it is immediately applied. The result of this may be a sympathetic contraction of distant and deeper vessels, or a driving in of blood to deeper parts. Cold to the surface, therefore, is an uncertain remedy in cases of internal inflammation.

The application of strong irritants, so as to inflame the

skin, in the early stage of acute internal inflammations, is a distressing and often a mischievous practice.

As a general rule, in cases of internal inflammation, those local remedies are the most efficacious which are the most painless, and which quicken in the greatest degree the cutaneous circulation.

Most of the general principles—physiological, pathological, and therapeutical—which we have discussed in the four preceding chapters will be found to throw light upon a great variety of morbid processes, to some of which, and especially to the collapse stage of cholera, I have already made repeated reference. Such cross-references, as they may be called, from general principles to special pathological states, and again from particular morbid conditions to the facts and laws of physiology, while of necessity involving some repetitions, can yet be made both interesting and instructive to the student of pathology, and will greatly assist him to obtain a many-sided view of disease. This method of studying pathological phenomena will, I trust, receive a practical illustration in the following chapter, in which I shall set forth and discuss in detail the symptoms of epidemic cholera, and then, guided by some of the general principles which we have before passed in review, I hope to carry my readers with me while I propound and endeavour to establish a theory which shall be acknowledged to be consistent with all the known facts of the disease, and which will also serve as a guide to a rational and trustworthy principle of treatment.

CHAPTER VI.

THE PATHOLOGY AND TREATMENT OF EPIDEMIC CHOLERA.

Prologue—Sec. I. The Relation between the Symptoms of Collapse and the loss of Liquid by Vomiting and Purging—Sec. II. The Symptoms of Collapse are not such as an excessive Drain of Liquid from the Blood would be likely to give rise to—Sec. III. The Effect of Various and Opposite Modes of Treatment on the Symptoms of Collapse affords no Support to the Theory that a Drain of Liquid from the Blood is the essential or the chief cause of that Condition—Sec. IV. The Mode of Communication of Cholera—Sec. V. Statement of Facts in Support of the Theory that the Poison—the Infecting Material of Cholera—enters the Circulation before it gives rise to the Characteristic Gastro-intestinal Symptoms—Sec. VI. The Pathology of Choleraic Collapse—Sec. VII. The Physical and Chemical Characters of the Blood—Sec. VIII. The Physical and Chemical Characters of the Intestinal Discharges—Sec. IX. The Stage of Reaction with or without Consecutive Fever—Sec. X. Prognosis—Sec. XI. The Treatment of Cholera and Choleraic Diarrhœa.

PROLOGUE.

More than twenty years ago, in 1866, I published a small book entitled 'Notes on Cholera.' In that volume I endeavoured to compress within a moderate space a statement of the facts and arguments in support of a theory of choleraic collapse, which had been set forth at greater length in a volume 'On Epidemic Diarrhœa and Cholera,' which I published in 1855.

I ventured to dedicate the 'Notes on Cholera' to Sir Thomas (then Dr.) Watson, and after a while I received from him the following letter:

'16 Henrietta Street, Cavendish Square, W.:
'Jan. 24, 1866.

'Dear Dr. Johnson,—If I have seemed tardy in acknowledging your gift of a copy of your little book on cholera, it has been because I wished to read it thoroughly and leisurely

before I thanked you for it, and there have arisen a thousand impediments which *you* will be at no loss to imagine.

'At length I have accomplished my task, and I am bound to say that I find no flaw in your reasonings. Your position is admirably well expounded and sustained, and the pathology that you adopt is rational and plausible in the best sense of that word. It seems also to be justified by the results of your peculiar treatment of the disease, and by treatment that resembles yours.

'There is, however, one hitch in the matter, on which I must talk with you when we meet. Briefly it is this :

'Happily for myself I have had very few cases of cholera to treat, and I fervently hope I may never have another ; but I have had to deal, in the midst of cholera epidemics, with very many cases of diarrhœa, and these have always and readily yeilded, without subsequent ill-effects, to the common combination of chalk, astringents, and opiates.

'Now there is much ground for believing that many at least of these diarrhœas might, and would if let alone, have run on into unequivocal choleras. Indeed, such diarrhœas, occurring in cholera seasons, are held by most writers and practisers to *be* mild or incipient instances of cholera.

'The cholera so prevalent and fatal at Bilston in 1832 began to be checked as soon as, and no sooner than, diarrhœa dispensaries were established. All this seems adverse to your practice and doctrine. How do you meet the objection ?

'For the compliment you have paid me in dedicating to me your little treatise I am very sincerely obliged to you.

'Believe me, dear Dr. Johnson, ever truly yours,

'THOMAS WATSON.'

I have here reproduced this letter, *verbatum et literatum*, believing that the time will come, even if it has not already arrived, when it will be read with especial interest. It will serve to show with what scrupulous care and diligence the great physician read the book before venturing to pass judgment upon it. It also displays his hesitation and caution in not fully accepting my doctrine until the one objection which he felt and candidly stated had been satisfactorily met.

The statement of facts and the arguments which served to convince Sir Thomas Watson, and to remove the 'one hitch,' to which he had referred, are fully set forth in the following pages.

So great an interest did he feel in the subject that, notwithstanding the many calls upon his time (he was then the President of the Royal College of Physicians), he wrote a review of the book, which was published in the *Saturday Review*, June 2, 1866.

After giving, in his own inimitable style, a lucid summary of my theory of collapse, the reviewer went on to say: 'Surely this seems a reasonable theory. It is founded on a true analogy, it is consistent with the symptoms noticed during life and with the conditions discovered after death. We may, therefore, legitimately regard it, until fairly refuted, as a sound as well as a most ingenious and important theory. In truth it derives strong confirmation from the fact that it unlocks, like the right key, the whole of the pathological intricacies of the disease.'

Sir Thomas Watson then concluded his article in the following terms, which, by the favour of the author, I was permitted to copy from his manuscript: 'We need not abstain from professing our belief that the true pathology of this awful disease, the sequence and relation of its various phenomena, have now, for the first time, been determined and made known. And we congratulate the eminent physician who has done the world this great service on the firmness of purpose with which he has withstood the force of ridicule in high places, and upon the perseverance and sagacity which have enabled him to solve so neatly and thoroughly a very complex and deeply interesting problem. The more his facts and reasonings are scrutinised, the more widely and entirely will they, in our judgment, compel assent, and the greater will be the consequent salvage of human life.'

This decided expression of opinion on the part of the then head of the medical profession in this country, of one, too, who has been often spoken of as 'the greatest English physician of the present century'—remarkable alike for his calm judgment and his unswerving integrity—was not allowed to

appear in the pages of the *Saturday Review*, but in place of it the then editor substituted the following sentence: 'On this point, as on others, Dr. Johnson's facts and reasonings are well deserving of careful and impartial consideration.'

A careful and impartial consideration of my facts and reasonings by candid and competent judges is all that I ask, and not more than I have a right to expect.

The present chapter on the Pathology and Treatment of Cholera is not a mere reprint of my previous publications on the same subject, but it embodies the result of a careful revision of the main facts and reasonings which have reference to the nature and treatment of the disease, with much additional evidence in support of my doctrine, and a reply to such published criticisms and objections as have appeared to me to be worthy of notice.

SECTION I.

The Relation between the Symptoms of Collapse and the Loss of Liquid by Vomiting and Purging.

There are few diseases the treatment of which has been more influenced by pathological theories than that of cholera. The theory which, since the introduction of cholera into Europe in 1831–2, has gained almost universal acceptance is, that the worst symptoms of the disease are due to the drain of fluid from the blood; in consequence of which the blood becomes so thick that it stagnates in the small vessels, and thus the circulation is arrested. The practice which has been based upon this theory is to check the purging by opiates and astringents. Before attempting to give what I believe to be the true explanation of the symptoms, it seems desirable to set forth certain facts which are inconsistent with the commonly accepted theory.

First, then, let us inquire, *What is the relation between the symptoms of choleraic collapse and the loss of fluid by vomiting and purging?* If the symptoms of collapse are occasioned by a drain of fluid from the blood, there must, as a rule, be a direct relation between the degree of collapse and the amount of

liquid which escapes from the blood. Now, so far is this from being the case, that there are few writers of any note or authority upon the subject of cholera who do not either assert distinctly, or record facts from which the inference plainly follows, that not only is there no direct relation between the loss of liquid by vomiting and purging and the degree of collapse, but that these conditions often bear an inverse ratio to each other. In confirmation of this statement I will now quote some of the best known authors on the subject of cholera.

Dr. Edmund Parkes says: 'My cases bear out the observations of Scot, Jameson, Orton, Kennedy, Copland, and, in fact, almost all the English writers of reputation, that there is absolutely no ratio between these two classes of symptoms' (*i.e.* between the purging and vomiting and the symptoms of collapse) 'or that they appear even to observe an inverse ratio to each other. Thus, at a period of the case when the algide symptoms were most fully developed—viz. in the last five hours—the purging ceased; in the cases where the algide symptoms were prominent throughout, and which cases were consequently the most malignant and the most rapidly fatal, the passage of fluid from the intestines was oftentimes trivial in degree and shortened in the period of its occurrence.[1] In cases in which the vomiting and purging were excessive, the algide symptoms often came on slowly, and were less marked and deadly.'[2]

Dr. Parkes then, in illustration of these remarks, cites some cases in which the frequency of the vomiting and purging was quite out of proportion to the severity of the other symptoms. He afterwards states that 'it may be objected to observations of this kind, that the number of stools is, after all, no certain indication of the amount of fluid passed. This objection would be of weight in cases where the stools were not very different in number; but in some of these cases cited

[1] Dr. Sutton quotes this sentence from Dr. Parkes, and says that 'he cannot but agree with it' (*Ninth Report of the Medical Officer of the Privy Council*, p. 388). He also says that 'some of the worst cases of cholera, cases which seemed almost sure to prove fatal, had very little, and often not any, purging while in the hospital.'

[2] *Researches into the Pathology and Treatment of the Asiatic or Algide Cholera*, p. 79.

above we have two or four stools attended by more rapid death than twenty-five or twenty stools; and yet, in the first case, it would be impossible not to suppose the quantity of the fluid passed to be much below that of the second case. He adds: 'It may be confidently asserted that there is no one who has seen much of cholera who does not know that, exclusive of the mildest forms of the disease, a case with little vomiting and purging is more malignant and more rapidly fatal than one in which these are prominent symptoms.'

Dr. Parkes puts the following question (p. 90): 'May not the fluid passed into the canal but not ejected have borne some relation to the severity of the disease? To this a partial reply only can be given. Certainly after death in malignant cases there appeared to be a considerable quantity of the thick clotted fluid in the canal; it was almost impossible to measure this, but, judging from the eye, it seemed to vary, like the stools, without much reference to the severity of the case, and to be small in quantity in some of the most rapid cases, attended with little purging.'

With reference to the varieties in the general symptoms of cholera, Scot makes the following statement: 'A frequent variety, the worst of all, is that which is noted for the very slight commotion in the system; in which there is no vomiting, hardly any purging—perhaps only one or two loose stools—no perceptible spasm, no pain of any kind; a mortal coldness with arrest of the circulation[1] comes on from the beginning, and the patient dies without a struggle. This has frequently manifested itself as the prevailing type, and almost all die who are attacked by it.[2]

The testimony of Bell is to the same effect. He says: 'It has been found that the more violent the prominent symptoms are, the more likely is a cure to be effected; and that, when the disease is attended with rapid collapse, little or no vomiting or purging, and no spasm, the prognosis is very unfavourable.[3]

[1] We shall see hereafter that the words 'arrest of the circulation' are the expression of a fundamental fact.

[2] *Report on the Epidemic Cholera*, p. xxi.

[3] *Treatise on Cholera, Asphyxia, or Epidemic Cholera*, p. 128.

Orton,[1] in several passages of his essay, alludes to the fact that, in the worst forms of the disease, vomiting and purging are slight or quickly cease, or even do not occur at all.

Twining gives cases which illustrate the same general principle. Thus, he reports[2] the case of a gentleman who died nine hours after the commencement of urgent symptoms, in whom there were occasional slight efforts to vomit at intervals of half an hour, and he had only four stools from the commencement to the termination of the attack. And, in another part of his work,[3] he says: 'We often see patients vomiting violently for hours, and others purged profusely for several days without cholera (*i.e.* collapse) coming on.'

The authors above cited have all observed the disease in India; but the testimony of those who have witnessed it in Europe is in strict accordance with that of the Indian authorities. Thus, Magendie, describing cholera as he saw it in Paris in 1832, makes the following statement: 'Some patients have no evacuations, insomuch that one is obliged to excite them; for the evacuations, though associated with the disease, are not one of the most serious symptoms; and those patients who have had copious evacuations have been more easily treated than those who have had none. This is a remark which has been made by many physicians.'[4]

Mr. French states, among other reasons for believing that the intestinal discharges are salutary, that while 'cholera in its most intense form produces death instantly without discharges, all those who recover from its attack experience the peculiar discharges more or less.' And, again, in favour of the same view, he says, is 'the ultimate recovery of persons who have continued in a state of collapse for a considerable length of time, often extending to a period of three days, and who, in all instances, sustained enormous discharges.'[5]

Sir William Gull[6] cites the evidence of several practitioners

[1] *An Essay on the Epidemic Cholera of India.*

[2] *Clinical Illustration of the More Important Diseases of Bengal*, p. 10.

[3] *Ibid.* p. 37.

[4] *Leçons sur le Choléra Morbus*, p. 66.

[5] *The Nature of Cholera Investigated*, p. 22.

[6] *Report on the Morbid Anatomy and Pathology of Cholera*, published by the Royal College of Physicians in 1853, p. 136.

to the effect that, in many cases, the 'evacuations appeared to be wholly insufficient to account for the fatal collapse.' And one case which came under Sir William Gull's observation affords a striking illustration of the same principle. 'On a *post-mortem* examination, the large intestines contained healthy fæces; whilst in the upper two-thirds of the small intestine the mucous membrane presented the ordinary changes induced by the cholera process, and the rice-water effusion was abundant.' Sir William Gull adduces this case to show that '*cholera sicca*, in a strict sense, does not occur; for although the disease may be fatal without any evacuation, the intestines after death, in such cases, have been found to contain the rice-water fluid.' It can scarcely be doubted, however, that when, as in this case, the purging has been insufficient to remove the fæculent contents of the large intestine, the loss of fluid must have been out of all proportion less than in most cases in which recovery takes place. And Sir William Gull, in another part of his report,[1] admits that 'the intensity of the symptoms is often in no inconsiderable degree greater than can be accounted for by the amount of the effusion.'[2]

Mr. Macnamara is an uncompromising advocate of the anhydræmic theory of collapse, yet his own book contains evidence adverse to his doctrine. He publishes in an appendix a report on cholera by Dr. Bruce, of which he says: 'It is an additional proof of the value of many of the records contained in the "Proceedings of the Bengal Medical Board."' Dr. Bruce in this valuable report, after referring to some rapidly fatal cases in which the discharge had been very scanty, and to three others in which the collapse and sinking appeared to have a direct ratio to the amount of exudation and discharge, goes on to say:[3] 'It is still a favourite opinion with many that this always holds good in cholera, viz. that the amount of discharge and the collapse stand in the direct relation of cause and effect. If it could be established that such is the case, a

[1] *Report on the Morbid Anatomy and Pathology of Cholera*, p. 211.

[2] We shall find hereafter that a painful and paralysing over-distension of the intestines by the rapid pouring out of the choleraic secretions is sometimes the immediate cause of death.

[3] *A Treatise on Asiatic Cholera*, by C. Macnamara, p. 522.

great step would be gained towards the successful treatment of the disease, and much of the anxiety and uncertainty that attend it removed; but experience is opposed to such a conclusion, for who has not seen marked cases in every epidemic where there was neither purging nor vomiting sufficient to cause any degree of collapse, and yet those cases have sunk at least as rapidly as others where the discharges were profuse?'

Dr. Sutton, in his 'Comments on the Clinical Characters of Cholera in 1866,'[1] mentions a class of cases attended with profuse vomiting and purging, yet with very slight collapse, short reaction, and rapid convalescence; and goes on to say: 'In striking contrast with the above was another class of cases, where many of the symptoms were almost the exact opposite of those just given. There were very little purging and vomiting, and I may briefly state all the severe algide symptoms and the signs of collapse.'

With respect, then, to the question whether, as a rule, there is a direct relation between the loss of fluid by purging and the symptoms of collapse, we have a large amount of concurrent testimony to the effect that no such relationship exists.

On the other hand, it must, I think, be conceded that the evidence of there being, in the worst and most rapidly fatal cases, an inverse rather than a direct ratio between the degree of collapse and the loss of liquid by vomiting and purging, is entirely inconsistent with the hypothesis so commonly received and acted upon.

SECTION II.

The Symptoms of Collapse are not such as an Excessive Drain of Liquid from the Blood would be likely to give rise to.

With reference to the hypothesis that the characteristic symptoms of choleraic collapse are caused by the loss of the watery constituents of the blood, we have next to inquire

[1] *Ninth Report of the Medical Officer of the Privy Council*, pp. 415–16.

whether the symptoms of collapse are such as an excessive drain of fluid from the blood would be likely to produce.

Now, what is the condition of a patient who has suffered a profuse drain from the blood, whether of water alone or all the blood-constituents? What is the effect of a copious hæmorrhage, or of excessive purging, whether the result of disease or of medicine? Is not the condition of a patient who has been exhausted by such means of the nature of syncope? There are a small and frequent pulse, a pallid skin, dimness of sight, and vertigo: these symptoms being much increased by the erect posture; and, in extreme cases, the head cannot be raised from the pillow, even for a moment, without the occurrence of syncope. For a patient in this condition to walk or stand, or even to sit up, is simply impossible. Now there is something in the collapse of cholera which is essentially different from the mere exhaustion which leads to syncope. In fact, almost the only symptom which is common to the two conditions is the extreme smallness and feebleness of the pulse.

One great distinction consists in the remarkable blueness, coldness, and other symptoms indicating that during the collapse of cholera either the aëration of the blood is greatly interfered with, or its passage through the lungs is so much impeded as to cause engorgement of the systemic veins; while no such symptoms of obstructed circulation and respiration occur in ordinary cases of exhaustion from excessive purging.

Another great and obvious distinction is this: that whereas a patient exhausted by hæmorrhage or by a drain of fluid from the blood, and therfore verging on syncope, is unable to assume the erect posture without fainting, a patient in the collapse of cholera, whose skin is blue and icy cold and whose pulse is imperceptible or extremely small and feeble, is often able to stand up without becoming faint, and even to walk a distance which must require a considerable amount of muscular exertion.

This is a fact alluded to by several authors; and no one can have watched cases of the disease without having observed the surprising amount of muscular exertion of which even a cold and pulseless patient is capable. It is scarcely necessary

to quote authorities for this statement; but I will refer to one in illustration of what has been here advanced.

Scot says:[1] 'Instances are not wanting of patients being able to walk, and to perform many of their usual avocations, even after the circulation has been so much arrested that the pulse has not been discernible at the wrist.' The same author states, in another place, when speaking of the effects of blood-letting:[2] 'It is remarkable that, in a disease like cholera, syncope should be so rare a symptom.' And again:[3] 'During the progress of this disorder, when the nervous energy seems to be almost annihilated, and the functions of the heart and arteries to be abolished, this symptom (syncope) is yet very rarely observed.'

It is not denied that a choleraic patient may be suddenly killed by imprudent exertion; but death occurs in such cases, not from failure of the heart's action as in syncope, but from a complete arrest of the flow of blood through the lungs, as will hereafter be proved.

Another remarkable difference between the collapse of cholera and the exhaustion caused by an excessive drain of liquid from the blood consists in *the rapidity with which a patient often recovers from the former condition*. As an instance of this I may quote the following:[4] 'I have seen' (says Mr. Grainger) 'a man stand at his door on Wednesday, who on Monday was in perfect collapse.' And this observation, as Sir William Gull remarks, is in accordance with the experience of others.

With reference to this remarkable feature of cholera, Twining makes the following statement:[5] 'In cases not fatal, the progress of recovery is often almost as rapid as the accession of cholera; and if the disease be treated at the very onset, it is not uncommon to see a person well on the third day, after an attack of the worst symptoms, which had commenced with coldness and collapse, and who, if left without remedies, would probably have died in six or eight hours. *In*

[1] *Report on the Epidemic Cholera*, p. 24.
[2] *Ibid.*, p. 58.
[3] *Ibid.*, p. 28.
[4] Sir William Gull's *Report*, p. 135.
[5] *Clinical Illustrations of the More Important Diseases of Bengal*, p. 20.

these instances recovery seems almost as sudden and complete as in cases of patients who are resuscitated after suspension of animation from submersion in water.' It is scarcely necessary to insist upon the fact, that no such instances of rapid recovery from extreme prostration consequent on a drain of fluids from the blood are ever known to occur; nor, from the nature of things, is it possible that a great loss of blood-constituents can be restored with such extreme rapidity.

Dr. Sutton says:[1] 'It was instructive to notice how quickly a patient would pass from the condition of collapse into that of reaction, and that without the aid of any drugs. Patients were seen pulseless, livid, wakeful, with the choleraic voice, and one or two hours afterwards the lividity had been replaced by flushed cheeks, pulse had become distinct, tongue warm, and the choleraic voice replaced by busy delirium.'

The natural and obvious inference from such facts appears to be, that there is an essential difference between the condition of a patient who has been exhausted by a profuse drain from the blood, and that of one in collapse with cholera. It is, therefore, incumbent on those who maintain that choleraic collapse is due to the loss of fluid by the intestinal canal to explain, if they can, the remarkable differences which have here been pointed out between the symptoms of collapse and those of ordinary exhaustion and syncope.

SECTION III.

The Effect of Various and Opposite Modes of Treatment on the Symptoms of Collapse affords no support to the Theory that a Drain of Liquid from the Blood is the essential or the chief Cause of that Condition.

We have shown in the two previous sections that two classes of facts are inconsistent with the theory that the collapse of cholera is due to a drain of liquid from the blood. We have seen that in the most rapidly fatal cases there is no such direct relation between the degree of collapse and the amount of liquid discharged from the blood as must exist if the hypothesis in question were true. We have also seen that the

[1] *Ninth Report of the Medical Officer of the Privy Council*, p. 395.

symptoms of collapse differ essentially from those which an excessive drain of fluid from the blood is known to produce. We have now to inquire *whether the effect of various and opposite modes of treatment affords support to the theory that a drain of liquid from the blood is the essential or the chief cause of that condition.*

The Effects of Alcoholic Stimulants.—The condition of a patient in collapse—cold and pulseless and apparently exhausted—is one which naturally suggests the use of stimulants. Anyone who has witnessed the speedy improvement in the pulse and in the other symptoms which usually follows the administration of wine or brandy to a patient who is fainting from loss of blood or exhausted by excessive purging, might reasonably expect to obtain similar results from the same means in the collapse of cholera. Accordingly, stimulants have been given, and given freely and boldly; and the result has been a very general conviction that, in the stage of collapse, they are not only useless, but positively injurious. Again and again have I seen a patient grow colder, and his pulse diminish in volume and power, after a dose of brandy, and apparently as a direct result of the brandy. Sir William Gull[1] states that, 'although opium and diffusible stimuli—brandy, camphor, and ammonia—were useful at an early stage of the disease, as collapse set in they not only failed to produce any favourable result, but often aggravated the symptoms.'

Dr. Paine, who has given an admirable description of cholera and its treatment in 1832, writes thus of stimulants:[2] 'We have seen no benefit from their liberal use, and it is even doubtful whether they contribute much in any quantities. It requires the conviction of experience, however, to enable us to abstain from their use, and to resist the impulse to apply them to the dying spark.'

The very general conviction as to the worse than uselessness of alcoholic stimulants in the collapse of cholera is the more to be relied upon, inasmuch as it has been forced upon men's minds in opposition to preconceived notions and pre-

[1] *Report on the Morbid Anatomy and Pathology of Cholera*, p. 185.

[2] *Letters on Cholera Asphyxia as it has appeared in New York*, p. 42.

vailing theories. The action of stimulants in the collapse of cholera being obviously very different from their influence upon patients who have been exhausted by the loss of blood-constituents, we infer that exhaustion verging on syncope and choleraic collapse are pathological conditions essentially different; and this conclusion is confirmed in a most striking manner by the effect of other modes of treatment.

The Effect of Venesection on the Symptoms of Collapse.—It is scarcely necessary to assert that no sane practitioner would think of abstracting blood from a patient who has been reduced to a state bordering on syncope by those common sources of exhaustion which have before been referred to (p. 79). It is obvious that the loss of blood in such cases might be attended with perilous and even fatal results. But what has been the effect of venesection in not one or two, but in a large number of cases of cholera, and in the hands of many different practitioners? Has the effect of this treatment been such as to afford support to the theory that collapse results from loss of liquid, or has it been to add to the cumulative evidence which stands opposed to that theory? We will endeavour now to answer these questions.

It is in the writings of the older Indian practitioners, who were not deterred from the practice of venesection by the erroneous modern theory of collapse, that the largest amount of evidence is to be obtained as to the influence of blood-letting in cholera. Scot makes the following remarks on this subject:[1] 'The abstraction of blood, unless as an antispasmodic, is a remedy so little indicated by the usual symptoms of cholera, that its employment in the cure of this fatal disease has afforded a signal triumph to the medical art. It requires no common effort of reasoning or reflection to arrive at the conclusion that, when the powers of life appear to be depressed to the lowest degree, the pulsation of the heart all but extinct, the natural heat of the body gone, and the functions of the system suspended and incapable of being revived by the strongest stimulants, the abstraction of blood might yet prove a remedy against a train of symptoms so desperate. Few remedies,' he says, 'on a fair trial, have been more generally

[1] *Report on Epidemic Cholera*, p. lviii.

and unequivocally advocated than free blood-letting; and the most that has been urged against it is, that it is not always successful.' He then quotes reports from medical officers furnishing very striking testimony to the benefit of bleeding in cases of extreme collapse.

Annesley states (quoted by Scot, p. lx.): 'In place of syncope being produced by bleeding, in the cases which I have treated, the pulse has invariably improved, and the feelings of faintness and debility disappeared.'

Bell makes the following statement:[1] 'The effect of blood-letting would indeed sometimes appear almost miraculous. A patient will be brought in in a cot, unable to move a limb, and but that he can speak and breathe, having the character, both to touch and sight, of a corpse, yet will he, by free venesection alone, be rendered, in the course of half an hour, able to walk home with his friends.'

Rogers gives the following description of the effects of venesection by a medical man who was himself the patient:[2] 'There was a sensation which I am at a loss to describe, as if my heart was ceasing to beat, and a dread of suffocation; this sensation was instantly relieved by bleeding, and I recovered immediately.'

Dr. Parkes (p. 216) mentions the case of a man in pulseless collapse, with 'an intolerable constriction and tightness across the chest, and an intense weight and oppression at the heart. With much difficulty, and drop by drop, about ʒviij. of blood were taken away. The patient immediately felt easier; the constricted feeling across the chest for a time almost disappeared, and the pulse at the wrist became perceptible.' In less than half an hour the cardiac oppression began to return, and he died in six hours after the bleeding.

The following striking case is recorded by Sir Ranald Martin:[3] 'On visiting my hospital in the morning, the European farrier-major was reported to be dying of cholera.

[1] *Treatise on Cholera Asphyxia*, p. 118.

[2] *Reports on Asiatic Cholera in Regiments of the Madras Army*, by Samuel Rogers, p. 259.

[3] *The Influence of Tropical Climates on European Constitutions*, 6th ed. p. 349. This case is quoted for another purpose at p. 59.

I found that during the night he had been drained of all the fluid portion of his blood. His appearance was surprisingly altered; his respiration was oppressed; the countenance sunk and livid; the circulation flagging in the extremities. I opened a vein in each arm, but it was long ere I could obtain anything but trickling of dark treacly matter. At length the blood flowed; and by degrees its darkness was exchanged for more of the hue of nature. The farrier was not of robust health; but I bled him largely, when he, whom but a moment before I thought a dying man, stood up and exclaimed, "Sir, you have made a new man of me." He is still alive and well.'

Now, let me ask, is it possible to reconcile facts of this kind, and they might be multiplied indefinitely by quotations from the earlier Indian records, with the theory that the collapse of cholera results from a loss of the liquid constituents of the blood? If Sir R. Martin's hypothetical statement, that his patient 'had been drained of all the fluid portion of his blood,' were an accurate expression of facts, can we conceive it possible that he could have 'made a new man' of him by abstracting largely the blood which remained in the vessels? I maintain that the numerous well-authenticated instances of great and immediate and permanent relief by means of venesection in the collapse stage of cholera are utterly and hopelessly irreconcilable with the hypothesis in question.

So obviously inconsistent are these well-authenticated cases with the theory of collapse by loss of water, that the advocates of that theory are driven to deny the facts. Mr. Macnamara says of Sir Ranald Martin's [1] case that it is 'unique' (p. 375), and that venesection is now neither common nor successful. That Sir Ranald Martin's case is not unique is evident from the preceding quotations. Venesection is not now common because an erroneous but generally accepted theory condemns it, and even if practised it would not be likely to be successful because it would probably be combined with opiates, alcoholic

[1] It is not a little remarkable that Mr. Macnamara's friend, Dr. Bruce (Macnamara on *Cholera*, p. 525), declares that he found bleeding eminently useful in checking spasms, 'especially when the blood was drawn from the temporal artery.' To draw blood from the nearly empty arteries instead of from the over-distended veins is a practice not likely to be very successful.

stimulants, and other antagonistic methods of treatment. I shall hereafter show that the rapid relief which has often been afforded by venesection is rendered perfectly intelligible by a true theory of choleraic collapse.

The Effect of Purgatives.—If the symptoms of collapse were due to the drain of liquid from the blood, and its escape by the intestinal canal, it would seem to be impossible that the symptoms of collapse should pass away while the drain of liquid by vomiting and purging is continually going on. It would seem, too, that the action of purgatives during the state of collapse must increase the mortality. I do not here propose to consider the practical merits of the purgative plan of treatment. I wish only to refer to the indisputable fact that there are on record numerous well-authenticated instances of recovery from collapse, while the intestinal discharges were encouraged by repeated doses of emetic and purgative medicine. And, further, I challenge the advocates of the theory which I am endeavouring to refute to refer to a single case of recovery from collapse in which the intestinal discharges have not continued, in a greater or less degree, while the symptoms of collapse were passing off. If the theory in question had a basis of truth, the cessation of the intestinal discharges must always, and of necessity, precede recovery from collapse; whereas, on the contrary, the complete and final arrest of the discharges during the stage of collapse is a sign of fatal import.

During the early part of the cholera epidemic of 1849, all the cases of cholera admitted into King's College Hospital were treated by liberal doses of brandy and opium. Under this mode of treatment the mortality was very great. The treatment was then entirely changed: brandy and opium were discontinued; and large quantities of salt and water were administered. The effect of this treatment was to excite frequent vomiting, and certainly not to check the purging; and the result was a very much larger proportion of recoveries than under the previous mode of treatment. I had no share in conducting the treatment on that occasion; but I was greatly struck by the different effects of the two opposite modes of treatment. I was also deeply impressed by observing that,

during that epidemic, the arrest of the purging by opiates was in several instances *followed* by the worst symptoms of collapse; and a painful question arose in my mind, whether the collapse in such cases was not a direct result of the arrest of the purging.

At the commencement of the epidemic of 1854 I had arrived at the conclusion that the commonly received theory of choleraic collapse is erroneous. I had the chief charge of the hospital during the whole period of the epidemic; and I gave emetics and purgatives to all the patients who came under my care. I afterwards published full particulars of all my cases.[1] I am convinced that in many instances I gave an excessive quantity of castor-oil; yet the result was a mortality, to say the least, below the average mortality in cases of equal severity. The first seven cases of cholera with collapse that came under my care, most of them of a very severe type, recovered; and the rapid reaction which occurred in these cases, while the intestinal discharge continued, convinced me that these discharges were the means of recovery and not the essential cause of collapse. During that epidemic, many cases of choleraic diarrhœa came under my observation—cases in which there were vomiting, bilious purging, and cramps. These were all treated by castor-oil, without opiates. They all recovered; and not one case so treated passed into collapse. Several of the medical assistants, pupils, and nurses, and a considerable number of patients who were in the hospital for other diseases, had the premonitory symptoms of cholera. All were treated in the same way, and all recovered. In contrast with this most satisfactory result stands the fact that, during the previous epidemic of 1849, several inmates of the hospital, nurses and patients, having been seized with choleraic symptoms, and being promptly treated by opiates, passed into a state of collapse and died.

I see no way in which the facts here stated can be reconciled with the hypothesis that the worst symptoms of cholera result from the loss of liquid, and that the main object of treatment is to check the vomiting and purging.

[1] *On Epidemic Diarrhœa and Cholera: their Pathology and Treatment.* With a Record of Cases. Longmans and Co., 1855.

The Effect of Injecting Hot Saline Solutions into the Veins.—It is well known that the injection of a hot saline fluid into the veins during the collapse of cholera has often been followed by great temporary relief. The pulse improves; the temperature rises; the countenance becomes natural; the voice recovers its strength; and, in short, all the worst symptoms speedily disappear—usually, however, to return with all their former severity within a very short time. This method of treatment appears to have been first resorted to by the late Dr. Latta, of Leith, with the view of restoring to the blood a liquid the loss of which he considered to be the main cause of death. The immediate results of the operation, as performed by Dr. Latta in six cases, are described in enthusiastic language by Dr. Lewins in the *Medical Gazette*,[1] but curiously enough he does not mention the ultimate result in a single case. The late Dr. Mackintosh, of Edinburgh, during the summer of 1832, injected the veins of 156 patients, of whom only twenty-five recovered. It was a very natural conclusion, since all the symptoms of collapse speedily disappear after the injection of a certain quantity of liquid into the veins, that the symptoms which previously existed must have resulted from the loss of a liquid similar in character to that which the operation restores to the blood.[2] I believe, however, that the true explanation of the manner in which the hot saline injections afford the surprising temporary relief which they are acknowledged to have done, has been missed; and that, rightly interpreted, the results of this experiment afford as little support to the hypothesis that collapse depends on loss of fluid as do the effects of venesection and of other modes of treatment to which reference has already been made.

In a future section I will give what I believe to be the true pathology of collapse; and then I shall explain the *modus operandi* of the saline injection into the veins.

Before attempting to give what I believe to be the correct

[1] Vol. x. p. 257.

[2] It is noteworthy that in a large proportion of cases severe rigors have followed the venous injection. This symptom may perhaps be due to the sudden transmission into the arteries of a large amount of morbid blood which had before been accumulated in the veins. (See the chapter *On the Pathology of Rigors*.)

interpretation of the symptoms of cholera, I have thought it desirable to direct attention to some of the facts and arguments which are opposed to the commonly received theory. There is so much of the superficial appearance of truth in the theory which explains the symptoms of collapse by the loss of the watery portion of the blood, that the practice of giving opium and other astringents to arrest the intestinal discharges will continue more or less, in spite of failure and disappointment, until it can be clearly shown that the state of collapse has an entirely different origin and cause from that which the theory in question assumes.

SECTION IV.

The Mode of Communication of Cholera.

Cholera is very generally believed to be a disease which, like enteric fever, is under certain conditions capable of being conveyed from the sick to the healthy.

Amongst other publications on this subject, Dr. Baly's *Royal College of Physicians' Report*, Dr. William Budd's series of papers under the signature of 'Common Sense' in the *Association Journal*, 1854–5, and Dr. Snow's treatise *On the Mode of Communication of Cholera*, 2nd ed., 1855, contain abundant proofs that cholera is, as Dr. Budd expresses it, 'a catching disease.'

There is good reason to believe that the infecting material of cholera is contained mainly, if not exclusively, in the gastro-intestinal discharges. In this respect there is a close resemblance between cholera and enteric fever.

The choleraic infection may enter the system either with the air through the lungs or with the food, water, or other liquids through the alimentary canal. In proof that the infection may be conveyed by the air, Dr. Budd gives,[1] amongst other conclusive pieces of evidence, the detailed history of an outbreak of cholera in a large workhouse, where the spread of the disease was clearly traced to the gaseous emanations

[1] *Association Journal*, 1854, p. 974.

from ill-constructed privies. The food and water were the same for the entire establishment, but while numerous inmates, who, from various causes, were debarred access to the infected privies, entirely escaped the disease, there was a frightful mortality amongst those who resorted to that source of infection.

Another instance in which the infection must have been received through the air is afforded by the case of a pilot and his assistant, who, in 1866, took the disease from the infected steamship 'England,' without going on board. These men went out in an open boat to pilot the ship into Halifax. Hearing that there was cholera on board, they remained in their boat, which was towed by a ten-fathom rope fixed to the stern of the ship. In this way the ship, with the boat in tow, was conducted to her place of anchor.[1] Neither of the men went on board, but both were seized with cholera on the day after they landed, and both communicated the disease to several members of their respective families at their homes near Halifax, where no case of cholera had occurred for years before. In these cases it is pretty evident that the infection was conveyed to the boatmen by the gaseous emanations from the choleraic discharges which were escaping from the stern of the steamship.

In connection with the subject of contaminated water as the vehicle of the choleraic poison, the name of the late Dr. Snow deserves most honourable mention. It is to his sagacious and persevering efforts, carried on in spite of the opposition and ridicule which an original research inevitably provokes, that we are mainly indebted for our knowledge of this important agency in the spread of cholera.

One of the most remarkable instances of a local outbreak resulting from contaminated water was that which occurred not far from Golden Square at the end of August 1854, and which caused more than 500 deaths within a very limited district in the course of about ten days. There is abundant evidence to connect this terrible outbreak with the escape of sewage into the Broad Street well.[2] While it was proved in

[1] *British Medical Journal*, Nov. 1866, p. 505.

[2] Dr. Snow *On the Mode of Communication of Cholera*, 1865.

the case of nearly every sufferer from the disease in that district and in two cases occurring at a distance, one at Hampstead and the other at Islington, that the patients had drunk the water of the contaminated well, it was also found that many persons drank the water about the time of the outbreak without being attacked by cholera; but this does not lessen the force of the positive evidence derived from the other facts. It rarely if ever happens that every individual of a number exposed at the same time to any source of infection succumbs to the infecting agency.

There are facts which tend to show that the choleraic discharges in a certain intermediate stage of decomposition have more infective power than either the fresh dejecta or those which have reached a more advanced stage of decomposition.

We are indebted to Mr. Charles Macnamara for some important information as to the influence of water contaminated with choleraic discharges in various stages of decomposition. He says :[1] 'I may mention the circumstances of a case in which the most positive evidence exists as to the fact of fresh cholera dejecta having found their way into a vessel of drinking water, the mixture being exposed to the heat of the sun during the day. Early the following morning a small quantity of this water was swallowed by nineteen persons. (When partaken of, the liquid attracted no attention, either by its appearance, taste, or smell.) They all remained perfectly well during the day, ate, drank, went to bed, and slept as usual. One of them, on waking next morning, was seized with cholera; the remainder of the party passed through the second day perfectly well, but two more of them were attacked with cholera the next morning. All the others continued in good health till sunrise of the third day, when two more cases of cholera occurred. This was the last of the disease; the other fourteen men escaped absolutely free from diarrhœa, cholera, or the slightest malaise.'

The author goes on to state that at the time of this remarkable occurrence there was no cholera in the place; there had been none there for years, nor has there been any since.

[1] *A Treatise on Asiatic Cholera*, p. 196.

'These details,' he says, 'leave us no room to doubt that water contaminated by the dejecta of a patient suffering from cholera produced the disease in five out of nineteen people who swallowed it, and that independently of either the season, nature of the soil, or any other appreciable circumstance, all of which were remarkably in favour of the persons attacked by the disease.' [1]

Mr. Macnamara has evidently had unusual opportunities for ascertaining the effect of cholera dejecta in various stages of decomposition. He believes that the fresh dejecta are harmless, but that water contaminated with choleraic secretions in the vibrio stage of decomposition, which under an Indian sun occurs in twenty-four hours, will communicate the disease to a certain proportion of those who drink it, while the same organic matter, in a yet further stage of decomposition, when the vibriones had disappeared and ciliated infusoria had taken their place, again becomes innocuous. 'This opinion of mine,' he says,[2] 'is based upon tolerably certain grounds; but I can assert it as a positive fact that the contaminated water in the third stage of decomposition is harmless.'

In the mode of communication from the sick to the healthy there is a close resemblance between cholera and enteric fever. Dr. Murchison [3] says of the latter disease: 'All evidence is in favour of the view that the *fresh* evacuations are harmless, and that the poison is developed during their putrefaction; in other words, that what has been demonstrated in cholera, both clinically and experimentally, holds good in the allied diseases—enteric fever and dysentery.'

The fact that the gastro-intestinal discharges contain the infecting material of cholera has been emphatically insisted upon in the following statement of Mr. John Simon: [4]

'It cannot be too distinctly understood that the person who contracts cholera in this country is *ipso facto* demonstrated with almost absolute certainty to have been exposed

[1] This occurrence is more briefly related by Mr. Macnamara in the article on 'Cholera' in *Quain's Dictionary of Medicine*, p. 240.

[2] P. 397. [3] *Treatise on Continued Fevers*, 1873, p. 466.

[4] *Ninth Report of the Medical Officer of the Privy Council*, p. 33.

to excremental pollution; that what gave him cholera was (mediately or immediately) cholera contagium discharged from another's bowels; that, in short, the diffusion of cholera among us depends entirely upon the numberless filthy facilities which are let exist, and especially in our larger towns, for the fouling of earth, air, and water, and thus, secondarily, for the infection of man with whatever may be contained in the miscellaneous outflowings of the population. Excrement-sodden earth, excrement-reeking air, excrement-tainted water —these are for us the causes of cholera.'

With our present knowledge of the mode of spread of cholera, we can understand that, when proper precautions are taken, whether in public institutions or in private houses, to ensure cleanliness and the effective disinfection and prompt removal of the excreta, the personal attendants upon choleraic patients run no more risk than is incurred by those who are brought into contact with sufferers from enteric fever; while, on the other hand, through the agency of infected drains or excrementally soiled clothes, the contagium of both diseases may be conveyed to a distance, and there exert its infecting influence upon those who are exposed to it.

Some years since I received from the late Dr. Perry, of Marden, in Kent, the following interesting account of a circumscribed visitation of cholera in that village: 'In 1849 a farm labourer left Marden on Monday to go to Sittingbourne for harvest work. Cholera was then prevalent at Sittingbourne. On the following Saturday the man felt unwell, and, getting worse on Sunday, he started for home, a distance of twenty miles. He was purged frequently and vomited on his way home, but stopped several times and took copious draughts of cider. He passed my house at 11 P.M., when I was sent for, and found him in a state of collapse from which he never rallied. He died at 4 A.M. on Monday. The nurse who attended him and laid him out did not know it was a case of cholera. On the Wednesday she was taken ill with vomiting and diarrhœa, from which on Friday she had rallied completely; but on Sunday, having eaten freely of French beans, she was in a few hours prostrated with vomiting and purging. At 10 P.M. she was in collapse, and she died at 5 A.M. on Mon-

day. I was with her all night, and, with her husband, did all the attendance on her, for no one else would go near her for fear.

'On Wednesday evening I was taken ill with diarrhœa and vomiting, and on Thursday morning the nurse's husband was taken ill. He and I were both attended by Dr. Sibbald, of Maidstone. He died on Friday. I was ill for a fortnight, and unable to work for a month, but at length I recovered.

'The man was nursed by his mother-in-law, I by my wife and sister. The complaint did not spread further. There were no more attacks in the parish.'

There can be no question that the occurrence of these three cases subsequent to the imported case was the result of neglect in the disposal of the excreta; and if there had been a public system of drainage in the village the disease would probably have been more widely diffused.

In Dr. Baly's *College of Physicians' Report* will be found recorded numerous instances in which the disease was conveyed from the sick to the healthy by soiled linen. The following is an example:[1]

'The first case that occurred at Sydenham, July 11, was that of a gentleman who came from Bath to attend the funeral of a friend who died of cholera in Bridge Street, Blackfriars, London. The second, third, and fourth cases were sons of the woman who washed the linen and clothes of the first patient. They were washed in a yard to which the second and third patients (children of eight and ten years old) had access. And the fourth patient, a young man of twenty-five years, had carried them, washed and unwashed, from one house to another. These cases, all fatal, occurred a few days after the first case. One other fatal case occurred at Sydenham about the beginning of September.'

Dr. Goodeve[2] very sensibly remarks that for the origin and spread of cholera two factors appear to be requisite—namely, some local condition and some unknown modification of the atmosphere which exists during an epidemic season. 'Contagion from cholera discharges may operate, but there must be something beyond this, since contagious diseases are not

[1] P. 297. [2] Reynolds's *System of Medicine*, vol. i. p. 688.

epidemic at all times even in the same places.' And he compares epidemics of cholera with epidemics of small-pox. The native small-pox inoculators inoculate perhaps the same number of persons year after year, yet for a succession of years there is no great diffusion of this highly infectious disease, but after a time a wide-spreading epidemic sweeps over the land. There is nothing discoverably different in the epidemic and the common years, the same people and the same habits, yet how different are the results!—a difference which can be explained only by some widely diffused atmospheric influence which exists during an epidemic season, and which then concurs with the local conditions in favouring the spread of disease.

A consideration of these facts enables us to understand why cholera does not always prevail in those localities which during an epidemic season are its favourite haunts.

SECTION V.

Statement of Facts in support of the Theory that the Poison—the Infecting Material of Cholera—enters the Circulation before it gives rise to the characteristic Gastro-intestinal Symptoms.

It is a general law of toxicology that the entrance of a poison into the circulation precedes the occurrence of the constitutional symptoms to which it gives rise. The cholera poison, if it were not absorbed, would therefore be a solitary exception to this general law.[1] But there are positive facts which support the theory of blood-poisoning. We have seen that the infecting material of cholera, although often swallowed, sometimes gains admission through the lungs; and in such cases it is obvious that the blood is the only channel by which it can reach its ultimate destination—namely, the alimentary

[1] It is notorious that even such insoluble substances as arsenious acid and calomel enter the circulation, and may be found in various tissues and organs and secretions. 'It is only when it enters the circulation that the poison can be truly said to be introduced into the body. A poisonous substance introduced into the stomach is as much outside the system as if it were applied to the skin.'—Guy and Ferrier's *Forensic Medicine*, p. 357.

canal. Vomiting and diarrhœa have not unfrequently been excited by breathing air which is contaminated by exhalations from decomposing animal matter.

Thus Dr. Christison[1] states that 'M. Ollivier, while visiting a cellar where old bones were stored, was seized with giddiness, nausea, tendency to vomit, and general uneasiness; and subsequently he suffered from violent colic, with profuse diarrhœa, which put on the dysenteric character and lasted for three days.'

Breathing the air of the dissecting-room and the dead-house is a common cause of diarrhœa; and Mr. Simon[2] remarks upon the instructive fact that when a diarrhœa has been excited by dissecting animals whose flesh has a peculiar odour, this odour may sometimes be detected in the evacuations of the patient. Some years since, I was present while a medical friend examined the dead body of a patient who had died of cancer of the bladder and bowel. The stench was singularly strong. Within a few hours my friend was seized with diarrhœa; and he noticed that his stools had the strong and peculiar odour which had pervaded the room when the body was being examined. In such cases as this, when the poison is known to have entered the system through the lungs, Mr. Simon remarks: 'The increased secretion from the mucous membrane of the alimentary canal gives undoubted evidence of a humoral origin and a curative tendency.'

It has been proved by experiments on animals that many of the symptoms of cholera may result from the injection of putrid matters into the blood. Mr. Henry Lee, in his *Pathological and Surgical Observations*, describes several experiments of the kind, which were followed by vomiting, diarrhœa, difficult breathing, great prostration, and death.

In cases of cholera there is usually an interval varying from one to three or four days between exposure to infection and the onset of the intestinal symptoms; during this period of incubation we may conclude from analogy that the poison is increasing at the expense of some of the blood-constituents; and at the same time there have often been observed symptoms

[1] *Treatise on Poisons*, 4th ed., p. 636.

[2] *Lectures on General Pathology*, p. 231.

which are probably due to the influence of the poisoned blood upon the nervous system. It would be of interest to ascertain whether during this incubation period there is a febrile elevation of temperature. The pathological theories which have unhappily prevailed in this country have led to an undue regard being paid to diarrhœa as a premonitory symptom of cholera, and to an almost entire disregard of other symptoms, to which the earlier Indian practitioners attached great importance. The *symptoms of invasion*, as they have been called, are those indicative of general discomfort and derangement of function, particularly affecting the nervous system. Twining says:[1] 'Prior to the more distinct and alarming attack, there are sometimes for a few hours, and in some cases for two or three days, symptoms of indisposition, evident not only to the patient himself, but to his friends. When cholera is raging severely, the disease is often ushered in by diarrhœa; at other times it begins with catarrh, nausea, and oppression at the scrobiculus cordis, which are not in an early stage to be distinguished from the slight indisposition which often precedes fever. The approach of cholera in this manner makes the patient suppose he is feverish or bilious; and *if recourse be had to some of the medicines commonly used in slight ailments of that sort, the disease is said to be caused by the dose of medicine taken, when, in fact, it had been insidiously making progress for some hours.*'

In this country it has frequently been asserted that an attack of cholera has been caused by the operation of a rhubarb pill or a dose of castor-oil. I can give a parallel to this *post hoc ergo propter hoc* argument. Some years since, when I was seeing out-patients at the hospital, a woman brought a child who was suffering from febrile symptoms, and for whom I prescribed a mixture containing nitrate of potash. Two days afterwards the mother returned, and said, in a complaining tone, 'Your medicine has brought out the small-pox.' Truly the small-pox had come out; but certainly it was in the blood before it came into the skin. So when symptoms of cholera follow speedily on the action of an aperient, the morbid poison was before in the blood, and probably caused

[1] *Op. cit.* p. 9.

the feeling of derangement for which the dose was taken. It is as probable that small-pox might result from rubbing croton-oil on the skin, as that a specific disease like cholera would be caused by a purgative.

In a military report on cholera in Malta in 1865,[1] it is stated that out of forty-two cases which were carefully noted, symptoms of invasion existed clearly and unmistakably in twenty-seven. The symptoms were a dull and listless appearance of the countenance and eyes; a dark rim round the orbits; a loss of all activity; want of appetite; prostration; vertigo; cramps; increased perspiration and coldness of the surface on the least exertion; thirst, and general malaise. After these symptoms had continued for a period varying in different cases from a few hours to two or three days, diarrhœa ensued, and the disease was then said to be present.

Annesley[2] gives an extract from the letter of 'a zealous and intelligent medical officer,' who strongly insists on the great practical importance of studying the initiatory symptoms of cholera. Referring to the purging, vomiting, and spasms, his correspondent says: 'I am so thoroughly convinced that these symptoms are only secondary, that, were the following marks present, I should not hesitate to pronounce the case one of epidemic cholera. As the patient is approached, an appearance of overpowering lassitude is at once perceived, with a pallid, anxious, and sorrowful cast of countenance; and, in more advanced stages, the countenance is dejected and sunk.' He then refers to a case in which he observed this peculiar expression of countenance; and, feeling confident that cholera was impending, he kept a close watch upon the man. After an interval of nine hours, vomiting, purging, and cramps set in.

Bell and Orton[3] describe the initiatory symptoms of cholera in much the same terms as Annesley; and Dr. Paine, whose experience of the disease was obtained in New York, says: 'Diarrhœa and vomiting do not always distinguish the premonitory stage; but it is sometimes denoted only by head-

[1] *Statistical, Sanitary, and Medical Reports*, Army Medical Department, 1866; also *British Medical Journal*, October, 1866, p. 409.

[2] *Op. cit.* p. 24.

[3] *Op. cit.* p. 89.

ache, loss of appetite, oppression at the chest, &c.; and, again, spasms are known to have been the earliest symptom, and at first the only prominent one.' These symptoms of general derangement, which often attend the invasion of cholera, appear to be strictly analogous to those which mark the commencement of other undoubted zymotic blood-diseases.

Again, Orton and others of the Indian authors have remarked on the striking resemblance between the symptoms of cholera and those which result from the bite of a snake or other venomous animal. The venom of these animals unquestionably enters the blood, and thus often destroys life. So, it is probable, does the cholera-poison enter the blood.

Another fact, which is almost certainly indicative of a morbid condition of blood in cholera, is the frequent occurrence of *albuminuria* during the progress of the disease. This symptom, in connection with cholera, cannot, I think, be a result of a merely passive congestion of the kidney. Much more probably is it due to an active congestion excited by a morbid quality of blood, and it is therefore analogous to scarlatinal albuminuria.

One of the most conclusive arguments in favour of the doctrine that a morbid poison in the blood is the essential cause of cholera is based upon the fact that the worst symptoms of collapse have often been observed to follow immediately upon the arrest of the vomiting and purging by opiates and astringents. The first case which painfully convinced me of the fatal mischief which may result from the treatment of diarrhœa by opium occurred during the cholera epidemic of 1849. A woman, about forty years of age, was seized with the usual symptoms of choleraic diarrhœa—vomiting, purging, and cramps. She had not a symptom of collapse. The countenance was natural, the skin warm, the pulse good. I gave her five grains of Dover's powder every hour until three doses had been taken. When I saw her again, in about three hours from the time of my first visit, the vomiting, purging, and cramps had ceased; and she was in full collapse, from which she never rallied. This case gave a terrible shock to my belief that collapse is a consequence of loss of fluid, and that it is to be prevented by arresting the vomiting and purging which

usually precede and accompany the symptoms of collapse.[1] I saw no way of escape from the conviction that my patient's condition had been made fearfully worse by my well-intended but mischievous interference. Since that time I have had frequent opportunities of ascertaining from the published reports of cases and from what I have seen in the practice of others, that the immediate occurrence of profound collapse is a not uncommon result of the sudden arrest of the vomiting and purging by opium. The salutary and curative efforts of Nature—the vomiting and purging—by which the morbid poison is being ejected are thus arrested; the poison then accumulates in the blood; the flow of blood through the lungs becomes obstructed; and the state of collapse is established.

MM. Briquet and Mignot have published some instructive particulars of the results obtained by them in the treatment of diarrhœa by opium.[2] Their practice was to prescribe rest, rice-diet, and from fifteen to thirty drops of 'laudanum of Sydenham'; the dose to be repeated in an hour, and to be followed by opiate enemata if necessary; their object being, as they say, 'by a stupefying influence upon the nervous system, to change the disposition of the organism towards the choleragenic poison.'[3] In obstinate cases they gave altogether as much as from eighty to a hundred drops of laudanum. The result of this practice was that, out of 200 patients who came under treatment at the commencement of the attack ('dès le début des premiers accidents'), no fewer than twenty-six—that is, 13 per cent.—passed into collapse. This, I have good reason to believe, is a far larger proportion of cases of collapse than would ordinarily occur if choleraic diarrhœa were allowed to take its own course and terminate, as it tends to do, in spontaneous recovery.[4]

[1] It is, of course, possible that collapse might have come on if no opium had been given; but this, at any rate, is certain, that the arrest of the discharges and the collapse were coincident, and that of the numerous cases of choleraic diarrhœa that I have since treated by evacuants not one has passed into collapse.

[2] *Traité du Choléra Morbus*, p. 514. [3] *Ibid.* p. 522.

[4] Contrast with this opiate treatment of diarrhœa the practice of Dr. Stilton, who treated choleraic diarrhœa by gr. xx. doses of calomel. He says: 'This mode of treatment succeeded in overcoming, in 223 individuals, the symptoms which usually precede cholera; though without such a system these persons

Those who advocate the use of opiates and astringents in the treatment of choleraic diarrhœa appear to forget that there is such a result as spontaneous recovery, and they claim the credit of a cure in every case of diarrhœa so treated which does not pass on to collapse. During the epidemic of cholera in 1854, the late Mr. Wakefield stated, in a letter to the *Times*, that in more than 150 cases of choleraic diarrhœa occurring amongst the prisoners at the Middlesex House of Correction, the only medicines which he gave were carbonate of soda and mint-tea; and not a single case passed into collapse. 'The disease was arrested with a rapidity that was quite magical.' It can scarcely be supposed that this plan of treatment had any other effect than to dilute the contents of the bowel, and so to assist their speedy expulsion—a mode of operation entirely different from that of opium and astringents.

Mr. French states, with reference to the treatment of diarrhœa:[1] 'I am satisfied from much experience that cases of epidemic diarrhœa generally subside speedily under the use of the simplest possible remedies which are wholly free from astringent properties.' In this statement I entirely concur.

But it may be asked how it could happen, if there be a morbid poison in the blood the escape of which it is dangerous to arrest by opium, that so large a proportion as nearly seven-eighths of MM. Briquet and Mignot's patients escaped without more serious consequences? Ought not the whole number thus treated by opium to have passed into a state of collapse? To these questions it may be replied that, happily for the patients, in a large proportion of cases the outward flux of liquid from the blood prevents the absorption of the drug, and the diarrhœa continues, in spite of repeated doses of opium, for a period varying from a few hours to several days. In such cases it is reasonable to suppose that the curative efforts of Nature succeed in ejecting the morbid poison from

would probably have passed into a state of collapse. In these 223 cases I found it necessary to repeat the calomel, to one individual five times, to some others three times; but in the generality of cases a single dose was sufficient.'—*The Cholera in Malta in* 1837, *from the Italian of Giuseppe Stilton, M.D.*, by Seth B. Watson, M.D., 1848, p. 124.

[1] *The Nature of Cholera investigated*, 2nd ed., 1854, p. 75.

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the blood and bowel, notwithstanding the opposing influence of the drug.

During the last two epidemics of cholera I saw several cases in which a diarrhœa had gone on for some days, in spite of large and repeated doses of opium and astringents. In each of these cases the diarrhœa quickly ceased after the exhibition of one or two doses of castor-oil. And it is evident that the experience of MM. Briquet and Mignot was in perfect agreement with this; for they state that if, in spite of the energetic employment of opium, the diarrhœa continues for a period of two days, opium is then of no avail; and they have found in such cases that an emetic of ipecacuanha has immediately put a stop to the disease. It is probable that a diarrhœa which thus continues for several days while opiates are being given, and which is so speedily arrested by an emetic or a purgative, is due, not to the continued presence of a morbid poison in the blood, but rather to the local irritation of the mucous membrane of the digestive canal by the morbid secretion which has been poured into it, and the complete escape of which has been retarded by the opium.

During the epidemic of 1866 Dr. Ferris, then the Resident Medical Officer of the Bloomsbury Dispensary, successfully treated 201 cases of diarrhœa by evacuants. If the diarrhœa was urgent the patient was directed to go to bed, to take a basin of warm broth, and then a pill with half a grain of calomel and three grains of rhubarb, followed in two hours by half an ounce of castor-oil. If the purging continued the next day a slightly astringent mixture was given, but this was necessary in only a small number of cases. There was no death among the 201 cases.[1]

During the epidemic of 1854 a so-called *Treatment Committee*, appointed by the General Board of Health, endeavoured to ascertain by statistics the most successful treatment of the disease. This was evidently a hopeless task, for in the vast majority of the returns several remedies, having different and even opposite modes of action, were so jumbled together that no clear and definite result as to the effect of any one remedy or plan of treatment could come out of these statis-

[1] *Lancet*, August 28, 1866.

tics. One of their tables is intended to show what treatment was most successful in preventing diarrhœa from passing into cholera. The comparison between calomel alone and calomel in combination with opium is interesting. When calomel alone was given in cases of diarrhœa only 2·4 passed into collapse, but when the calomel was combined with opium no less than 6·9 per cent. passed into the more advanced stage of the disease. It appears, then, that when in the treatment of diarrhœa the purgative action of calomel is lessened by its combination with opium, the risk of the disease passing into the stage of collapse is nearly three times as great as when calomel is given alone. The table containing these figures will be found at p. 26 of the *Report of the Treatment Committee.* We have before seen that of the cases of MM. Briquet and Mignot, which were treated by opium alone, 13 per cent. passed into collapse. These figures all point to the conclusion that to check the diarrhœa by opium is to increase the risk of collapse.

Dr. Lavies, in a very able paper on the 'Pathology and Treatment of Cholera'[1] gave the results of his large experience of the disease in a public institution during the epidemics of 1849 and 1854. And referring to choleraic diarrhœa he said: 'I have no hesitation in asserting that I never treated a case with brandy or opium, or both, that I did not soon regret having done so. The brandy was by far the less harmful of the two, because, as a rule, it acted as an emetic and returned immediately. The opium in some cases remained in the stomach; when it did so the state of the sufferers became worse and worse; they soon sank into a condition of hopeless collapse, expressed themselves "very comfortable,"[1] and died. This occurred over and over again; and it was not until mustard emetics and small doses of aperient medicines were resorted to that any good results seemed to follow.'

The choleraic discharges are often spoken of as if they consisted only or chiefly of the watery part of the blood; but they are something more than and very different from that. We have already seen that in a certain stage of decomposition

[1] *British Medical Journal*, August 11, 1866.

they contain the infecting material by which the disease is conveyed from the sick to the healthy. So that, as Dr. William Budd remarks, 'a single case of cholera may, through the sewers, infect a whole district.'

The very peculiar odour of the rice-water stools—so entirely unlike any other odour that it alone would suffice for the diagnosis of cholera—shows that the stools contain some new material, and that some constituent of the body has undergone a peculiar and specific morbid change.

Now the only probable explanation of these peculiar discharges is that the cholera infection, having entered the circulation, gives rise to a morbid change in some blood-constituents, which are then thrown into the gastro-intestinal canal, and finally expelled by vomiting and purging. The supposition that the entire choleraic process takes place within the alimentary canal, and that there is no blood-infection, is in the highest degree unreasonable and improbable.

The most conclusive evidence of blood-poisoning being an essential feature of cholera is afforded by the fact that the fœtus *in utero* has often been fatally infected by cholera.

M. Briquet, in a report on Cholera,[1] quotes from MM. Bouchut and Millet the statement that 54 out of 120 pregnant females seized with cholera suffered abortion, and in most cases the infant was believed to have perished before its expulsion.

Mr. Simon says: [2] 'From our earliest knowledge of the disease it has been on record that when pregnant women have cholera the intra-uterine offspring almost invariably dies; and more recently, in proportion as the anatomy of the disease has got to be better studied, cases have accumulated, giving detailed evidence in support of an opinion which had from the first been entertained, that the infant in such cases dies of true choleraic infection.' He then quotes the observations of Dr. Goldbaum, who, during three epidemics, had carefully anatomised twenty-two such infants, and never failed to find appearances which, collectively, he deems characteristic of cholera.

Mr. Simon goes on to say: 'It may, I think, be assumed

[1] *Mémoires de l'Académie Impériale de Médecine*, vol. xxviii. p. 238.

[2] *Ninth Report of the Medical Officer of the Privy Council*, p. 433.

for certain that the death of the fœtus is death by cholera, and that the fœtus is infected by its blood. And since its blood is a mere derivative from its mother's blood, the fact seems to be beyond dispute that the mother's blood had cholera contagium in it.'

Mr. Simon, having before expressed his belief[1] that 'cholera begins as a bowel disease, without even a passive intervention of the blood, and that all asphyctic phenomena of the disease are supervenient sympathetic phenomena,' says, with reference to the intra-uterine cases: 'Is there any reason to suppose that the cholera contagium in the mother's blood was not a secondary product of disease—was not let into her circulation from the ferment-seething interior of her bowels?'

In reply to this question I would ask, Is there any reason to suppose that there exists such an essential difference between the primary contagium which conveys the disease to the mother and the secondary product which imparts the same disease to her offspring; that while one is absolutely excluded from the maternal circulation, the other is freely admitted by a process for which the new term of being 'let into' has to be invented? But Mr. Simon's strange theory is obviously irreconcilable with the indisputable facts. For it is manifest that before the poison can act upon the alimentary canal of the fœtus, not only must it have entered the blood of the mother, but it must also have passed through the circulation of her offspring. If it were true, as Mr. Simon believes, or did believe when his report was published twenty years ago, that cholera begins as a bowel disease, producible by direct contagion without even the passive intervention of the blood, the fœtus *in utero* would be absolutely protected from the operation of the poison, which could not, under the assumed conditions, be brought into contact with its alimentary canal.[2] As the intra-uterine offspring may die of cholera, so it may be killed

[1] P. 432.

[2] If the subject were not in its practical bearings of such grave importance, it would be very amusing to see the way in which Mr. Simon's deservedly high authority has imposed upon his unreflecting imitators this strange doctrine of the non-absorption of the cholera poison.

by small-pox, the virus being conveyed to it, like that of cholera, by the maternal blood. What now would be thought of this question if it were seriously put? Is there not reason to believe that small-pox begins as a skin disease, and that the virus in the mother's blood was a secondary product of the cutaneous affection—was 'let into her circulation from the ferment-seething' surface of her skin? Mr. Simon, by his reference to cases of intra-uterine cholera, has afforded valuable confirmatory evidence in favour of the doctrine that the cholera contagium, in whatever way it enters the system, whether with the air through the lungs or with food or water through the alimentary canal, is absorbed into the blood before it gives rise to its characteristic effects. It is obvious that an inhaled poison could reach the alimentary canal only through the circulation.

I scarcely need insist upon the inconsistency of those who, believing that the cholera contagium does not enter the circulation, but that it increases solely within the alimentary canal, by a process which they do not even attempt to explain, are amongst the most strenuous advocates of a repressive treatment by opiates and astringents. Surely, the most rational plan of treatment based upon such a theory would be that which has for its object to expel the pestilent ferment before it has had time to work its destructive influence upon the system. It is, however, inconceivable that any morbific agent not entering the circulation should be able to withdraw from the blood the peculiar and characteristic choleraic secretions.

There are yet other phenomena which receive their explanation by the theory of blood-poisoning. The painful cramps which form so remarkable a feature of the disease are probably due to this cause. This explanation of the cramps receives confirmation from the fact that, however severe they may have been during the initiatory diarrhœa and collapse stages, they are rarely complained of during the stage of reaction, when the morbid poison is assumed to have been eliminated. The poisoned blood is also the probable cause of the peculiar muscular contractions which sometimes occur after death, to such an extent as not rarely to effect movements

in one or more limbs of the corpse. In confirmation of this view I may refer to an observation of Blake's.[1] He found that when a dog has been killed by the injection of a salt of lead, barium, or strontium into the veins, muscular contractions always occur for a considerable period after death. The force of contraction is sometimes so considerable that the posterior part of the trunk has been moved by the contraction of the hinder muscles, and on two occasions the body was pushed away when a *point d'appui* was furnished for the sole of the foot. Blake's experiments were performed without any reference to the *post-mortem* movements of cholera, but they appear to be strictly analogous.

With reference to the question whether these muscular contractions are due to the influence of the poison upon the nervous centres or upon the muscles, Blake mentions one fact which, as he says, is in favour of the latter mode of action. 'When these substances have been injected into the *arteries*, the contractions are generally most marked in the limb through the artery of which the injection had been introduced.'

SECTION VI.

The Pathology of Choleraic Collapse.

Having shown in previous sections that the generally accepted theory of choleraic collapse is inconsistent with the acknowledged facts of the disease, I now proceed to give what I believe to be the true interpretation of the phenomena. The subject is one of extreme interest and of surpassing practical importance.

The symptoms of choleraic collapse are so well known as to need no minute description. The most important and characteristic of them are the following: coldness and blueness of the skin, which is often covered by a cold sweat; great diminution of the volume and force, and often complete extinction of the pulse; shrinking of the features, with a corpse-like sinking of the eyeballs; more or less hurry and difficulty of breathing, with a short, dry cough; a sense of

[1] *Edinburgh Med. and Surgical Journal*, vol. lvi. p. 43.

oppression and often acute pain in the region of the heart; a peculiar feebleness of the voice; coldness of the tongue and breath; a sensation of burning heat in the epigastric region; great thirst; more or less complete suppression of bile and urine; vomiting and purging of a rice-water fluid; torpor and drowsiness in a variable degree, but usually without delirium; and, lastly, cramps in the muscles. Most of these symptoms are present in every case of collapse; some, however, may be absent.

What is the pathological explanation of this remarkable train of symptoms? The one great central fact is this, that, *during the state of collapse, the passage of blood through the lungs from the right to the left side of the heart is, in a greater or less degree, impeded, and in a corresponding degree the supply of blood to the systemic arteries is diminished.* Let us now consider the evidence of there being this impediment to the pulmonary circulation; and let us endeavour to ascertain the probable cause and the consequences of this obstruction to the flow of blood.

Very conclusive evidence as to the existence of impeded pulmonary circulation during life is afforded by the appearances observed in the heart, blood-vessels and lungs after death. We are indebted to the late Dr. Edmund Parkes[1] for the first accurate and entirely trustworthy description of the appearances found in the bodies of those who have died in the collapse stage of cholera, and I will now give a summary of his description of the appearance found in the heart and lungs; premising that in order to ascertain the exact distribution of the blood during life the examination should be made as soon as possible after death.

The average time after death at which Dr. Parkes made the thirty-nine examinations which form the basis of his conclusions was seven and a half hours. The longest period was twenty-two hours, and the shortest under an hour.

In the great majority of cases Dr. Parkes found the right side of the heart and the pulmonary artery filled and, in some instances, distended with blood, while the left cavities of the heart were generally empty or contained only a small quantity

[1] *On Asiatic or Algide Cholera*, p. 7.

of blood, the auricle being partially and the ventricle firmly contracted. 'On cutting through the roots of the lungs a quantity of blood usually escaped from the divided vessels, and particularly from the pulmonary artery. In one case the quantity of this was two pints, in one case one pint, in one twenty-four ounces, in one eight ounces, in one six ounces, in nine between two and five ounces.'[1]

The tissue of the lungs was in most cases of pale colour, dense in texture, non-crepitant, and contained less than the usual amount of blood and air. 'In fourteen cases the lungs were completely collapsed, appearing in some cases almost like the lungs of a fœtus.[2] In three cases they were considerably, and in eight cases they were slightly, collapsed; and in the remaining fourteen cases the collapse was, in some altogether, and in others partially, prevented by old adhesions.'

The deficiency of blood in the minute texture of the lungs was shown by their light weight as compared with that of the lungs in a normal condition; and also by the very slight loss of weight consequent on incising, washing, and draining the lung.

'The quantity of blood in the substance of the lungs was considerable in three cases; in one case there was extravasation of blood in the lower lobe of one lung; in six cases there was a considerable quantity in the vessels of the lower lobes, but none in the upper; in eleven cases there was no blood in the minute texture of the lungs.'

'Although there was often little blood in the pulmonary texture, there was sometimes a considerable quantity of frothy and perhaps bloody serum, which I (Dr. Parkes) am inclined to think is not altogether cadaveric, as muco-crepitant and mucous râles are sometimes heard for some hours before

[1] P. 14.

[2] The collapse of the lung, when not prevented by adhesions to the chest-walls, bears a direct relation to the emptiness of the minute blood-vessels; in consequence of which, as soon as the chest is opened, the air is completely expelled by the elastic resiliency of the lung tissue. If a ligature were placed on the trachea before the chest is opened, so as to prevent the escape of air, the lungs would, of course, remain in contact with the walls of the chest. Yet some writers on cholera evidently suppose that the pulmonary collapse occurs during life, and causes the dyspnœa!

death in the posterior part of the lungs.[1] In the most rapidly fatal cases there was the least serum and blood in the lungs; and in inverse ratio to the amount of serum and blood there was a singular deficiency of air in the tubes and cells and a consequent partial or complete want of crepitation. The lung, though apparently denser than usual, floated on water, could readily be inflated, and its condition was very different from the results of pneumonia. The colour of the lung, both on the surface and on section, varied considerably. In many cases it was dark on the surface and pale on section; in some it was dark throughout; in the most collapsed cases it was generally pale, becoming red on exposure to the air, and on inflation becoming vividly red.'

The coronary veins were generally congested, and when the veins were full the cardiac substance generally was dark with blood. In one case there were numerous hæmorrhagic points beneath the serous membrane in the vicinity of the posterior coronary vein, which was much congested. Dr. Parkes thus summarises the condition of the heart's cavities: 'The right side of the heart and the pulmonary arteries were generally filled, and in some cases distended, with blood; the left side and the aorta were generally empty, or contained only a small quantity of dark blood. The inference which was drawn from the state of the cavities in the greater number of cases was, that the right side had continued to receive blood till, in some cases, it became full and even distended, while the left side had received little or no blood, but had continued to contract, in some cases even violently, on the last drop of blood which had entered it.'

Dr. Parkes, in a subsequent chapter of his treatise, attempts a 'hypothetical arrangement of the phenomena,' and, with reference to the *post-mortem* appearances before described, he says:[2] 'The conditions of the heart and lungs seem to point out unequivocally that in cholera the blood does not pass (freely) through the lungs; and this agrees with the independent inference drawn from the symptoms, that the essential

[1] This condition of the bronchial tubes, and the occasional engorgement of the lungs, we shall see hereafter, admit of a simple explanation.

[2] P. 104.

signs of loss of animal heat, embarrassment of the respiration, and practical arrest of the circulation are produced by some aberration of, or impediment to, the proper respiratory changes.'

Then, after alluding to the chief symptoms of the disease, he arrives at the following conclusion :[1] 'As, therefore, the mechanical part of the respiration is perfect, and as the heart evidently beats in many cases till stopped by the want of blood on the left side, and by its accumulation on the right side, we are compelled to look for the cause of such arrest of the circulation in the only remaining element of respiration—namely, in the blood itself. So far the induction appears to me authorised and legitimate ; beyond this, it is doubtful and obscure.'

In attempting to solve the problem he again asks,[2] 'Does the heart refuse to propel the blood ? or does the lung shrink in some inexplicable way and refuse to admit it ? '

In another place he says :[3] 'That there is some impediment or arrest of the circulation in the capillary system generally, and in the pulmonary capillaries in particular, appears almost certain ; and it is by no means improbable that this is due to a chemical change in the fibrine,' and he supposes that there may be 'a deposition of fibrine in the capillaries or minute arteries of the lungs.'[4]

Dr. Goodeve, in his article on Epidemic Cholera,[5] quotes Dr. Parkes's account of the distribution of blood in the heart and lungs after death in collapse, and says that 'most persons

[1] P. 107. [2] P. 105. [3] P. 113.

[4] P. 122. These conjectures of Dr. Parkes were published forty years ago, long before Bernard and Brown-Séquard had taught us the function of the muscular arterioles, a knowledge of which could scarcely have failed to suggest to him a much better interpretation of the phenomena. The following is an extract from a letter which I received from Dr. Parkes, dated November 16, 1865. The letter refers to some papers of mine on cholera which were then being published in the *British Medical Journal*: 'I feel pretty sure that you are right in attaching great importance to the difficulty of passage of blood through the lungs, which I always fancied depended on some condition of the blood itself, but I see that you attribute great effect to contraction of the arteries. This is certainly a very important point to work out, and seems to me to have evidence in its favour—at any rate, that it is a co-operating cause. From what I have seen of your papers it seems to me that they will be a very important addition to our knowledge of cholera.'

[5] Reynolds's *System of Medicine*, vol. i. p. 703.

who have had opportunities of making *post-mortem* examinations of cholera cases will confirm his statements.'

The results of Dr. Sutton's carefully conducted and recorded *post-mortem* examinations in fifty cases of cholera during the epidemic of 1866, are in every respect in accord with the observations of Dr. Parkes. Dr. Sutton says of the state of the heart after death in collapse :[1] 'The right side was seen to be very much distended. This was particularly noticeable in the greatly distended condition of the right auricle and the auricular appendage.[2] The coronary veins were greatly engorged. These were very evident on the posterior surface of the heart. There were numerous spots of ecchymosis scattered over the surface of the heart, and abundant over the posterior surface and at the base of the heart. The eccyhmoses followed the course of the coronary veins, and extended in some cases downwards to the apex of the heart. Occasionally they were noticed in considerable quantity in front of the left apex. The left ventricle was usually contracted, often firmly so ; and, on cutting off the apex of the ventricle and squeezing it, the cavity was seen to be empty, or contained very little blood indeed.'

'The lungs weighed very much less than normal. In some cases they were very pale in the anterior two thirds, and of a darker colour in their posterior third. The pale portions on exposure to the air rapidly became of a scarlet colour. In other cases the lungs throughout were of a dark red colour, but both

[1] *London Hospital Reports*, vol. iv. p. 489.

[2] When, in exceptional cases, the right cavities of the heart are found comparatively empty, this is probably the result of the large veins having been wounded in opening the chest. Dr. Sutton remarks as to this, that 'when the large veins of the neck were accidentally wounded, as in the act of raising the sternum, the blood escaped from the veins, and the right ventricle was emptied in two or three minutes' (*loc. cit.* p. 493) ; and he refers to one case in which this occurred from the wounded jugular veins (p. 448). In the *British Medical Journal* (May 7, 1870), Mr. Worley has recorded an interesting case of clot in the pulmonary artery which had caused death after ten minutes of dyspnœa, screaming, and struggling. In that case, although a clot was found 'completely filling' the pulmonary artery, *all* the cavities of the heart were quite empty. As, from the mode of death, it is certain that the right cavities must have been greatly distended during life, the only probable explanation of their being found empty is that the large veins had been wounded in opening the chest: and it is obvious that the disgorgement of the cavities would be rapid in proportion to their previous fulness and the consequent tension of their walls.

the pale and the dark-looking lungs were dry, and on pressure gave but very little blood. Dark thick blood was seen flowing out of the branches of the pulmonary artery. The lungs were often collapsed.' [1]

Again, Griesinger's description of the condition of the lungs and heart after death in collapse is in entire agreement with the account given by Drs. Parkes, Goodeve, and Sutton. He says: [2] 'The lungs are much contracted, pale, dry, and bloodless. On section some dark drops of black blood escape; below and behind the tissue contains more blood and moisture. Rarely some hæmorrhagic infarctions are found. Ecchymoses in the pleuræ are frequent. The coronary veins are much congested. The left side of the heart is firmly contracted, almost empty; the right side is distended with dark red, soft or gelatinous colourless coagula. Rarely there is some ecchymosis in the endocardium, often much ecchymosis near the base of the heart. The pulmonary artery is full of blood, the systemic arteries are contracted and almost empty, but, especially at the commencement of the aortic system, a moderate quantity of dark blood is found. The bulk of the blood in the whole body is collected in the larger venous trunks.' [3]

[1] *London Hospital Reports*, vol. iv. p. 504. A case is recorded by Dr. Sutton of so exceptional a character and with so imperfect a history, that it is impossible to decide as to its nature (*Ninth Report of Medical Officer*, p. 392). A man about forty to fifty years of age was seen walking along the pavement near the London Hospital; he suddenly cried out, bent forward, put his hand to his belly and fell. He was picked up at once and brought to the hospital, but he was dead before he got there. There was a large quantity of pale choleraic secretion in the small intestines, while the large intestine contained solid fæces. Dr. Sutton remarks that the morbid appearance of all the organs except the contents of the ileum was strikingly different from that usually found after death from cholera. The lungs were highly œdematous, and a large quantity of fluid exuded from them when squeezed. The most plausible conjecture with regard to the nature of the case is that the man was in bad health with œdematous lungs, and that, being seized with choleraic flux and cramp in the street, he died suddenly of syncope. Clearly the case has no bearing upon any theory of cholera collapse.

[2] Virchow's *Handbuch der Special Path. und Therap.*, Band ii. Abth. 2, p. 312.

[3] I have given these anatomical details at greater length than I should have thought necessary but for the consideration that some controversialists have recklessly denied facts which have been demonstrated and placed beyond all controversy by various thoroughly competent and trustworthy observers.

The above is in substance Griesinger's concise description of the appearances found after death in collapse, appearances which indicate, as Dr. Parkes says, 'that the right side has continued to receive blood till, in some cases, it became full and even distended, while the left side had received little or no blood, but had continued to contract, in some cases even violently, on the last drop of blood that had entered it.'

Yet in a subsequent part of his treatise [1] Griesinger says: 'The distension of the right cavities of the heart appears not to be present during life, as percussion gives (invariably?) a small area of cardiac dulness.' This passage, which was first quoted by Mr. Simon,[2] has since been copied by several authors, but with the omission of the query in the parenthesis which obviously indicates a doubt as to the constancy of the decreased cardiac area. Even if it were constant, and not the result of the occasional overlapping of the heart by an emphysematous lung, it surely would not suffice to prove that, notwithstanding the conclusive evidence of impeded pulmonary circulation and of resulting systemic venous engorgement during life, the great distension of the right side of the heart is the result of a *post-mortem* movement of the blood.[3]

Inasmuch as the distension of the right side is associated with a corresponding emptiness and collapse of the left, the actual area of the heart remains much the same as in health; but it may be that the shrinking of the left ventricle, which normally comes in contact with the chest wall, and thus gives the dull note on percussion, accounts for the lessened area of cardiac dulness during collapse; [4] and so this physical sign of a shrunken left ventricle may be held to afford indirect evidence of the correlated distension of the right cavities, which are more overlapped by lung than the left ventricle.[5] It is probable, too, that the extreme anæmia of the lung during collapse may materially modify the percussion note in the cardiac region.

[1] P. 326. [2] *Ninth Report*, p. 429, *note*.

[3] If anyone can believe that the engorgement of the right cavities of the heart and the pulmonary artery occurs after death, he might surely say, *Credo quia incredibile est*.

[4] Whether, and to what extent, the area of cardiac dulness is actually lessened during collapse must be determined by future observation.

[5] See fig. 2, p. 27.

The very anæmic condition of the lungs after death in collapse forms a striking contrast with the great engorgement of those organs which is almost invariably found when death occurs during the febrile stage which often follows reaction.

Now, it is evident from the appearances before described that, during the stage of collapse, there is an arrest of blood in the branches of the pulmonary artery *before* it has reached the pulmonary capillaries. The arrest at this point explains the remarkable anæmia of the minute texture of the lungs during collapse, while the hyperæmia of the lungs after reaction is due to engorgement of the pulmonary capillaries. Before I attempt to explain this remarkable arrest of blood, it may be well to allude briefly to certain phenomena in the living but collapsed patient which afford confirmatory evidence that the pulmonary circulation is greatly impeded.

The impeded flow of blood through the lungs resulting, as it must, in a very scanty supply of blood to the arteries, accounts for the failure of the pulse in collapse. It accounts, too, for the fact that the pulse has often been observed to increase in power and volume under the influence of venesection, which, by relieving the over-distension of the right cavities of the heart, increases their contractile power.[1]

Another appearance which receives explanation from the small stream of blood in the arteries, is that of the shrinking of the integuments, and especially the collapse of the features and the sinking of the eyeballs. The eyes of a patient in deep collapse are often as much sunk as those of a corpse; and the chief cause of this, in the case of both the cholera-patient and the corpse, is the more or less complete emptiness of the branches of the ophthalmic artery.

That the arterial stream during collapse is reduced to a minimum is proved by the fact that arteries of considerable size have been opened during life without the escape of blood. Magendie states[2] that on one occasion he cut across the temporal artery of a patient in collapse and no blood escaped.

[1] See Dr. Reid's essay, *On the Effects of Venesection in Renewing and Increasing the Heart's Action under Certain Circumstances*, *op. cit.* p. 51, and the chapter on *The Action of Blood-letting, &c.*, p. 56; also an experiment by Professor Gerald Yeo, related at the end of this section, p. 137.

[2] *Leçons sur le Choléra Morbus*, p. 21.

('Il ne s'écoula pas une goutte de liquide.') Scot says [1] that, the temporal artery having been frequently opened, 'little or no blood could be obtained, the artery merely emptying itself in a languid stream, not in a jet, and then collapsing.' He also states that a surgeon, despairing of other means, cut down upon the *brachial* artery; but so completely had the circulation failed that no blood flowed.

While, therefore, the small and feeble pulse, the collapse of the features, and the occasional absence of hæmorrhage from a wounded artery are explicable on the supposition that the arteries receive a very scanty supply of blood, these phenomena afford evidence confirmatory of that derived from *post-mortem* appearances, that during the stage of collapse the passage of blood through the lungs is much impeded, with resulting distension of the pulmonary artery, of the right cavities of the heart, and of the whole systemic venous system. This systemic venous distension, involving the coronary and bronchial veins, explains the patches of ecchymosis which are often seen beneath the pericardium and the pleura. It explains the dark colour of the lungs, by bronchial capillary engorgement, while the pulmonary capillaries are bloodless; it also explains the mucous and sometimes blood-tinged exudation into the bronchi during collapse.[2]

It appears, then, that, during the stage of collapse, the blood which is sent into the pulmonary artery is, in great part, arrested in the minute branches of the artery *before* it reaches the capillaries of the lungs. What is the cause of this arrest of blood? Some writers have suggested that the blood has been rendered so thick by the loss of serum that it cannot pass through the minute vessels. This theory is inconsistent with the acknowledged facts of the disease. It is entirely at variance with the fact before referred to (Sec. I. p. 73), that in the worst class of cases there is no direct relation between collapse and loss of fluid by the bowels; and again, with the fact that the state of collapse passes off, while the loss of fluid by purging continues, and while, therefore, the thickening of the blood which, according to this theory, has stopped its passage through the lungs, should be continually increasing (Sec. III.

[1] *Report on Epidemic Cholera*, p. xxx.

[2] See chapter iv. on *The Results of Retrograde Engorgement*, p. 46.

p. 86). The suddenness with which collapse often occurs is quite inexplicable by the theory that thickening of the blood through loss of its water is the cause of that condition. In illustration of the sudden onset of the collapse I may refer to the following statement by Sir William Burnet.[1]

'The first to be attacked were men already on the list for diarrhœa, several of whom fell into a state of collapse one after the other; but about the same time, robust, healthy men, who had fallen suddenly down in a state of collapse, began to be brought in from various parts of the ship (the *Britannia*), even from the yards, where they were seized while reefing sails.' And the surgeon on board the *Albion* reported that 'the attacks in many instances were so sudden that many men fell as if they had drunk the concentrated poison of the upas tree.'

The supporters of this theory of blood-thickening as the cause of collapse would have us believe that, in the course of a few minutes, the blood of these robust men had become so thickened by the loss of water as to be incapable of transmission through the minute vessels of the lungs. It is noteworthy that during collapse the *bronchial* capillaries are deeply injected with the same blood as is supposed to be too thick to enter the pulmonary capillaries. If blood-thickening were the cause of the impeded flow of blood through the lungs the arrest would occur in the smallest vessels, the capillaries, and instead of anæmia of the pulmonary capillaries engorgement of those vessels would be as common in the stage of collapse as it is during febrile reaction.

I believe the true explanation of the arrest of blood in the lungs to be this. *The blood contains a poison whose irritant action upon the muscular tissue is shown by the painful cramps which it occasions; the blood thus poisoned excites contraction of the muscular walls of the pulmonary arterioles, the effect of which is to diminish, and in fatal cases entirely to arrest, the flow of blood through the lungs.*[2]

[1] *Report on Cholera in the Black Sea Fleet in* 1854.

[2] Some pathologists have suggested that during the collapse stage not only the pulmonary but the systemic arterioles are contracted; but since the effect of contraction of the arterioles is to cause distension of the arterial trunks which lead up to them, as for instance in the first stage of apnœa (pp. 26–7) and in Bright's disease, and since during choleraic collapse the systemic arteries are nearly empty, the evidence of systemic arteriole contraction is wanting.

We have seen that the condition of the lungs after death during collapse affords conclusive evidence that the arrest of the blood occurs, not in the capillaries, but in the minute branches of the pulmonary artery, before the capillaries are reached by the blood. We know that the walls of the arterioles are muscular, and that they have the power of contracting upon their contents under the influence of a stimulus, such as cold, electricity, or mechanical irritation. No physiologist at the present day would deny that spasm of the arteries is as real a fact as spasm of the voluntary muscles.

The distribution of the blood in the heart's cavities and in the pulmonary and systemic vessels after death in choleraic collapse is precisely the same as after death from apnœa, and the mechanism by which the circulation is arrested is identical in the two classes of cases.[1]

Even before the discovery of the muscularity of the arterioles many experiments and observations proved that contraction of the walls of the arteries has great influence on the passage of their contents. For instance, it is a well-known fact that the tissues of an animal immediately after death cannot, without a force which endangers the integrity of the vessels, be injected with any of the coloured fluids which are commonly used for that purpose. The coats of the arteries, so long as their vital tonicity remains, contract upon their strange contents, and impede the passage of the injection into the capillaries.

More than a century ago, Hales[2] performed some ingenious experiments to demonstrate the power which arteries possess to control the flow of various liquids through them. His experiments were performed on animals recently killed; and he found that, while warm water passed very readily through the arteries, cold water, decoction of bark, and brandy, passed much more slowly.

Some experiments performed by Blake[3] on living animals bear upon this question. He found that a concentrated solu-

[1] See the chapter on the Physiology of the Circulation (p. 14), where this is fully explained.

[2] *Statical Essays*, 1769.

[3] *Edinburgh Medical and Surgical Journal*, vols. liii., liv., and lvi.

tion of a salt of soda, when injected into the jugular vein of a dog, killed the animal in less than a minute. On examination after death, the right side of the heart was found greatly distended, while the left contained only a little black blood. A few grains of nitrate of silver in solution destroyed life in precisely the same way. The passage of blood through the lungs is arrested, and the animal dies with the right cavities of the heart distended, while the left cavities are nearly empty. Here again we have precisely the same distribution of blood in the heart and vessels as in cases of choleraic collapse.[1]

The sudden arrest of the flow of blood through the lungs in consequence of the accidental admission of air through a wounded vein in the neck or axilla has some relation to this subject. The entrance of air has usually been indicated by a peculiar gurgling sound in the wound, and the symptoms which rapidly follow are thus described : [2] 'Speedy occurrence of syncope,[3] which is either preceded by a cry, with the expression, "I die," "I am dead," "I suffocate," or by anxiety and tremblings. Or, without any such precursors, the syncope rapidly reaches such a degree that all consciousness is lost, and the patient falls down; cold sweat breaks out on the forehead; and, in a quarter of an hour, sometimes sooner, sometimes later, he is dead.' After death, both in the human subject, and in animals that have been the subjects of experiments, the right side of the heart is found to be much distended with frothy blood; and the same mixture of air and blood is usually found in the pulmonary arteries. The left side of the heart is usually empty; but a small quantity of frothy blood is occasionally found in the left cavities and in the aorta. The cause of death in these cases is the distension of the right side of heart, which results from the impeded

[1] Blake ascertained by careful experiment that while some salts, those of zinc for instance, cause no impediment in passing through either the pulmonary or the systemic vessels, and while the salts of soda and silver are impeded in passing through both systems of vessels, the salts of potash and ammonia pass freely through the pulmonary vessels, but are impeded in their passage through the minute systemic arteries.

[2] Dr. John Reid's *Physiological, Anatomical, and Pathological Researches*, p. 553.

[3] Strictly speaking this is not syncope, but a form of asphyxia (pulselessness) the same as occurs in cholera.

transit of frothy blood through the vessels of the lungs. Mr. Erichsen found, by experiments on a dog recently killed, that beaten bullock's blood, mixed with air, required nearly twice the pressure to drive it through the pulmonary vessels that would suffice to drive unmixed blood through the lungs.[1]

I have referred to these experiments in illustration of the general principle: 1, that the movement of blood through the lungs may be quickly arrested by the addition of some foreign ingredient to the blood; and 2, that this arrest is probably, nay certainly, due to the power which the smallest arteries possess to contract upon their contents. No other explanation of the sudden and complete arrest of the circulation in the various circumstances to which I have referred appears probable or even possible.

We can now understand the sudden coming on of collapse, and its sudden passing off. Robust men falling down 'as if they had drunk the concentrated poison of the upas tree,'[2] and others recovering almost as rapidly as 'patients who are resuscitated after suspension of animation from submersion in water.'[3]

We can also understand that there will be, in cases of sudden collapse, an inverse ratio between the amount of the discharges and the degree of collapse. So long as the circulation is unimpeded the amount of the dejecta is a measure of the severity of the attack, but from the time that the impeded circulation through the lungs lessens the blood-supply to the systemic arteries, and so checks secretion from the alimentary canal, there ceases to be a direct relation between the discharges and the degree of collapse.[4]

[1] *Edinburgh Medical and Surgical Journal*, vol. lxi.

[2] See p. 117. [3] See pp. 80, 81.

[4] Mr. John Simon's contention (*Ninth Report, &c.*, p. 429), that the state of the circulation in collapse may be explained by 'feebleness of heart contraction so far as this affects (or at least predominantly affects) the right side of the heart,' may, I think, be easily disposed of. I am not aware of any form of disease in which the heart, *when free from structural change*, is found to be specially weakened on one side. That the heart is enfeebled during collapse is a necessary consequence of the defective blood-supply to the coronary arteries; but this is a consequence, and not the cause, of the arrest of blood in the lungs, and it affects equally both sides of the heart. The facts which are not explained by Mr. Simon's hypothesis are the following. The right cavities of the heart are not only *filled* but *distended* with blood, which spurts out when a

The proof that the blood is arrested during the stage of collapse is, as before stated, partly the anatomical condition of the heart and lungs after death, partly the complete agreement of this condition with the symptoms observed during life.

Many cases have been recorded in which the *obvious mechanical* arrest or impediment of the pulmonary circulation by a fibrinous coagulum has suddenly developed a train of symptoms having a striking resemblance to choleraic collapse.

Some years since, I had under my care in the hospital a woman (S. B.) who was suffering from dropsy, the result of heart disease of long standing. On going through the ward one afternoon, I saw her sitting up in bed, no worse than usual. In less than five minutes after I left her to go into another ward, I was sent for in consequence of her having become suddenly worse. I found her gasping for breath, cold, and pulseless, with a blue and shrunken appearance of the features, exactly resembling the collapse of cholera. I at first thought that she would die immediately; but she rallied in some degree, and lived forty-eight hours, during which time her dropsical legs became rapidly gangrenous. We found, as I had predicted, that, besides old-standing disease of the mitral valve, there were firm fibrinous coagula in the branches of the pulmonary artery. The sudden obstruction of the pulmonary arteries by fibrine caused a state of collapse exactly resembling that of cholera.[1]

One of the most remarkable cases in which a clot in the pulmonary artery gave rise to symptoms bearing a remarkable likeness to the collapse of cholera has been recorded by Dr. Alfred Carpenter.[2]

puncture is made soon after death; yet, in spite of this evidence that the heart has forcibly contracted up to the last moment of life, the anæmia of the pulmonary capillaries shows that some force more powerful than the heart has arrested the blood before it reached those vessels. The ecchymoses beneath the pericardium and pleura—results of a forcible retrograde engorgement in the systemic veins; the frequency of rapid recovery from extreme collapse, the impotence of brandy to assist the circulation, and, on the other hand, the many instances in which free venesection has been followed by an improvement of the pulse and relief from the pain and feeling of oppression which result from an over-distended and labouring right heart, are all inconsistent with Mr. Simon's hypothesis.

[1] *Hospital Case-Book*, vol. xix. p. 59. [2] *Lancet*, September 23, 1871.

In that case Dr. Carpenter remarks: 'The only symptoms wanting to make it apparently a case of cholera were alvine discharges and cramps in the limbs.' The symptoms actually present were blueness of the surface, icy coldness of the uncovered parts of the body, cold clammy perspiration, coldness of the breath, sinking of the eyes, feebleness of the voice, a feeble thready pulse, with quick breathing, constant pain in the chest and epigastrium, intense thirst,[1] and almost complete suppression of urine, two ounces of urine only having been passed one day and on another day less than two ounces. On examination after death the right side of the heart was found fully distended with dark-coloured blood; the left side was empty. The pulmonary artery at its origin was partially blocked by a clot which sent branches into the ramifications of the artery for several inches.

The lung structure was slightly œdematous; it was darker in colour than natural, but it was not gorged with blood, though this point was not proved by weighing the whole of the lung. Here, at any rate, we have the history of a case of collapse from arrested pulmonary circulation without loss of fluid. It was a typical case of dry collapse.

Dr. William Martyn has given some particulars of a similar case,[2] in which the pulmonary artery became plugged during the course of rheumatic fever. The nose, cheeks, and ears were cold and pinched; her hands and feet were of a dirty white colour, cold and shrunken; her forehead was hot; her breath markedly cold; she had occasional cramps in her legs; her pulse—at first scarcely a thread—soon ceased to be felt at either wrist, though in her carotids there was a feeble pulsation. The respirations were forty-eight in the minute, while she constantly complained that she could not breathe. The respiratory sounds were everywhere loud. The heart was beating tumultuously and irregularly, but without morbid sounds. She died in little more than an hour, and was conscious to the last.

[1] The dyspnœa and the thirst in cases of pulmonary embolism and in choleraic collapse are results of the small stream of oxygen-bearing blood that reaches the tissues.

[2] *Pathological Transactions*, vol. xxi. p. 71.

Both the right and left branch of the pulmonary artery were nearly filled by a fibrinous plug. Yet with all this evidence of obstructed circulation through the lung, *all* the cavities of the heart were 'pretty nearly empty of blood;' the explanation being, as I have before suggested (p. 112, *note*), that the right cavities had emptied themselves through the veins which had been wounded in opening the chest.

Dr. William Martyn has also published a somewhat similar case[1] occurring after parturition. She complained of distress in her chest and want of air; the pulse was very rapid and feeble, sometimes scarcely perceptible; her extremities became more and more livid and decidedly coldish; 'reminding me very much,' Dr. Martyn says, 'of the cold stage of cholera;' her breath was markedly cold, towards the end her mind wandered, drowsiness passed into stupor, in which condition the body became warmer, the air-passages filling with mucus, and death occurred at six in the evening of the day on which she was seized. There was no *post-mortem* inspection, but it was clearly a case of obstruction at the right side of the heart, and Dr. Martyn remarks: 'The collapsed state connected with obstruction in the pulmonary arteries, it appears to me, closely resembles the collapse of cholera.'

An interesting and instructive illustration of the physiological mechanism by which the pulmonary circulation is arrested when certain poisons are introduced into the circulation is afforded by an experiment performed by Dr. Brunton with *muscaria*, the active principle of a poisonous mushroom (the *Amanita muscaria* or *Agaricus muscarius*).[2] Professor Schmiedeberg, who discovered and isolated the poisonous alkaloid, found that when given to animals it caused great dyspnœa, at the same time that the arteries became so empty that when cut across hardly a drop of blood issued from them. In this respect there is a close resemblance to the collapse of cholera. He also found that the injection of atropine quickly counteracted the effects of the poison. It occurred to Dr. Brunton that the effect of the muscaria poison might be due to its action upon the pulmonary arterioles, and this explanation he was

[1] *Transactions of the Obstetrical Society*, vol. x. p. 263.

[2] *The Practitioner*, December 1884, p. 412.

able to verify by the following experiment:—A rabbit having been narcotised by chloral was kept alive by artificial respiration while its chest was opened so as to expose the lungs and heart. Dr. Brunton goes on to say: 'Our preparations being complete, I injected a little muscaria into the jugular vein. Scarcely was the injection finished when the lungs, which had previously been rosy, became blanched, the right side of the heart swelled up, the veins passing to it became enormously distended, and the left side of the heart almost empty. After allowing this state of things to continue for a short time, I injected a little atropine into the jugular vein—at once the effects of the muscaria disappeared, and everything seemed again to present its normal appearance. The lungs again became rosy, the right side of the heart and the veins contracted, and simultaneously the collapsed and shrunken left side of the heart regained its normal fulness.'

This experiment affords an actual demonstration of the influence of the muscaria poison upon the pulmonary vessels, and, as a secondary result, upon the systemic vessels. The facts thus brought under the eye of the experimenter are in perfect accord with what we believe to occur during the collapse of cholera;[1] but unfortunately it does not follow that because atropine relaxes the arterial spasm induced by muscaria it would have the same relaxing effect upon the arterioles which are contracting under the influence of the cholera poison, or that such arterial relaxation, if it occurred, would be equivalent to a cure of the disease, though it might afford time and opportunity for the employment of other remedies.

Dr. Brunton, however, quotes a paper in the *American Practitioner* (July 1873) by Dr. Saunders, who had treated a number of cases of cholera by the hypodermic injection of from a fiftieth to a thirtieth of a grain of sulphate of atropine 'with the happiest results.' 'When the atropine was used in sufficient quantities to produce the specific scarlatinal rash, dry throat,

[1] The transient contraction of the pulmonary arterioles during the collapse of cholera, or as a result of poisons artificially introduced, leaves no anatomical trace behind; but we shall see hereafter, when we come to the subject of chronic Bright's disease, that the long-continued over-action of the systemic arterioles excited by the abnormal condition of the blood in that disease results in hypertrophy of their muscular walls.

and dilatation of the pupils, in some cases the relief afforded was astonishing; the skin grew warm, the pulse rose, the surface, previously clammy and shrivelled, assumed its natural condition, and in some instances the patient slept soundly for several hours.' Dr. Saunders attaches great importance to the use of calomel in addition to the injections of atropine.

It would appear from these observations that atropine has some power of relaxing the pulmonary arterioles, not only when they have been excited to contract by the muscaria poison, but also when the cholera poison has been the exciting cause of their contraction.

To assert, as some writers have done, that a drug which has the power of relaxing the arterioles in the normal condition—such agents, for instance, as nitrite of amyl or chloral—should relax the spasm of the pulmonary arterioles excited by the cholera poison, is to make a most unwarrantable assumption. It would not be more unreasonable to assume that any agent which causes muscular relaxation in health would relax the spasm of tetanus.

The most interesting and conclusive evidence that the partial arrest of blood in the lungs and the consequent defective systemic circulation is the true key to the pathology of choleraic collapse is to be found in the simple yet complete explanation which it affords of all the most striking chemical phenomena of the disease—the imperfect aëration of the blood, the fall of temperature, the dark and thick appearance of the blood, and the suppression of bile and urine.

Chemical Consequences of the Obstructed Flow of Blood through the Pulmonary Arteries.—It is obvious that the stream of blood from the pulmonary capillaries to the left side of the heart is the channel by which the supply of oxygen is introduced into the system. One necessary consequence, then, of a great diminution in the volume of blood transmitted to the left side of the heart must be that the supply of oxygen is lessened in a corresponding degree. This position probably will not be disputed by any physiologist who will give the subject careful consideration.

The combustion of those constituents of the blood and the tissues which are normally subjected to the action of oxygen

will be diminished in proportion to the deficiency of that gas; and thence follow of necessity a reduction of temperature, a diminished exhalation of carbonic acid, and so scanty a formation of biliary and urinary constituents, which are joint products of oxidation, that while the state of collapse continues the functions of the liver and kidneys are virtually suspended. Twining [1] found that during collapse, while the expired air is not more than one half the normal amount, the proportion of carbonic acid is less than one-half the normal average. More recently, M. L. Doyère [2] has shown, by a series of very exact analyses, that one of the most constant chemical phenomena of cholera is a more or less considerable diminution of the amount of carbonic acid exhaled in proportion to the volume of oxygen absorbed by the blood. In the normal state, the volumes of oxygen absorbed and of carbonic acid exhaled are almost exactly equal. In cholera, with rare exceptions, the amount of carbonic acid given out is much less than that of the oxygen taken in. These exact observations indicate that abnormal chemical changes take place in the blood and tissues of a cholera patient. It is probable that, during the blood-changes which result from the action of the cholera poison, a portion of the oxygen enters into combination with some abnormal products, forming compounds which, as it were, take the place of the carbonic acid. The blood which passes through the pulmonary capillaries to the left side of the heart during collapse appears to be duly oxidised, but the total oxidation of the blood and the tissues is lessened in direct proportion to the diminished current of arterial blood. It is an acknowledged physiological fact that carbonic acid, bile, and urine, are joint products of oxidation. There is thus a constant and most intimate bond between the lung, the liver, and the kidneys. They are physiologically correlated in a very striking manner; and their active work begins simultaneously at the moment of birth. During intra-uterine life the lungs are entirely inactive: no air is admitted into their cells, and the blood from the pulmonary artery passes directly through

[1] *Clinical Illustrations of the More Important Diseases of Bengal*, p. 15.

[2] *Mémoire sur la Respiration et la Chaleur humaine dans le Choléra*, Paris, 1863.

the ductus arteriosus into the aorta. The kidneys and the liver are nearly as inactive as the lungs. The bladder, it is true, usually contains some urine; and the *meconium* which is contained in the intestines is a modified form of bile: but the amount of these secretions formed during intra-uterine life is infinitely small in comparison with the abundant excretion which begins immediately after birth, when, with the establishment of the function of respiration, there is an evolution of carbonic acid, and a continuous formation of the two correlative secretions—the two joint products of oxidation—bile and urine. During the collapse of cholera there is a near approach to that inactive state of the lungs, the liver, and the kidneys, which is the natural condition of these organs in the fœtus; and as the primary cause of their joint activity at the moment of birth is the establishment of the process of respiration, so the essential cause of their conjointly diminished activity during cholera collapse is the partial arrest of blood in the lungs, and the consequent impairment of the function of respiration.

One fact confirmatory of the view that the very scanty formation of bile, urine, and carbonic acid during collapse is a result of the diminished supply of oxygen, is, that the *secretion of milk continues apparently undiminished.* I have myself observed this fact, and it has been mentioned by several authors.[1] Thus Magendie states that, one of his patients having been delivered of a child a few days before she was seized with cholera, the secretion of milk continued so abundant through a first and a second collapse, which ended fatally, that it was necessary to empty the breasts in order to relieve the pain which their distension occasioned. In Mr. M'Carthy's 'Report on Cases of Cholera under the Care of Sir Andrew Clark,'[2] during the epidemic of 1866, it is stated that 'in four cases attacked with cholera while suckling, the secretion of milk continued undiminished, and proved

[1] Amongst others by the following:—Magendie, *Leçons sur le Choléra Morbus*, 1832, p. 27; Dr. Hutchinson, *History and Observations on Asiatic Cholera in Brooklyn, New York, in* 1854, p. 10; Dr. Robertson, *Edinburgh Monthly Journal*, 1848, p. 393; and Dr. Gairdner, 'On the Pathological Anatomy of Cholera,' *Edinburgh Monthly Journal*, July, 1849.

[2] *London Hospital Reports*, vol. iii. p. 449.

very troublesome.' Now, if the other secretions are suppressed on account of the deficiency of water in the blood, why does the secretion of milk continue? Magendie says, because the blood reaches the breasts, and supplies the materials for their secretion, on account of those glands being nearer to the heart than the liver and kidneys, which, being at a greater distance, do not receive the supply of blood necessary for the discharge of their functions! My explanation of this remarkable and instructive fact is simply this. The chief constituents of milk —casein, sugar, oil, and water—may be obtained from the blood without the addition of oxygen. The secretion of milk, therefore, continues during the stage of collapse; while the highly oxygenised secretions are suspended; their essential constituents being formed only in very minute quantities during that stage, on account of the defective supply of oxygen.[1]

Explanation of the great temporary Relief resulting from the Injection of a Hot Saline Solution into the Veins.—No theory of collapse can be considered complete which does not give a satisfactory explanation of the great temporary benefit which, in most cases, immediately follows the injection of a hot saline solution into the veins. I have before referred to this subject (p. 88), and I deferred my explanation until I had given some account of the phenomena of collapse.

I have adduced many facts and arguments in proof of the position that the essential cause of collapse is an arrest of blood in the lungs, occasioned by a spasmodic contraction of

[1] It is probable that the milk secreted during collapse is of poor quality and deficient in its normal solid constituents, but the remarkable point is that an abundant watery secretion should continue during collapse. With reference to this subject Mr. Macnamara makes the following remark (*op. cit.* p. 417): 'With regard to the supposed secretion of milk in cholera I can only say from my own experience that the mother's milk ceases as collapse comes on. If, as some state, it increases I cannot understand where its watery element comes from.' I am not aware that anyone has asserted that the secretion of milk *increases*, but only that it *continues* during collapse; and I would venture to suggest to my friend Mr. Macnamara, that a fact which he cannot reconcile with his peculiar pathology should rather lead him to doubt the truth of his theory than the reality of the fact. It would seem, moreover, that, with respect to the continuance of the milk secretion, his experience differs from that of every other writer who has referred to the subject.

the muscular walls of the pulmonary arteries. If this be the actual cause of collapse, we might, *à priori*, expect that for a time it would be removed by the injection of a hot fluid into the veins. The temperature of the fluid injected by Dr. Mackintosh varied from 106° to 120° F.; but he states that 'the good effects of the injection were rapid in proportion to the heat of the solution.'[1] The fluid, rapidly mixing with the blood in the right side of the heart and in the pulmonary artery, would, as it were, dilute the poisoned blood and render it less irritating, just as diluents render the urine less irritating to an inflamed bladder or urethra. It is probable, however, that the chief action of the injection would be to relax the spasm of the minute arteries by its high temperature.[2] Thus, the impediment to the circulation being overcome, the blood rapidly flows on to the left side of the heart and the arteries, and the phenomena of collapse pass away with marvellous rapidity.[3] The benefit, however, is usually of but short duration; for the primary cause of the impeded circulation—namely, the poisoned condition of the blood—being still in operation, and the originally hot solution being

[1] *Op. cit.*, vol. i. p. 365.

[2] Dr. Parkes, who tried the injection in some cases, appears to have obtained much less striking results from its employment than Dr. Mackintosh and others describe (*op. cit.*, p. 219). The probable reason of this is, that the temperature of his injection was too low. In one case, he says, it did not exceed 98° F.; while in another it was 'tepid.' In the other cases the temperature is not stated. I think, therefore, that Dr. Parkes's failure to do much good by a tepid injection is confirmatory of my view, that the high temperature of the injection employed by Dr. Mackintosh and others had more influence than the mere mixture of water with the blood. It is certain, however, that fluid at 98° F. would be some degrees warmer than the blood in a superficial vein of the arm during extreme collapse. During the epidemic of 1866 Mr. Little injected twenty cases in extreme collapse (*London Hospital Reports*, vols. iii. and iv.), and in five instances the patients were rescued from an apparently hopeless condition. The temperature of the fluid injected was 110° F., and he states (*Medical Times and Gazette*, April, 1867), that 'a temperature below 100° F. is decidedly injurious.' 'The immediate consequences of the operation were to restore the pulse and voice, improve the colour, relieve the epigastric suffering, and lessen the frequency and difficulty of respiration.'

[3] Sir Joseph Lister, in the course of his observations on the frog's web, states that a stream of water at about 120° F. thrown for about a second on the foot caused relaxation of the arterioles, which had before been excited to contract by irritation of the spinal cord, the contraction returning after a few minutes. (*Phil. Trans.* 1858, p. 616.)

cooled down by its diffusion through the entire mass of the circulating blood, the stream of blood through the lungs will soon again be obstructed; and the patient thus passes into a state of collapse as profound as before. It appears, therefore, that the hot saline injection into the veins, and the operation of venesection, when it rapidly relieves, as it often has done, the symptoms of collapse, have this effect in common, that they facilitate the passage of the blood through the lungs, and thus they lessen that embarrassment of the pulmonary circulation which is the essential cause of choleraic collapse. But, whereas the hot injections act by removing the impediment which results from spasmodic contraction of the arterioles, venesection acts by relieving over-distension of the right cavities of the heart, and thus increasing the contractile power of their walls.

I now ask those pathologists who have hitherto thought that the temporary benefit following injections into the veins affords conclusive evidence that collapse results from loss of water to consider whether the explanation here offered is not more probable, complete, and consistent with all the known facts of the problem, than that which is commonly received.

The feeble, whispering voice of cholera I formerly attributed to the small volume of tidal air respired, corresponding with the scantiness of the pulmonary circulation during collapse; so that, to borrow Sir Thomas Watson's expression, 'the vocal pipe feebly blown through refuses to speak.' I still think that this may be a concurring cause, but it is probable that, in consequence of the defective systemic circulation and the scanty supply of arterial blood to the tissues, the tone of the entire muscular system is lessened, and that the impaired vigour of the respiratory muscles in general and of the laryngeal muscles in particular is the main cause of the whispering choleraic voice. It is remarkable how speedily the voice recovers its tone when the circulation is set free by the hot saline injection into the veins.

Cases have been recorded in which symptoms resembling cholera have been attributed to the merely local action of an irritant upon the mucous membrane of the stomach and intestines. Thus in the second volume of the *Transactions of the Clinical Society*, the late Dr. Murchison records 'a case

of gastro-enteritis from local irritants simulating cholera on two occasions in the same individual.' On the first occasion pungent decayed cheese was the exciting cause of the illness. The first symptoms were severe cramps in the abdomen, thighs and calves of the legs, accompanied, *after two hours*, by violent vomiting and purging. On admission, she was in a condition approaching collapse. She had no vomiting or purging after admission, but for thirty-six hours she passed no urine. The urine first passed was albuminous, and for two days the albumen persisted. The second attack, which occurred about three months later, commenced five hours after eating American lobster preserved in tin. On admission, the collapse was greater than on the first occasion. The eyeballs were sunk, and the features pinched. The pulse could not be felt in either the radial or the brachial artery. The whole surface of the body felt cold, and the face and extremities were livid. There was intense thirst, and there were severe cramps in the abdomen and legs. She was treated by ♏iv. doses of liq. opii sed. The purging at once ceased, but for some hours there was urgent vomiting of a colourless liquid. In about seven hours after admission the pulse could be counted in the brachial arteries and, after another seven hours, in the radials. No urine was passed during an entire day. The following day she was decidedly better and passed a pint of urine, which contained a small but decided amount of albumen, and which, with nitric acid, assumed a very dark hue—deep red by transmitted, but almost black by reflected, light. The following day the urine was free from albumen, and had lost the abnormal reaction with nitric acid.

Both the attacks from which this patient suffered, no doubt, had a striking resemblance to cholera, but to attribute the collapse to the merely local irritant action of the decayed cheese and the tinned lobster is surely a pathological error. That some products of decomposing food (poisonous ptomaïnes) entered the circulation, caused the painful cramps which, in the first attack, preceded by two hours the vomiting and purging, excited contraction of the pulmonary arterioles, causing collapse, and irritated the kidneys, so as to excite albuminuria, is a much more probable explanation of the phenomena.

In a foot-note to his paper Dr. Murchison refers to the cases of a mother and three children who were admitted into the London Fever Hospital 'with symptoms of gastro-enteritis,' which commenced three-quarters of an hour after eating decayed American cheese. The youngest boy, aged 6, died with symptoms of collapse on the second day after his admission. The mother, who had not previously been subject to epilepsy, had an epileptiform seizure on the first day after her admission, and three others on the second day, in the last of which she died. The two eldest boys recovered. On *post-mortem* examination the mucous membrane of the stomach in both the fatal cases was intensely red and inflamed, and pieces of the cheese were found in the intestines, which were also at many places very red. In the female, nothing was seen in the brain to explain the convulsive fits, but the kidneys were slightly granular.

Surely the most reasonable explanation of the fatal result in these two cases is that, in addition to the local irritation, there was blood-poisoning, causing interrupted circulation through the lungs and brain, with resulting collapse and convulsions. Dr. Murchison does not tell us whether in these cases, as in the previous one, frequent doses of opium were given; but the fact that, although both patients lived for two days after the seizure, portions of the poisonous cheese were still found in the intestines indicates that evacuants rather than astringents were required.

The fact that portions of the cheese were found in the intestines after death is quite consistent with the doctrine that other portions had entered the circulation and had there caused the gravest constitutional symptoms. It has often happened that, while concrete masses of arsenious acid are found adhering to the inflamed mucous membrane, the poison is found extensively diffused through the fluids and tissues of the body.

Dr. Christison, in his work on Poisons, refers to numerous cases of poisoning by various articles of food in a particular stage of decomposition. In most of these cases there were signs of gastro-intestinal irritation, and in most, too, there were the clearest indications of blood-poisoning. With refer-

ence to some cases of poisoning by pork he says :[1] 'Stimulants, opiates, and blood-letting are of no avail, and the only useful remedies are emetics and cathartics, which speedily put an end to the symptoms by removing their cause.'

In 1826, on the coast of Galloway, four adults and ten children ate a stew made with the flesh of a dead calf which had been found on the sea-shore. 'For three hours no ill-effects followed. But they were then all seized with pain in the stomach, efforts to vomit, purging and lividity of the face, succeeded by a soporose state like the stupor caused by opium, except that when roused the patient had a peculiar wild expression. One person died comatose in the course of six hours. The rest, being freely purged and made to vomit, eventually got well.'[2]

In most cases of poisoning by unwholesome *mushrooms* the symptoms of local irritation of the stomach and bowels are associated with unequivocal signs of blood-poisoning, and in many of the worst cases the latter symptoms predominate. Dr. Taylor quotes the cases of a man æt. 43 and his daughter æt. 5 who suffered severely from having eaten the *Amanita pantherina*. The earliest symptoms appeared in two hours and a half after the meal. They were thirst, faintness, delirium, nausea, paleness of the face, and cold extremities. After eleven hours there was stupor and tenderness of the abdomen. In the child there was cyanosis of the legs, with contracted pupils. It was remarked that even fourteen hours after the fungi had been eaten, portions of them were discharged by vomiting from the action of emetics. They both recovered, which they probably would not have done if, in place of emetics, opiates and astringents had been given. Dr. Taylor prescribes the treatment of this class of cases in a single line, 'The free use of emetics and castor oil.'[3]

Dr. Brunton's experiment with muscaria, before described (p. 123), elucidates the *modus operandi* of the poisonous fungi in causing the state of collapse. A curious fact in the history of the *Amanita muscaria* is that it imparts an intoxicating quality to the urine of those who take it; and this fact is utilised by many Siberian tribes in a very disgusting way. 'A man, for

[1] P. 649. [2] Christison, p. 647. [3] *On Poisons*, p. 689.

instance, may have intoxicated himself to-day by eating some of the fungi; by the next morning he will have slept himself sober; but by drinking a teacupful of his urine he will become as powerfully intoxicated as on the preceding day. Thus, with a very few *Amanitæ* a party of drunkards may keep up their debauch for a week.'[1] It goes without saying that the poison enters the circulation before being excreted by the kidneys.

The following case of poisoning by lobster is an interesting example of unequivocal blood-poisoning, and, as such, may be compared with Dr. Murchison's case before quoted. My friend, Dr. E. L., when he was living as a pupil with the late Dr. Brinton, awoke one morning with urgent dyspnœa. After a time the dyspnœa passed off, but soon again returned, so that Dr. Brinton was much alarmed by his collapsed appearance. After a while flushes of heat were felt in the skin, and soon an eruption of nettle-rash appeared. As soon as the rash came out the dyspnœa finally ceased. He had no vomiting or purging until, remembering that on the previous evening he had eaten curried lobster, he took a dose of opening medicine. He had often before eaten lobster without inconvenience, but Dr. Brinton suggested that the stomach of the lobster might have contained some jelly-fish, and that this had got into the curry. In this case the poisonous food could be traced from the stomach into the venous blood through the lungs, where it caused dyspnœa, to its final exit by the skin.

If the poison, finding an outlet from the circulation by the alimentary canal, had excited vomiting and purging, a pathologist who ignores blood-poisoning, might have attributed the pulmonary distress to the drain of liquid from the blood or the local irritation of the mucous membrane.

It is a notorious fact that such violent local irritants as arsenic and antimony often cause symptoms referable to the nervous system which can be explained in no other way than by the entrance of the poison into the circulation.

Dr. Taylor remarks:[2] 'Some irritants have been observed to affect the brain or spinal cord remotely—i.e. through the circulation and as the result of absorption. This is the case with oxalic acid and arsenic. These common poisons have, in some

[1] Pereira's *Mat. Med.*, 4th ed. vol. i. p. 143. [2] *On Poisons*, p. 63.

instances, from the first given rise to symptoms closely resembling those of narcotic poisoning—namely, coma, paralysis, and tetanic convulsions. In a case of poisoning by arsenic which occurred to Dr. Muirhead, of Bombay, the symptoms of narcotism were so strongly marked that it was believed at first the man had taken a narcotic.' And in illustration of the great extent to which arsenic may be diffused when it has once entered the blood, Dr. Taylor [1] cites the case of a woman poisoned by arsenic in the fourth month of pregnancy, when the poison was detected, not only in the uterus and placenta, but also in the body of the fœtus. This poisoning of the fœtus by arsenic is exactly parallel with the infection of the fœtus by the cholera poison.[2]

Again, tartar emetic affords a good illustration of a poison which, when swallowed, has a local irritant action on the mucous membrane of the alimentary canal, while part of the poison becomes absorbed and exerts a peculiar depressing influence on the nervous system.

When I gave tartar emetic as a medicine more frequently and freely than I now do, I observed that the peculiar nervous depression did not occur to the same extent when the medicine acted quickly as an emetic and purgative; the reason being that the nervous depression results from the absorption of the antimony, which the vomiting and purging tend to prevent. To the same effect, Dr. Taylor remarks [3] that when in cases of antimonial poisoning, vomiting and purging are absent, 'the symptoms affecting the nervous system are generally more prominent.' He also relates two cases of poisoning by a large dose of tartar emetic. In both there was extreme collapse within an hour after the poison had been swallowed, then for several hours profuse watery purging succeeded, and as this continued the state of collapse gradually passed away. In one case there was a copious flow of urine, and pain in passing it, and 'on the third day the whole of the body was covered with genuine tartar emetic pustules.' Here we have conclusive evidence of collapse from blood-poisoning and of conservative elimination by the skin and kidneys as well as by the alimentary canal.

[1] Taylor, p. 29. [2] See *ante*, p. 104. [3] Taylor, p. 458.

The symptoms observed in the notorious case of the late Mr. Charles Bravo, of Balham, afford an instructive illustration of the same principles. Very soon after swallowing a poison which was afterwards found to be antimony (probably in the form of tartar emetic), he vomited the food of which he had recently partaken, and in this vomited matter Professor Redwood found a large quantity of the metal. He then quickly became unconscious, with dilated pupils, pallor and coldness of the surface, and a scarcely perceptible pulse. In this condition he was found by Mr. Harrison and Dr. Moore an hour after the onset of the symptoms. When I arrived four hours later he was still unconscious, but in about a quarter of an hour he was aroused by the voices round his bed, and he recognised me and his cousin, the late Mr. Royes Bell. Up to this time there had been no gastro-intestinal disturbance since the first act of vomiting; but in another quarter of an hour he complained of severe abdominal pain, began to vomit blood-tinged mucus, and had frequent bloody stools. At once the case was seen to be one of irritant poisoning. The gastro-intestinal symptoms, with perfect consciousness, continued until his death, which occurred thirty-two hours after the onset of the symptoms.

At the *post-mortem* examination, which was made by Dr. Payne, the mucous membrane of the stomach and of the entire tract of small intestines was found pale and free from ecchymosis. On the other hand, the mucous membrane of the large intestine, from the cæcum almost to the rectum, was intensely congested and ecchymosed, and the bowel was half full of dark blood.

The most probable interpretation of this remarkable train of symptoms appears to be that the first act of vomiting, excited by the local action of the antimony, expelled, together with the food, a large portion of the poison, but that about the same time another large portion had become absorbed from the stomach and upper part of the small intestine. The entrance of the poison into the circulation caused the state of collapse with narcotism. Subsequently the elimination of the poison through the large intestine excited serious and extensive

structural change in the mucous membrane, and thus destroyed life.

In the hope and expectation of being able to throw some light upon the *modus operandi* of a gastro-intestinal irritant and purgative, my friend Professor Gerald Yeo did me the favour to perform the following experiment in the presence of Dr. Hughlings Jackson and myself.

A cat was rendered insensible by chloroform. The chest and pericardium were opened, so as to expose the lungs and heart, the animal being kept alive by artificial respiration.

Five drops of croton oil were then injected beneath the skin in four places, twenty drops in all. In about five minutes the right cavities of the heart became much distended, the auricle becoming nearly as large as the ventricle, while the left auricle became somewhat smaller than before the injection. The lungs assumed a pale patchy appearance. After about twenty minutes, when the heart had nearly ceased to beat, the two ventricles were punctured. There was very little blood in the left ventricle, but a large quantity of blood spurted from the punctured right ventricle.

The distension of the right cavities indicated an impeded flow of blood through the lungs, and the only probable explanation of this is that the pulmonary arterioles were excited to contract by the stimulus of the croton oil.

On another occasion Professor Yeo repeated the experiment on another cat under exactly the same conditions, Dr. Hughlings Jackson being present with me as on the first occasion. The injection of the oil, five minims in four places, occupied half a minute. Within twelve minutes after the completion of the injection the right side of the heart suddenly became greatly distended, and in a few minutes had ceased to beat in consequence, apparently, of the impossibility of overcoming the impediment in front. The lungs at the same time assumed an exceedingly pallid and anæmic appearance.

After the contraction of the distended right ventricle had ceased for about a minute Professor Yeo opened a superficial jugular vein. The blood flowed freely from *the direction of the heart*, and upwards of an ounce quickly escaped ; the result was that the ventricle became less distended and began to contract

quite visibly though feebly. The contractions ceased in about a minute, the animal having been virtually dead before the vein was opened. The left side of the heart contained very little blood.

These experiments serve to explain the symptoms which have resulted from a poisonous dose of croton oil. Dr. Pereira[1] quotes the case of a man aged 25 who, while suffering from severe typhoid, swallowed in mistake two and a half drachms of croton oil. In three-quarters of an hour the skin was covered with a cold sweat, the pulse and action of the heart were scarcely perceptible, respiration difficult, the points of the toes and fingers, the parts around the eyes and the lips, blue as in malignant cholera—no vomiting. In an hour and a half there were excessive and involuntary alvine evacuations, sensation of burning in the œsophagus, acute sensibility of the abdomen, skin colder, respiration and circulation difficult; the cyanosis extended over the whole body, the skin became insensible, and death occurred, with some of the symptoms of asphyxia, four hours after the poison was swallowed. No lesion was found in the stomach. The intestines presented the characteristic ulcerations of typhoid fever. No mention is made of the state of the lungs and heart.

This case, looked at in the light of Professor Yeo's experiments, is explained by the theory that the collapse, which preceded the intestinal symptoms by three-quarters of an hour, was the result of the poison having entered the circulation and then excited the contraction of the pulmonary arterioles. All the symptoms are consistent with this theory, and with no other.

The mistake which has generally been committed in the interpretation of such cases has been that of attributing the collapse to the local action of the poison upon the gastro-intestinal mucous membrane, or to the copious dejecta; the occurrence of blood-poisoning and the effect thereof upon the circulation being entirely ignored.

The effect of opening the vein in Professor Yeo's second experiment throws light upon the *modus operandi* of venesection in relieving the painful distension of the right cavities of the heart during the collapse stage of cholera. (See p. 83.)

[1] *Materia Medica*, 4th ed., vol. ii. part 1, p. 407.

Pereira [1] gives the history of a case in which a man who had inhaled for eight hours the *dust* of croton seeds, while engaged in unpacking them, became giddy and fell down insensible. When admitted into the London Hospital he appeared in a state of collapse, with short and hurried breathing, dilated pupils, distressed countenance, and cold surface. There was epigastric pain but no action of the bowels, and therefore, on the day following his admission, several doses of castor oil were given. During his several days' stay in the hospital he gradually improved, but still complained of pain in the epigastrium. This case of dry collapse was indisputably the result of the poisonous dust having entered the circulation through the lungs.

The collapse of cholera has often been erroneously compared with a form of collapse which is essentially different in its nature and cause; I refer to the collapse which results from perforation of the stomach and the consequent escape of its contents into the cavity of the peritoneum. In these cases the defective circulation which is the immediate cause of collapse, is due, not to any impediment resulting from contracting arterioles, but to the depressing influence of the pain and nervous shock upon the propulsive force of the heart. The contrasted effects of two modes of treatment in the two classes of cases would alone suffice to prove the essential diversity of the pathological conditions. Opium, which is now, by almost universal consent, admitted to have a fatal influence in choleraic collapse, is the main medicinal resource in cases of perforation. On the other hand, venesection, which has often relieved the choleraic patient of the agony resulting from an over-distended right heart, would never be resorted to by any prudent practitioner in the case of a patient rendered cold and pulseless by the pain and shock caused by perforation of the stomach or intestine.

Here it may not be out of place to remark that the true remedy for cases of collapse resulting from the escape of the contents of one or other of the hollow viscera into the cavity of the peritoneum consists in a prompt resort to evacuant treatment. Surgery can now succeed in doing that which medicine,

[1] *Materia Medica*, 4th ed., vol. ii. part 1, p. 406.

except in very rare cases, fails to accomplish. In illustration of this I may refer to two cases of ruptured urinary bladder successfully treated by Sir William MacCormac.[1] The bladder was stitched and the peritoneum emptied and washed with entire success. In all similar cases in future, whether the stomach or intestine, the urinary or the gall bladder be the seat of rupture or perforation, the same method of treatment should at once be adopted.

SECTION VII.

The Physical and Chemical Characters of the Blood.

The physical and chemical characters of the blood in cholera have been examined by O'Shaughnessy,[2] Parkes,[3] Garrod,[4] Schmidt,[5] Professor Dundas Thomson,[6] Thudichum,[7] and others.

A full abstract of the results obtained by various observers will be found in Sir William Gull's *College of Physicians' Report*. The following is a condensed statement of Dr. Garrod's revision of the results of his own inquiries as compared with those of previous observers.

All observers agree in stating that the blood becomes more tenacious, of a darker colour, with less disposition to coagulate, and that its specific gravity is increased. While the maximum specific gravity of the blood in the healthy male is 1062, and in the female 1060, in cholera cases Dr. Garrod found the specific gravity in adult males to be 1076 and 1081, and in females 1068, 1074 and 1076. There is a diminution of the watery portion and a corresponding increase of the solids. The blood-globules are in excess, being as high as 166 and 171 in 1,000 parts, instead of 140, which is considered a high healthy average. The fibrine is either reduced in quantity or so

[1] *Lancet*, December 11, 1866.
[2] *Report on the Chemical Pathology of Malignant Cholera*, 1832.
[3] *Op. cit.* [4] *London Journal of Medicine*, vol. i. p. 409.
[5] *Characteristik des Epidem. Cholera*, 1850.
[6] *Med.-Chir. Trans.* vol. xxxiii.
[7] *Ninth Report of the Medical Officer of the Privy Council.*

altered in quality that it can no longer be collected by coagulation. The density of the serum is increased. Healthy serum having a density of 1028, it was found in cholera to be 1039 and 1041. The albumen is increased. The salts of the blood are relatively to the other constituents in excess. The blood tends to lose its normal alkaline reaction and to become neutral or even acid. During the stage of collapse, urea in small amount is found; during reaction, more especially when the kidneys fail to act, the amount of urea in the blood is excessive. The obvious explanation is that during collapse, in consequence of defective oxidation, very little urea is formed.

Dr. Thudichum[1] found no urea, or very little, in the blood of patients dead in early collapse, more after prolonged collapse, and much during the state of consecutive fever.

Now, the usual explanation of the blood-thickening of cholera is, that it is due simply to the drain of fluid through the alimentary canal. The fact stated by Dr. Garrod appears to be inconsistent with this theory—namely, that 'the amount of intestinal evacuations in any case is by no means an indication of the extent to which the blood has been altered.'

Then Dr. O'Shaughnessy states that in all the cases of diarrhœa—that is, cases of cholera without collapse—that he had examined, amounting to seven in number, the various constituents of the blood preserved their normal proportions.[2]

The blood-thickening, then, *follows* the onset of collapse; and during reaction, while the intestinal discharges continue to be more or less copious, the blood-constituents rapidly resume their normal proportions. There are other well-established facts which throw doubt upon the commonly accepted explanation of the blood-thickening in choleraic collapse. A precisely similar form of blood-thickening occurs in pathological states unassociated with loss of water. It occurs constantly in cases of extreme and prolonged apnœa.

Dr. Dundas Thomson gives the analysis of a specimen of blood from the nose of a patient 'who laboured under an affection of the mucous membrane of the air-passages,' and in that specimen the proportion of solids to water was higher than in cholera, as shown by the following table:

[1] *Ninth Report, &c.*, p. 471. [2] *Op. cit.*, p. 71.

Health-relation of solids to water . . .	1 to 3·91
Cholera	1 to 2·62
Patient above mentioned	1 to 2·25

The blood during the cold stage of a severe ague fit has the same black and treacly character as during choleraic collapse. Dr. Mackintosh[1] and others who have bled patients during the so-called cold stage of ague describe the blood as flowing from an opened vein in the arm, at first in a slowly trickling stream, and as the system is relieved the stream becomes larger and stronger, till at last it springs from the orifice and the pulse becomes stronger.

The principles which serve to explain the blood-thickening of cholera are set forth at some length in Chapter III. 'On Certain Physical Phenomena connected with the Circulation,' &c. (See p. 41 *et seq.*)

Referring to that chapter for details, I will here briefly restate the main points of the explanation. During the diarrhœa stage the water which is lost by the intestinal discharges is replaced by water absorbed from the soft tissues—the muscles, viscera and connective tissues, which contain four-fifths by weight of easily separable water. The result is that, while there is a partial dehydration of the tissues, as shown by the diminished weight of some of the viscera, the blood maintains its normal proportion of water.

This compensating process has been entirely overlooked by those pathologists who have attempted exact arithmetical calculations as to the amount of water that must escape from the vessels in order to render the blood as viscid as it is in cholera. In these calculations the arithmetic may be right, but the physiology is wrong; for the calculations are based upon the erroneous assumption that the capillaries have impervious walls, and that therefore the blood has no power of borrowing liquid from the neighbouring tissues—an error similar to that of attempting to calculate the time required to empty a closed room of air, by estimating the volume of rarefied air rushing up the chimney from the fire, while taking no account of the influx of air through the crevices of doors and windows.

During the stage of collapse it is evident that some of the

[1] *Practice of Physic*, vol. i. p. 86 *et seq.*

physical forces which are in operation while the circulation in the diarrhœa stage is free, have their direction reversed when the circulation is obstructed.

Water now passes from the over-distended systemic veins through the capillaries into the tissues, which before were partially dehydrated, and this partly explains the increased density of the blood.[1] But this is not the sole cause of the increased density of the blood during collapse. In consequence of the defective supply of oxygen to the system, resulting from the partially arrested pulmonary circulation, those solids of the blood, especially the coloured corpuscles, which normally, under the disintegrating influence of oxygen, are converted into carbonic acid and biliary and urinary constituents, accumulate in the blood and increase its density.[2] On the other hand, during the period of reaction, when the full current of arterial blood brings with it a free supply of oxygen, there is a rapid disintegration of the accumulated blood-corpuscles, with a correspondingly abundant secretion of bile and urine ; as a result of which the normal proportion of solids and water in the blood is speedily restored, although the intestinal discharges continue for a time to be more or less copious.

It appears, then, that during the stage of collapse the blood-thickening is due, in part, to the escape of water through the capillaries which are connected with the distended systemic veins, and in part to the accumulation in the blood of those solid constituents which, when the circulation is free, are continuously being disintegrated under the influence of oxygen.

Further, it is evident that in consequence of the partially arrested pulmonary circulation, and the resulting distension

[1] The rapid absorption of water from the soft tissues is well shown by the speedy removal of a dropsical effusion under the hydragogue action of elaterium; and, on the other hand, the rapid diffusion from the blood into the tissues is demonstrated, when within a few hours after the obstruction of the gall-duct by a calculus, the urine and all the soft tissues become deeply bile-tinged.

[2] 'If the circulation be impeded in any part of the body, and it is prevented from receiving its due supply of oxygen, the metamorphosis of the corpuscles will likewise be impeded and rendered imperfect, the matured blood-corpuscles which are approaching the stage of solution will not be dissolved, and there will consequently be an accumulation of colouring matter, especially of hæmoglobin, which is the most abundant pigment in the natural corpuscles.'—Dr. Franz Simon's *Animal Chemistry*, Sydenham Society's Translation, vol. i. p. 160.

of the systemic veins during collapse, the loss of water through the intestinal discharges can no longer be compensated by the absorption of water from the soft tissues. The aqueous currents are now reversed, and water passes from the gorged systemic veins into the, before, partially dehydrated tissues. So long as the state of collapse continues, therefore, the discharges from the alimentary canal tend to increase the blood-thickening.

There is yet another not unlikely co-operating influence. The extremely contracted pulmonary arterioles probably act as a filter, which allows the more liquid part of the blood to pass on into the systemic arteries while the more solid constituents are retained and accumulate.[1]

An interesting observation of Sir Joseph Lister's[2] on the circulation in the web of the frog lends support to this view. He says: 'When the arteries are considerably constricted the blood moves more slowly though the capillaries than when the tubes of supply are of medium size, and, at the same time, the narrowed arteries appear to filter the blood more or less of corpuscles, which are found in smaller numbers in proportion to the liquor sanguinis in the capillaries; and if the constriction of the arteries is sufficiently great, the web is rendered quite pale, in consequence of the small number of corpuscles in it; which nevertheless continue to move among the tortuous capillaries, producing in the field of the microscope an appearance something like that of a few flies playing about in a room. Finally, if the arteries are completely constricted, all appearance of flow in the capillaries vanishes, and the web has a wholly ex-sanguine aspect.'

These very interesting observations of Sir Joseph Lister should help us to see with the mind's eye the condition of the pulmonary circulation during the collapse stage of cholera. A considerable narrowing of the minutest arteries by contrac-

[1] In so far as this filtering action of the pulmonary arterioles is operative, it will probably be found that the blood which passes on into the systemic arteries is of less specific gravity and contains a less proportion of solids than the blood in the pulmonary artery, in the right side of the heart, and in the large systemic veins.

[2] *On the Early Stages of Inflammation*, Phil. Trans. 1858, p. 657.

tion of their muscular walls lessens the onward current of blood and deprives it of a portion of its more solid constituents, which consequently accumulate behind the obstruction, while a yet further contraction of the arterioles causes a final and fatal arrest of the circulation.

We have seen that blood-thickening, to an equal extent, occurs in cases of extreme and prolonged apnœa and in the stage of choleraic collapse. The physical and chemical influences which are common to these two pathological states are—first, the transudation of water through the systemic capillaries resulting from the venous engorgement; second, the suspended or lessened disintegration and consequent accumulation of the red corpuscles and other solid constituents of the blood, resulting from defective oxidation; third, the filtering action of the contracted pulmonary arterioles upon the blood.

The only probable source of blood-thickening which is peculiar to cholera consists in the continuance of the intestinal discharges during collapse, when the impeded pulmonary circulation, with resulting systemic venous engorgement, not only prevents that passage of water from the soft tissues into the blood which constantly occurs while the circulation is free, but actually determines the escape of fluid in the opposite direction—namely, from the blood of the engorged systemic veins into the tissues. A comparison of the conditions prevailing in choleraic collapse, and in cases of apnœa, warrants the conclusion that the intestinal discharges, even during the stage of choleraic collapse, are the least influential amongst the causes of the increased density of the blood in that disease. One not infrequent result of the blood-thickening, and the stasis which is common to cases of cholera collapse and prolonged apnœa is that coagula form in the pulmonary artery and the right side of the heart to such an extent as to be the immediate cause of death.

The true explanation of the difficulty experienced in obtaining a flow of blood by venesection during collapse is not, as is commonly supposed, that the blood is too thick to flow, but that, in consequence of the impediment in the pulmonary circulation and the resulting scanty stream through the

arteries, the blood in the systemic veins is nearly stagnant. That this is the true explanation is proved by the fact, often observed and recorded, that when, by the application of warmth and friction to the arm, a sufficient outflow of blood is obtained to lessen the distension of the right heart, and thus to increase its contractile power, the blood escapes from the vein in a fuller and more forcible stream and of a lighter colour.

SECTION VIII.

The Physical and Chemical Characters of the Intestinal Discharges.

The characters of the intestinal discharges vary according to the stage of the disease. In the primary diarrhœa stage they are bilious, but, being more or less diluted with water, they have a dirty white or yellowish appearance. In the stage of collapse they are colourless and flocculent; and, having the appearance of boiled rice suspended in water, have been designated 'rice-water' stools.

During the stage of reaction the discharges again become bilious. One of the earliest signs of reaction is the appearance of green vomit, the yellow bile being changed to green by the acid secretions of the stomach. Soon after the vomited matter appears green the stools are found to have a yellow, bilious tinge.

The relative proportion of solids and liquids varies considerably: in some cases the stools appear to consist of nearly pure water, and it is probable that, when the patient drinks much water without vomiting, this liquid dilutes the stools. In other cases the stools passed during collapse, after the acute watery purging has ceased, have the appearance and consistence of jelly or blanc-mange. I have seen several cases in which this gelatinous stuff, having a peculiar fœtid odour, has been brought away by the action of castor oil; and some cases are recorded in which, copious watery purging having occurred, this viscid material has been abundantly present in the intestines after death.

In some instances the earlier stools during collapse have the

colour of port wine. The dark colour is believed to be due to the presence of blood. And the most probable explanation of the escape of this venous blood is that it is due to passive engorgement of the systemic veins and capillaries consequent on the impeded pulmonary circulation; a result of venous engorgement, therefore similar to the ecchymosis beneath the pleura and pericardium. These 'port-wine' stools occurred in four of the cases which came under my care in the Hospital during the epidemic of 1854. Of these cases, two were fatal, the other two recovered. Occasionally during reaction the stools have a *pink* tinge, from the admixture of blood; and such cases are generally fatal by continued hæmorrhage and exhaustion. Two such cases occurred amongst my patients in 1854. In both cases patches of ecchymosis were found beneath the mucous membrane of the small intestines, and in one there were similar patches in the descending colon. In this second case the 'port-wine' stools had been passed during the early period of collapse, and the pink stools during reaction.

The presence of blood, of course, renders the discharges more or less coagulable by albumen-precipitants; but this coagulability is a rare exception to a general rule; for though it is the custom to describe the cholera stools as 'serous,' it is, as Dr. Parkes observes, 'incorrect to speak of the cholera fluid as the serum of the blood. The fluid is derived from the serum, but it is not composed of all its ingredients; it consists of its water, and of its salts, with a very small proportion of its organic elements;[1] and the greatest amount of albumen, except in the rare cases of actual hæmorrhage, is 'very trifling.'

In some 'rice-water' stools traces of bile-constituents may be detected by chemical tests.

The 'rice-water' stools have a peculiar and characteristic odour, which Dr. Mackintosh compared to that produced by macerating fish in water. The odour chiefly resides in the more solid portions; it is greater in proportion to the number of flocculi, and it is especially strong in the flaky gelatinous stools before mentioned. Some flocculi are entirely composed of intestinal epithelium which has been shed by a desquama-

[1] *London Journal of Medicine*, vol. i. p. 134.

tive process during life; but in most stools no entire epithelium can be found.

It has been conclusively proved that the denudation of the villi upon which some pathologists have based their theory of collapse, is a result of maceration in the intestinal fluids after death.[1]

In the process of desquamation which occurs during life the old cells are displaced by new ones formed beneath (as in the cutaneous and renal desquamation of scarlet fever) and the mucous surfaces are not thereby denuded. That the choleraic affection of the bowels is a heat-making or inflammatory process is, as Mr. Simon[2] says, conclusively proved by the fact that while the axillary thermometer shows a temperature perhaps little above 90° F., the instrument in the rectum or vagina indicates a temperature some degrees above the normal.

SECTION IX.

The Stage of Reaction with or without Consecutive Fever.

One of the earliest indications of reaction after collapse is a less sunken appearance of the eyes; and with this is always associated a return of the pulse, when it has before been imperceptible, or an increase in the volume and power of the previously small and feeble pulse. The blood is now beginning to pass more freely through the lungs, bringing with it an increased supply of oxygen; the temperature rises, and the colour of the skin improves; the backward venous engorgement gives place to an increased arterial supply, which, restoring the red colour to the cheeks and lips, forms a pleasant contrast to the cadaverous appearance which it displaces. The expired air now contains its full proportion of carbonic acid. Twining found[3] that during reaction the expired air contained from 3·5 to 4 per cent. of carbonic acid, and sometimes more than that amount. The restored secretion of the liver is indicated by the appearance of green vomit, and, somewhat later, by

[1] Drs. Parkes and Gairdner in *Medical Times and Gazette*, August and September, 1866.

[2] *Ninth Report*, p. 511.

[3] *Op. cit.*, p. 15.

yellow bile-tinged stools. The continuance of the gastro-intestinal discharges, to a greater or less amount, is a constant phenomenon during reaction.

The danger is not passed with the occurrence of reaction, and the risk of serious complications is greater in proportion to the degree and the duration of the previous collapse, during which the normal proportion of the blood-constituents has been seriously deranged. The accumulated unoxidised materials now, with the restored freedom of the circulation, undergo a rapid process of oxidation, and the safety of the patient depends mainly upon the free escape of these oxidised products through their proper excretory channels. The two chief sources of danger now are—the occurrence of pulmonary congestion, and a defective or surpressed secretion of urine.

One of the earliest signs of pulmonary congestion is a slight return of sinking of the eyes, with a diminution of the power and volume of the pulse, and a slight fall of temperature; symptoms indicative that the flow of blood through the lungs is again impeded. There is dulness on percussion over the bases of the lungs, with more or less extensively diffused crepitation. The breathing is either hurried or it is slow and laboured, and there is more or less drowsiness, which may gradually deepen into fatal coma.

The condition of the lungs after death, in striking contrast with their appearance in the collapse stage, is one of extreme capillary engorgement. They contain a large amount of blood and serum, they are of dark colour, their weight is much increased, and they collapse but slightly when the chest is opened. In some cases this condition of capillary engorgement passes into one of pneumonic consolidation.

The urine which is first passed during reaction almost invariably contains albumen, and deposits a sediment containing hyaline and epithelial casts. In most cases the albumen disappears after a few days. The secretion of urine is usually scanty until the previously dehydrated tissues have reclaimed the water of which they had been deprived. A complete suppression of urine at this stage is a very grave condition, and is soon followed by the usual results of uræmia—delirium, drowsiness, convulsions, and coma.

The occurrence of these grave complications during reaction is certainly more frequent when opiates and alcholic stimulants have been given during the earlier stages of the disease. I have before mentioned that the occasional occurrence of pink blood-tinged stools during reaction is usually a sign of fatal import.

It is obvious that the previous existence of organic disease, especially of disease affecting the heart, or lungs, or kidneys, greatly increases the danger during reaction.

SECTION X.

Prognosis.

The prognosis in choleraic attacks depends upon a variety of circumstances, some of the chief of which I will now indicate.

The Stage of the Disease.—In the diarrhœa stage the prognosis is much influenced by the treatment adopted. The unfavourable result of the repressive opiate treatment is conspicuously shown by MM. Briquet and Mignot; 13 per cent. of whose cases passed from the diarrhœa stage into collapse.[1] On the other hand, of the numerous cases of diarrhœa that I have seen treated by evacuants during the last two epidemics, many of them of a most severe type, not one has passed into collapse.

Drs. M'Cloy and Robertson, in their most able and interesting paper,[2] which has received far less attention than it deserves, give the results of their experience in the treatment of diarrhœa during the epidemic of 1866 in the following terms:[3] 'Our experience of diarrhœa was very extensive. Several thousand cases came under our observation in the different dispensaries connected with the West Derby Union and in the Liverpool Parish Infirmary. Among these were doubtless many that would have recovered under any mode of treatment or by the *vis medicatrix naturæ* alone. But there were many, too, of a most severe choleraic type. The treat-

[1] *See antea*, p. 100. [2] *Med.-Chir. Trans.* vol. 50. [3] P. 180.

ment adopted was generally evacuant in its nature, and consisted in the administration of castor oil, calomel, rhubarb, or magnesia. In every case relief was afforded "pleasantly, quickly, and safely." It was seldom that more than two or three doses of oil were required. We found the ordinary fluid magnesia a safe and active aperient in the diarrhœa of infants. We never saw a diarrhœa patient treated with evacuants from the commencement of his attack require subsequent removal to the hospital.'

It must often have happened that when diarrhœa patients have been treated at their own homes, the disease has been perpetuated and aggravated by continued exposure to the exciting cause of the malady—namely, contaminated air or water. In the case of patients removed from their unhealthy surroundings, admitted into a well-regulated hospital, and treated promptly by evacuants, it will rarely, if ever, happen that the disease passes on to the collapse stage.

In the stage of full collapse, the prognosis is incalculably more grave, and the mortality under any system of treatment will be large. With our present knowledge of the pathology of collapse we can fully appreciate the danger which attends that condition. The impeded pulmonary circulation interferes with, and in extreme cases entirely arrests, the elimination of the poison, and, in proportion to its degree and duration, destroys the normal proportion and balance of the blood-constituents; thus increasing the risk of serious and often destructive consecutive fever. When, in consequence of continued exposure to the exciting cause of the disease, diarrhœa has existed for some days before the onset of collapse, the exhausted patient is much less likely to recover than one in whom the collapse has come on earlier.

When the diarrhœa has been thus prolonged, the mucous membrane of the intestine is often structurally changed, and fatal hæmorrhage from the bowels is apt to occur during reaction.

So long as, during collapse, the discharges are not entirely arrested there is ground for hope that reaction will occur; on the other hand, their entire cessation greatly aggravates the danger. The arrest of the alvine discharges may be due either

to the more or less complete cessation of secretion, in consequence of extreme obstruction of the pulmonary circulation, or to a paralysing over-distension of the bowel by the copious effusion of the choleraic secretions. If, in this condition, the attempts to stimulate the bowel to expel its contents are unsuccessful the result is inevitably fatal.

When, during collapse, the obstruction to the pulmonary circulation does not exceed a certain degree, the breathing is usually slow and shallow, but in cases of extreme obstruction with extinction of the radial pulse the respiration becomes rapid and laborious; and unless the circulation can be set free by venesection or by hot injection into a vein it soon comes to a fatal stop.

Consecutive fever is generally severe and prolonged in proportion to the degree and duration of the previous collapse, and it is more frequent and more fatal amongst those who, in the earlier stages of the disease, have been treated by opium and alcoholic stimulants. The two chief sources of danger during this stage are—congestion of the lungs, with resulting dyspnœa and drowsiness, passing into coma; and congestion of the kidneys, with suppression of urine and uræmia.

Age has great influence on the mortality, which is much greater amongst the very young and the very aged than in the intermediate periods of life.

Habitual intemperance greatly increases the danger of a choleraic attack; and it is obvious that those who have been weakened by insufficient food and destitution are more likely to succumb to the disease.

Organic Disease.—The mortality is always great amongst those in whom there exists some organic disease—whether of the heart, or lungs, or liver, or kidneys, or of the alimentary canal.

Period of the Epidemic.—It has often been observed that those cases which occur at the first outbreak of an epidemic are of a more severe and intractable character than those occurring at a later period; but this, like most other rules, is not without exceptions; and the prognosis in each case must be mainly deduced from the antecedents and the present condition of the patient.

Statistics unaccompanied by some details as to the character of the symptoms and the various remedies employed afford but an imperfect index of the mortality under various modes of treatment. Such particulars are given by Drs. M'Cloy and Robertson, and their paper is therefore of extreme value. They had under their care in the Liverpool Parish Infirmary 375 cases of choleraic collapse. Of these, 91 were treated by astringents, hypodermic injections, and alcoholic stimulants, with a mortality of 71·42 per cent.; 87 had castor oil, with liberal use of food and alcohol, and of these 41·37 per cent. died; 197 had castor oil alone, with little or no alcoholic stimulants or food of any kind until reaction was fairly established, and of these 30·45 per cent. only died. The second and third class of cases were treated simultaneously, and the authors express their conviction that the combination of castor oil with large quantities of alcoholic stimulants, beef-tea, arrowroot, &c., 'was almost as faulty a combination as would have been that of castor oil with powerful astringents.'

During the epidemic of 1854, 37 cases of choleraic collapse came under my care in King's College Hospital; they were all treated by castor oil, and there were 23 recoveries to 14 deaths. During the same epidemic 17 cases of choleraic diarrhœa on the verge of collapse were admitted, and rapidly recovered under the same treatment. These favourable results, occurring at a time when I had no definite idea of the nature of choleraic collapse, and under a treatment somewhat roughly carried out, convinced me that recovery from collapse occurs not by the arrest, but by the continuance of the discharges until reaction has set in, when, of course, they gradually cease.[1]

During the last epidemic of 1866 I again had charge of the cholera wards, and I adopted the same plan of treatment, but with much more caution, and guided by the knowledge which, in the meantime, I had acquired of the pathology of the disease. I have related my experience of that epidemic in a clinical lecture which was published in the *British Medical Journal*.[2] Only 21 cases of cholera with collapse were admitted, and 13

[1] Full particulars of these 54 cases are given in my work, *On Epidemic Diarrhœa and Cholera*, 1885.

[2] November 10, 1866.

of these were fatal. This, as a mere statistical statement, appears to be anything but a favourable result; but I am confident that no one who saw the cases thought that a better result, if so good, could have been obtained by any other method of treatment. It so happened that amongst this small number of cases the majority were of so desperate a character when admitted as to be beyond the reach of any known remedies. The following strictly accurate and impartial statement by an anonymous reporter appeared in the *Lancet*:[1]

'Twenty-one cases of cholera have been admitted under Dr. Johnson's care during the present epidemic. There have been 8 recoveries and 13 deaths. Of the 13 deaths, 6 occurred during collapse, 7 after reaction. All the cases were treated by evacuant doses of castor oil. It is remarkable that in four out of the six cases fatal during collapse there was neither vomiting nor purging after the patients came under treatment; in two of these four cases death occurred within an hour after admission. In all the fatal cases the disease was of extreme severity, the patients being for the most part pulseless when admitted; and in a large proportion of the cases there were specially unfavourable circumstances which rendered them nearly hopeless from the first. We give brief particulars of the seven cases which were fatal during reaction.

'An infant 18 months old rallied from extreme collapse, then got congestion of the lungs, with drowsiness and convulsions. A woman aged 47, but looking much older, had been a great invalid (as her husband said) all her life. For seventeen hours before admission she had been profusely purged. When admitted she was pulseless; vomiting and purging had ceased, but the intestines were painfully distended with fluid. With great difficulty the bowels were emptied by castor oil, to which on two occasions croton oil was added, aided by enemata. She made an imperfect rally, the temperature rose from 94·6 to 98·6; then she quickly died with pulmonary engorgement and dyspnœa. Then there occurred three cases in one family—three sisters, aged respectively 5, 6, and 9 years. The children had been ill fed and ill lodged, and were strumous. They all rallied from collapse,

[1] November 17, 1866.

which in two cases had been of extreme severity, and all died comatose after reaction. In the two younger children there was advanced Bright's disease of the kidneys, the organs being in an extreme stage of fatty degeneration, pale and wax-like; in the other child there was mesenteric disease.

'A man aged 23, of weak intellect, had been very poorly fed, and had a chronic ulcer on his leg in a most offensive condition. He was pulseless and in extreme collapse for many hours, and repeated doses of castor oil excited only very moderate purging. An unsuccessful attempt was made to inject hot water into a vein; he made an imperfect rally and died twenty-four hours after his admission.

'The last case was that of a widow who had been over-worked to support her seven children. There had been very copious vomiting and purging for some days before her admission. When admitted at 6 P.M. on October 12 she was pulseless and in extreme collapse. There had been no action of the bowels since 1 P.M. At 8 P.M. Dr. Johnson saw her. Four ounces of oil had been given, but neither vomiting nor purging had occurred. There was urgent dyspnœa with severe pain, apparently over the right cavities of the heart. It appeared that injection into the veins afforded the only hope of relief, and forty-two ounces of water at 118° F. were slowly injected. The pulse returned after the injection, and there was some relief from dyspnœa and the pain over the heart; but the effect of the injection was much less than had been anticipated, and she died at four o'clock the following morning, ten hours after her admission. She vomited occasionally after the injection, but there was no evacuation of the bowels while she was in the hospital. After death a firm decolourised clot of fibrin was found adhering to the inner surface of the right ventricle, near the apex, and extending some inches into the pulmonary artery. This clot had evidently been formed during life, and probably before the injection into the vein. It must have obstructed the circulation, not only by the space which it occupied in the artery, but also by preventing the closure of the semilunar valves; hence probably the painful distension of the right cavities of the heart and the small amount of relief afforded by the hot injection into the vein.'

I may add that there was nothing in my experience of the last epidemic to lessen my faith in the efficacy of the evacuant principle of treatment, if only the condition of the patients be not such as to place them beyond the reach of all known remedial measures.

It is quite certain that not one of these patients died from excessive purging. It will be seen that in five of the cases there were no alvine evacuations after the patients entered the hospital; it will also be seen that in seven cases death occurred not in the stage of collapse, but in the after stage of reaction. To those practitioners who look upon cholera as a flux, the arrest of which is the main object of treatment, it will appear a startling assertion that the immediate cause of death in a large proportion of cases is either a more or less complete arrest of the secretion into the bowel, in consequence of the block in the pulmonary circulation, or a loss of the power to expel the secretions which have accumulated in, and caused a paralysing over-distension of the bowel; a form of bowel-obstruction which, without doubt, is often increased, if not entirely caused, by the benumbing influence of opium in the earlier stages of the disease.

It is obvious that in order to fairly test the efficacy of the evacuant treatment, the patient must be submitted to that treatment from the very commencement of the malady. When patients who have been treated in the earlier stages by opium and other astringents are not subjected to the evacuant treatment until they have passed into a state of all but hopeless collapse, the result is to bring upon this plan of treatment the discredit which really belongs to the earlier and opposite method. That was the disadvantageous position in which I was placed in having to undertake the treatment of the few cases of choleraic collapse that came under my care in 1866.

SECTION XI.

The Treatment of Cholera and Choleraic Diarrhœa.

Exactly a century ago, in 1787, Mr. Duffin, an Indian surgeon, described his treatment of cholera in the following terms:[1] 'I have found no medicine equal to the castor oil, as it carries off the putrid colluvies without irritating the intestinal canal, which even the mildest purgatives in some measure do; whereas the oil sheathes the internal coats, and blunts the irritating matter. I have always observed the spasms to abate when the oil has operated; but frequently it is necessary to repeat the dose two or three times in a day, when the spasms return. Opiates, in most cases where the nervous system is affected, are found useful, but in the cholera they ought to be used with great caution, and never while there is the least apprehension of putridity. As long as the spasms continue, there is a certainty of offending matter in the *primæ viæ*, which, if locked up by the opiates, even for one hour, too often a fatal mortification ensues.'

This common-sense treatment of cholera was based upon the observation of the fact that the intestinal discharges are of a putrid character, with the obvious inference that their retention within the alimentary canal must be attended with danger.

Until epidemic cholera first appeared in Europe in 1831–2, it was the usual practice in India, while giving opium occasionally to relieve the painful spasms, to give purgatives habitually to bring away the offensive morbid secretions. Annesley particularly insists upon the importance of clearing away the peculiar whitish, putrid, viscid secretion which constitutes, in fact, the more solid portion of the rice-water stool, and which is often found in great quantities in the intestines after death; and he expresses his conviction that a relapse has frequently been occasioned by its retention within the bowel.[2]

The fact that in many instances the treatment adopted by those earlier practitioners was a combination of opiates with

[1] Scot's *Report on the Epidemic Cholera*, Madras, p. 239.

[2] *Annesley Sketches*, p. 169.

purgatives, renders their writings the more instructive, since it appears, on a careful study of their cases, that in proportion as the opiates or the purgatives prevailed the mortality was increased or lessened. In not one of the writings of the earlier Indian practitioners is there the slightest indication that they gave opium for the purpose of arresting the discharges. This repressive opiate treatment was suggested by the erroneous theory that the discharges are the essential cause of collapse, instead of being, as we now know them to be, as essential a part of the process of recovery as is the cutaneous eruption in the case of small-pox.

Dr. Parkes, writing sixty years after Mr. Duffin, before quoted, 'cannot account for the astounding success which attended the practice of several gentlemen in the earlier periods in India. One gentleman saved 98 out of 100; another, 88 out of 90; and, although it is obvious that in some of these cases those cured were of simple pseudo-cholera, with a full pulse and a warm skin, yet in other examples, it must be concluded, the cases were really authentic examples of the algide cholera, and yet the mortality was not more than 25 to 35 per cent. I therefore conclude that the epidemics I witnessed were above the ordinary degree of severity, and the mortality proportionably great.' [1]

It does not appear to have occurred to Dr. Parkes that his own comparatively unfavourable results might be due to the different operation of purgatives given to remove offensive secretions, and opiates given with the object of preventing their escape, which latter was the practice of Dr. Parkes.

It cannot but appear surprising to any unprejudiced observer that practitioners should have been so blinded by theory as not to see that the practice of preventing the escape of the offensive morbid secretion by opiates must be dangerous. So far as I know, the history of medicine affords only one similar example of theory leading to the general adoption of a practice which common-sense, apart from theory, would have condemned as unreasonable and injurious. I allude to the theory which assumed that the maturation of the pustules of small-pox required that the patient should be kept in a

[1] P. 195.

heated atmosphere; to ensure which, not only was he covered by heaps of bed-clothes, but the doors and windows were kept carefully closed. The result of this systematic and enforced neglect of ventilation obviously was, that the poison which was thrown off from the patient's skin re-entered his system through the lungs, and the mortality was very great. Sydenham reversed all this; he insisted upon the free ventilation of the small-pox chamber; and the result was a greatly reduced mortality. He did not, however, effect this reform without incurring the displeasure and opposition of his contemporaries. 'I venture to assert,' he said, 'that the physician who has much to do with small-pox runs many risks with his reputation. The vulgar are ever in the habit of ascribing deaths to the officiousness of the attendants; whilst physicians themselves catch greedily at opportunities for slander. They make out their case before incompetent judges and procure most uncharitable verdicts. They act thus in order that they may build up a name for themselves upon the ruined reputation of others—a proceeding disgraceful to even honest artisans, doubly disgraceful to scholars.' [1]

Now surely, the pernicious practice for the abolition of which Sydenham had to fight so hard against the deeply-rooted prejudice of his contemporaries, was in no degree more irrational or mischievous than that of treating cholera by opiates; with the avowed object of preventing the drain of water from the system, but with the inevitable risk of retaining putrid and poisonous secretions; and thus often converting a mild and readily curable form of disease into a severe and intractable one.

In no stage of choleraic disease are the beneficial effects of the evacuant plan and the ill results of the repressive opiate treatment more conspicuously shown than in the diarrhœa stage. The least of the evil consequences of attempting to arrest choleraic diarrhœa by opium is that of prolonging the disease and causing more or less pain and distension of the bowels, with a coated tongue, headache, and fever.

The two following cases, amongst many similar which I

[1] The works of Sydenham, translated by Dr. R. G. Latham for the Sydenham Society, vol. i. p. 137.

saw during the epidemic of 1854 may serve as illustrations of this form of *malapraxis*.

An eminent surgeon, Sir. W. F., was seized with diarrhœa immediately after his return to town at the end of September, and he endeavoured to cut short the attack by opium. He first took ten drops of laudanum; the diarrhœa continued, and he repeated the dose, but without benefit. He then had an opiate injection, but, the purging going on, he next took a compound soap pill. In the meantime he had been so discomposed by the disease and its unsuccessful treatment that he passed two restless nights; his face was pale, his tongue coated, and he had severe griping pains in the bowels, for the relief of which I persuaded him to take a dose of castor oil, which he afterwards repeated with great benefit. The result of this attempt to arrest a diarrhœa by opium while the intestines contained offensive morbid secretions was a serious derangement of health extending over a period of nearly a week.

Another practitioner consulted me for a diarrhœa which had continued for a week in spite of opiates and almost every variety of astringent. Feeling very unwell, he had become extremely nervous about himself, and was afraid to go near a case of cholera lest he should take the disease. I advised him to take a table-spoonful of castor oil, which acted twice, and the diarrhœa then ceased.

In contrast with these two cases of diarrhœa prolonged and aggravated by opiates, and ultimately arrested by an evacuant dose, I give the two following cases, in which the evacuant was given at the commencement of the attack.

In the midst of the epidemic of 1854, when I was in almost constant attendance upon cholera cases, I was seized with diarrhœa at one o'clock in the morning. There was much pain and commotion in the bowels, and two copious liquid stools occurred in quick succession. After the second stool I took a table-spoonful of castor oil; the dose acted twice before breakfast. I went to work as usual during the day, remained quite well, and had no further action of the bowels for two days.

The following more severe attack is related by the patient himself, one of my hospital assistants.

'I was in my usual state of health up to the evening of September 4, and, having taken a particular interest in the cholera patients in our hospital, I had, for three or four days previous to my being taken ill, almost lived in the wards allotted to these patients.

'On Monday night, at about eleven o'clock, I awoke with violent pain and spasm in my stomach, and a most distressing state of oppression, with an inclination to vomit. These symptoms were quickly followed by cramps in my hands and feet. I at once got up and took an emetic, and as soon as the vomiting which it excited had ceased I took an ounce of castor oil. In about half an hour's time I was violently purged, and so much relief did I experience from it, that I repeated the dose of castor oil during the following hour. From this time until eight o'clock in the morning I was purged, and vomited so frequently that I became quite exhausted; yet I suffered little pain, and had lost that dreadful sense of oppression to which I before alluded. I then had two hours' sleep, and awoke much refreshed.

'Throughout the day my bowels were opened several times, and I vomited once or twice after taking food; but in the evening I went down to Blackheath, passed a most comfortable night there, and the next day returned to town ready to resume my duties, although much pulled down by my severe though short illness.'

'C. N. M., M.R.C.S.

'King's College Hospital, Sept. 1854.'

'The dreadful sense of oppression' was probably due to the commencing embarrassment of the pulmonary circulation by the action of the cholera poison; from this he was speedily relieved by his prompt recourse to evacuant treatment. If instead of the emetic and the castor oil, of which, however, he took at least twice as much as was necessary, he had taken a repressive dose of opium, the result might have been very different.

The result of treating choleraic diarrhœa by repeated doses of opium and alcoholic stimulants may be much more serious than in the cases before referred to. In some cases the patient becomes feverish, with a brown dry tongue, heat of

skin, thirst, headache, and drowsiness; and if the treatment be not promptly reversed delirium and fatal coma supervene.

One author, to whom it would be invidious to make a more definite reference, reports several fatal cases of this kind as cases of 'consecutive fever without collapse.' They were, in fact, cases of fever consecutive to the pernicious repressive treatment of diarrhœa by opium.

It is not surprising that under the influence of such treatment as this diarrhœa should cause so many deaths as it does during a cholera epidemic.

During the epidemic of 1853 and 1854, according to the returns of the Registrar-General, while the deaths from cholera in London were 11,495, the deaths from diarrhœa during the same period amounted to 4,267; so that diarrhœa during that epidemic season caused a mortality more than a third as great as cholera.

Those practitioners who treat the diarrhœa stage of cholera by opium and other repressive means are, unconsciously and unintentionally, but not the less really, performing upon the human subject an experiment like that by which Koch finds that he can, with great certainty, induce cholera in guinea-pigs.[1] After introducing the cholera-bacteria into the stomach of the animal, he injects into the peritoneal cavity a narcotic dose of tincture of opium; his object being to render 'it possible for the comma bacillus to remain longer and gain a footing in the intestine.' In this manner he induced fatal cholera in thirty out of thirty-five guinea-pigs experimented upon.[2]

I have before (p. 99) referred to cases in which the abrupt arrest of the discharges by opium has been directly followed by collapse.

The following case is another illustration of the same principle, and of the good result of reversing the repressive treatment.

At the beginning of August, 1866, I attended with Dr. Halse, a youth, aged 15, who had been only a few days in London, and who had drunk water from one of the pumps

[1] *British Medical Journal*, January, 1886.

[2] *Ibid.*, January 9, 1886, pp. 63–4.

in the Temple—water which was believed to have been the source of other cases of cholera. He had diarrhœa, which was stopped by two doses of opium. Then, after a few hours' cessation of the diarrhœa, he was seized with purging and cramps, and he rapidly passed into collapse. When I saw him at 11 A.M. on August 6th, his pulse could scarcely be felt, and was too feeble to be counted; his eyes were sunk, his skin and tongue were icy cold; the cramps were very severe. There had been no purging for several hours, but his intestines on palpation and percussion were found to be full of fluid. There was a state of torpor, and no natural effort to expel the contents of the bowel; and I have no doubt that with a merely expectant treatment he would have died with over-distended and paralysed bowels. He was ordered to have half an ounce of castor oil every two hours, cold water to drink, hot fomentations to the cramped limbs. At 8 P.M. he had vomited once and had passed three fœtid rice-water stools. The intestines were much less distended. He was free from cramp. In other respects the condition was unchanged. To have one or two doses of oil during the night. On the 7th, at 9.30 A.M., he had rallied completely, his countenance being natural, and his skin warm. There had been five or six bile-tinged stools during the night, and the intestines were now everywhere resonant on percussion. After this he was drowsy for some hours, and on the 8th a rash of roseola appeared on the trunk and limbs, but he made steady progress towards complete convalescence. This life was as unquestionably saved by the evacuant method of treatment as if we had pulled him, in a state of unconsciousness, out of deep water.

The brief history of cases like this throws more light upon the influence of opiates on the one hand and evacuants on the other than whole columns of statistics without details of the cases.

We are sometimes told that the best that can be done for a patient in collapse is to let him lie in bed, drink cold water, and recover if he can. I have seen something of the treatment of choleraic collapse by coloured water. I have seen patients thus treated lying in full collapse, having had neither

vomiting nor purging for hours, and suffering agonising pain from over-distension of the bowels by the choleraic secretion. It was evident that all the natural expulsive efforts had ceased, and that, without some artificial aid, death was inevitable. It appears to me that to look on at a patient in such mortal agony and to make no attempt to evacuate the over-distended bowel is as unjustifiable and as cruel as to leave a patient with retention of urine unrelieved. I do not remember to have seen this bowel-distension as a condition requiring active treatment referred to by any writer on cholera. Yet it is a condition of by no means infrequent occurrence, and especially so in cases which, during the diarrhœa stage, have been treated by opium; the effect of which has been to lessen the sensibility and the muscular contractility of the intestines. Several such cases came under my care in 1854, which were effectually treated by castor oil. But in one case all our efforts to excite the expulsive efforts of the bowel were unavailing, and after death the whole length of both the small and large intestine was found distended and dilated by the choleraic secretion.[1]

Another condition which calls for the prompt and judicious use of evacuants has been noticed by several writers on cholera. It has often been observed that, while the more watery parts of the intestinal secretions are discharged, there remains behind a considerable amount of a semi-solid gelatinous material. Dr. Parkes says: 'A proteine constituent is found in large quantities in the intestinal canal, but is not discharged by stool; sometimes, when there are only two or three watery stools, the intestines after death are found to contain a large quantity of this thick whitish or yellow substance, which dissolves in a warm solution of soda and is precipitated by acids.'[2]

When the discharges have been copious and watery, with very few flocculi, it will often be found that the gelatinous stuff is left behind in the bowel; and a case which came under my care in 1854 is an illustration of this and of the beneficial action of castor oil.

[1] See Case 31 in my work *On Epidemic Diarrhœa and Cholera*, 1855, p. 52.

[2] P. 110.

A male æt. 19 was admitted at noon on September 18. He was in good health until 3 A.M. the day of his admission. when he was seized with violent cramp in the legs, occasional vomiting, and incessant purging; he thinks he must have been purged a hundred times (?), the stools being like water.

On admission the eyes were much sunken, the surface of the body and the feet and hands cold; tongue cold and coated; voice very husky; much thirst; drowsy; a noise in his ears like that of a hive of bees; no cramps during the last hour. To have half an ounce of castor oil every half-hour.

10 P.M.—Has vomited twice and had four stools like dirty white jelly and with a very offensive odour; he is warm and free from pain; tongue warm but coated; P. 84; voice still husky; has passed some urine. Continue the oil every hour.

19th, 10 A.M.—Has had eight stools of the same gelatinous character mixed with oil. Feels much better; no pain or cramps; voice less husky; P. 72; good. 1 P.M.: two stools since the morning. Continue the oil every two hours.

20th.—Four stools during the night, less offensive and of a more natural character; slept well; P. 66; still rather hoarse, with a short dry cough. Take the oil every four hours.

21st.—Very much better; has regained his voice and lost his cough. Seven stools since yesterday morning, of much more healthy character. Omit the oil.

22nd.—Is sitting up, and feels quite well.

23rd.—Discharged cured.

It is probable that when this patient was admitted, although the signs of deep collapse were still present, very little of the choleraic virus remained in the blood, and its powerful purgative action had ceased. I have given the particulars of the case to show that the frequent doses of castor oil did no more than expel from the bowel the fœtid gelatinous material, the retention of which would probably have given rise to unpleasant symptoms, by being reabsorbed and again entering the circulation. The castor oil obviously caused no further drain of liquid from the blood, but it was admirably suited by its bulk and its lubricity to loosen and expel the viscid gelatinous stuff from the bowel.

It has often been asserted by writers on cholera that during the stage of collapse absorption from the gastro-intestinal canal is entirely suspended, and that, therefore, medicines administered during that stage can do neither good nor harm. But Magendie has proved by a very simple experiment that absorption, although retarded and lessened, is not entirely suspended during collapse.[1] He injected camphor into the rectum of a patient during the cold stage, and in five minutes he detected the odour of the drug in the breath. In the normal condition he says the odour may be detected in the breath within one minute after the injection. He also detected the smell of ether in the breath within a few minutes after its injection into the bowel of a patient in collapse. There can be no question that the oppressive influence of opiates and alcoholic stimulants, when they have been given in the stage of collapse, is the result of these agents entering the circulation.

In a *Report on the Cholera Epidemic of* 1865 *in the Maltese Islands*, by Surgeon-Major A. Leith Adams and Assistant-Surgeon R. H. Welch, a brief account is given of the results obtained by administering strychnine to six patients during the stage of collapse. In one case three doses of a quarter of a grain each within three-quarters of an hour induced the specific effects of the poison, and 'death quickly followed, leaving no doubt as to the cause.' In two cases one grain and a half and one grain respectively brought the system within the influence of the drug, with no beneficial results; the fatal termination was not retarded. In another case one grain was given during collapse without obvious effect, but reaction ensued, and with it tetanic spasm, which caused death when the indications were of the most favourable kind. In one case only out of the six the patient recovered, but it is not even suggested that the strychnine had any curative influence.

I have cited these cases as affording additional evidence that the process of absorption is not entirely suspended during collapse, and as a warning against the incautious administration of poisonous drugs.

Although it has been conclusively proved that absorption

[1] *Leçons sur le Choléra Morbus*, p. 97.

is not entirely suspended during collapse, it is obvious that for the operation of those remedies which are most efficacious during collapse—namely, stimulating emetics and laxative doses of castor oil—their absorption is not necessary, since they have mainly a local action on the mucous and muscular coats of the stomach and intestines.

In the preceding pages I have endeavoured to explain the principles upon which the treatment of cholera should be conducted, and I have given some illustrations of the practical application of these principles.

I now venture to set forth in detail the following directions for the treatment of the disease in its different stages.

Diarrhœa, during an epidemic season, is in many instances, though not in all, an early stage or a mild form of cholera; and in the great majority of cases of actual cholera, an attack of bilious diarrhœa marks the onset of the disease. A diarrhœa, when it is not the actual beginning of cholera, will weaken the patient, and so may predispose him to suffer from the more serious form of disease. *Diarrhœa, therefore, during an epidemic season, ought not to be neglected even for an hour.* That plan of treatment for diarrhœa is obviously the best which most speedily and completely puts a stop to the disease without subsequent ill-effects.

It may be stated as a general proposition that the immediate cause of diarrhœa or looseness of the bowels is the presence of offending materials in the alimentary canal. These offending materials are of various kinds in different classes of cases. In one case, unwholesome and indigestible food is the exciting cause of the purging; in another case, food of a wholesome character which a dyspeptic patient has not the power to digest; in another case, a large and unnatural accumulation of the fæculent contents of the bowel; while, in another class of cases, noxious secretions are poured from the blood into the bowel, in consequence of the action of a morbid poison upon some of the ingredients of the blood. To this last class of cases belongs what is called *choleraic diarrhœa.*

The most reasonable theory of choleraic diarrhœa is, that a morbid poison enters the blood either with the air through the lungs, or with the food and drink through the alimentary

canal; and that this poison excites certain changes in the blood, in consequence of which some blood-materials are spoiled, and thus rendered not only useless but noxious. These morbidly changed blood-materials are then discharged from the blood-vessels through the mucous membrane of the stomach and bowels, and are ultimately ejected by vomiting and purging.

Various as are the remote and primary causes of diarrhœa, this one condition is common to all classes of cases—viz. that the contents of the bowel are unnatural and offensive. These offending materials are the immediate cause of the purging; and they must be expelled from the bowel before the diarrhœa can come to an end.[1]

From the above considerations we deduce one important and guiding rule of treatment, which is this—*not to attempt by opiates, or by other directly repressive means, to arrest a diarrhœa while there is reason to believe that the bowel contains a considerable amount of morbid and offensive materials.* It is certain that these offending materials must be cast out from the bowel before the diarrhœa can permanently cease. The effect of an opiate at this stage, if, as frequently happens, its absorption be not prevented by the rapid outward flux of liquid from the blood into the stomach and bowels, is to prolong the disease, and to increase the risk of mischief, from the retention and reabsorption of the morbid contents of the bowel. If the opiate have the effect of retaining within the blood-vessels some of the morbidly changed blood-constituents, this astringent action will probably be more injurious and even deadly than the retention of morbid secretions within the bowel.[2] The abrupt arrest of the discharges by opiates is not unfrequently followed by fatal collapse.

The purging is the natural way of getting rid of the irritant cause. We may *favour* the recovery by directing the patient to drink copiously any simple diluent liquid—water

[1] We need not here take into consideration those cases of diarrhœa which result from ulceration or other local disease of the bowel itself.

[2] I have before pointed out that, so far as we know, no drug has any *direct* influence in increasing the specific choleraic discharges, but opium, by retaining within the circulation a self-multiplying morbid poison, may *indirectly* contribute to this undesirable result.

cold or tepid, toast-water, barley-water, or weak tea; and we may often *accelerate* the recovery by sweeping out the alimentary canal by some safe purgative, and then, if necessary, soothing it by an opiate. Castor oil, notwithstanding its unpleasant taste, is, on the whole, the safest and best purgative for this purpose. It has the advantage of being very mild and unirritating, yet withal very quick in its action. A table-spoonful of the oil may be taken, floating on cold water or any other simple liquid which may be preferred by the patient. A mixture of orange-juice or of lemon-juice with water forms an agreeable vehicle for the oil. Some prefer to take it with weak brandy and water, and there is no serious objection to this mixture, but it is very likely to excite vomiting. Another mode of giving the oil is in the form of an emulsion, with mucilage and cinnamon water, which very completely covers and disguises the taste of the oil. If the dose be vomited, it should be repeated immediately; and the patient should lie still, and take no more liquid for half an hour, by which time the oil will have passed from the stomach into the bowels. Within an hour or two the oil will usually have acted freely. Then a table-spoonful of brandy may be taken in some thin arrowroot or gruel; and if there be much feeling of irritation, with a sense of sinking, from five to ten drops of laudanum may be given in cold water. These means will suffice for the speedy arrest of most cases of choleraic diarrhœa. If the patient have an insuperable objection to castor oil, or if the oil cannot be retained on the stomach, ten or fifteen grains of powdered rhubarb, or a table-spoonful of the tincture of rhubarb, or a teaspoonful of Gregory's powder, may be substituted for the oil.

When the tongue is much coated, with sallowness of the skin and eyes, headache and other symptoms of what is commonly called 'bilious derangement,' two or three grains of calomel may be combined with the rhubarb or Gregory's powder. This dose, however, is rarely necessary, and it should not be given unnecessarily.

If the diarrhœa have continued for some hours, the stools having been copious and liquid; if there be no griping pain in the bowels, no feeling or appearance of distension of the

intestines; the abdomen being flaccid and empty, and the tongue clean—we may conclude that the morbid agent has already purged itself away. There will, therefore, be no need for the castor oil or other laxative, and we may immediately give the brandy in arrowroot, and the laudanum, as before directed. The rule in all cases is, *not to give the opiate until the morbid poison and its products have for the most part escaped; not to close the door until 'the enemy' has been expelled.*[1] While there are some cases in which the evacuant dose is not required even at the commencement of the attack, there are many more in which the opiate is unnecessary in the later stage. In some cases of severe and prolonged diarrhœa, it may be necessary to repeat the oil and the laudanum alternately more than once, at intervals of three or four hours. Practical skill and tact are required to discriminate these cases. It must be borne in mind that when the choleraic secretions are being actively poured out from the blood-vessels, the bowel, though it may have been completely evacuated by a dose of oil, may quickly again become filled with morbid secretions, and hence the need for an occasional repetition of the evacuant dose.

With reference to the use of opiates in the treatment of diarrhœa, the principle to be kept constantly in view is this: *opiates are useful to soothe irritation after the evacuation of the bowel; they are useless and even dangerous when the blood is poisoned or the bowel filled with morbid secretions.* Opiates, in the early stage of diarrhœa, would be more frequently and decidedly injurious were it not for the fact that, their absorption being lessened if not entirely prevented by the active eliminative efforts, they are quickly expelled with the morbid secretions, and they are therefore powerless to arrest the discharges. There is one symptom which especially forbids the use of opium, and that symptom is *cramps of the muscles.* These cramps show that the poison which excites them is still in the blood. The painful spasms can be effectually relieved only by the escape of the poison. An opiate dose might

[1] When an opiate is reported to have at once put a stop to a choleraic attack without subsequent ill-effects, the explanation is that the dose was given after the morbid secretions had been ejected.

hinder the exit of this poison from the blood, and thereby induce that perilous, though painless, spasm of the small arteries of the lungs which is the essential cause of collapse.

It has been a common practice to give calomel and opium combined. The effect of this combination will depend upon the proportions of the ingredients. Equal weights of opium and calomel will have, for the most part, a narcotic and astringent action, but in proportion to the preponderance of the calomel the operation will be evacuant and purgative (see p. 103).

If the diarrhœa be associated with vomiting, this should be encouraged and assisted by copious draughts of tepid water. The vomiting affords relief, partly by the stimulus which it gives to the circulation, but mainly by the speedy ejection of morbid secretions.

If there be nausea without vomiting, and more especially if the stomach be supposed to contain undigested or unwholesome food or morbid secretions, an emetic may be given—either a teaspoonful of powdered mustard or a table-spoonful of common salt, or twenty grains of ipecacuanha powder in warm water.

When vomiting is excessive in violence or in frequency, it may sometimes be checked by small draughts of iced water at short intervals. Another plan is to place three grains of calomel on the tongue and wash it down with cold water. The calomel probably acts by reversing the action of the stomach and bowels, and thus causing evacuation downwards. When given as a powder it is less likely to be rejected by the act of vomiting than when made into a pill.

Thirst may be allayed by drinking cold water, which may be acidulated by the addition of lemon-juice or a few drops of dilute sulphuric acid. *Care should be taken that the water for drinking is pure.* Organic impurities, such as result from the admixture of sewage, are especially to be dreaded. If the water be of doubtful purity, it should be boiled and then carefully filtered through sand and charcoal. Impure water is a common exciting cause of cholera.

While the diarrhœa continues, the diet should consist mainly of milk, rice or arrowroot, gruel or broth, or beef-tea.

In all cases of severe diarrhœa the patient should remain in bed.

If, while the purging continues, the stools become colourless and watery (the purging being of the kind commonly called 'rice-water' purging), or if, without a continuance of the purging, the surface of the body become cold and blue, the disease is now passing, or has actually passed, into the stage of collapse.

In the worst forms of collapse, as cholera presents itself often at the outbreak of an epidemic, the disease is so deadly that no treatment is of any avail; and a sure way to bring discredit upon any method of treatment is to apply it in this desperate class of cases.

Choleraic collapse, as I have before explained, results from a peculiar arrest of the flow of blood through the lungs, occasioned by a morbid poison. It is not a condition of mere exhaustion. It is not relieved by the remedies for exhaustion; and it is made worse by opiates and by spirituous stimulants, which must, therefore, be avoided. The injurious influence of opium and alcohol in the collapse stage is now almost universally admitted. Their noxious effects are intelligible, if we bear in mind that during collapse the oxidation of the blood and tissues is greatly diminished, and that both opium and alcohol still further impede oxidation, and thus add to the peril, while opium also retards the escape of the poison from the blood and the bowels.

It is obvious that while, on the one hand, alcohol and opium are injurious, evacuants which are so speedily beneficial in the diarrhœa stage of the disease, can have no *direct* influence upon the perilous obstruction of the pulmonary circulation, which is the essential cause of collapse. Nevertheless, by judicious management we may do much to favour recovery from this alarming condition.

The collapsed patient should be strictly kept in the recumbent position; *he must not be raised even to go to stool.* He should be abundantly supplied with fresh air, and should be allowed to drink pure water freely. The water may usually be taken *cold*, but it should not be *iced*, except occasionally when given to check excessive vomiting. Large quantities of

iced water may do harm, by chilling the patient and checking the natural eliminative efforts. Some care is required not to over-distend the stomach by liquid. Unless vomiting occur from time to time, the drinking of large quantities of liquid may so distend the stomach as to impede the breathing, and thus cause much distress.

Hot flannels, or bottles, or bags of sand should be applied to the feet and legs.

Cramps in the muscles are, as a rule, more frequent and severe during the diarrhœa stage than during the stage of collapse. Whenever they occur, they may be relieved by rubbing the affected parts with the warm hand. There can be no objection to the use of anodyne or stimulating embrocations, but they are of doubtful advantage. Cramps in the legs may often be relieved by enveloping the limbs in flannel wrung out of hot water, with a covering of macintosh or oiled silk.

Hot baths, whether of water or of air, although they relieve the painful cramps and often effect a temporary improvement of the circulation, probably by warming the cooled blood in the superficial veins on its way back to the heart, have been found to be, on the whole, more distressing and exhausting than beneficial.

From ten to twenty or thirty minims of liquor ammoniæ, well diluted, may be given every hour or two as a diffusible stimulant, and as a means of preventing the coagulation of the blood in the systemic venous system.

The discharges from the bowels, and the condition of the abdomen, should be carefully observed. In favourable cases the discharges always continue, more or less, during the stage of collapse and until reaction has set in. One of the earliest and surest signs of reaction is the reappearance of bile in the vomited matters and in the stools. When vomiting and purging entirely cease during the stage of collapse, the disease is always fatal.

One of the main objects of treatment during this stage is to facilitate the escape of the morbid secretions from the alimentary canal. This may be done partly by the copious use of diluent drinks, and partly by an occasional dose of

castor oil. If we carefully observe the condition of a patient in collapse, we shall often find that the intestines are more or less distended with liquid, and this, too, while perhaps there is general torpor and little or no effort at expulsion. Again, it has often been found that, although there has been copious watery purging during life, the small intestines contain after death a large amount of a peculiar viscid dirty white material, having a very offensive odour. An occasional dose of castor oil may be useful in removing both these conditions—namely, over-distension of the bowel by liquid, and the accumulation and retention of offensive viscid semi-solid secretions.

In one or two cases I have found it necessary to combine a drop or two of croton oil with the castor oil before the distended bowel could be stimulated to expel its contents, and in more than one case even this combination has proved ineffectual.

I have no doubt that by this treatment I have rescued patients who would have died if they had been left to the unaided efforts of nature. I can lay down no rule as to the number of doses of oil that may be required. It should be borne in mind that the object and the effect of the evacuant or cleansing treatment is not to increase the amount of liquid which is poured from the blood into the stomach and bowels, but simply to assist and to quicken the expulsion of the morbid secretions from the alimentary canal.

It may be confidently maintained that, inasmuch as the peculiar choleraic discharges are the result of specific blood-changes, induced by a morbid poison, no ordinary purgative can *increase* those discharges, although it may facilitate and quicken their expulsion from the bowel.

The volume of liquid which is discharged from the stomach and bowels, is, in the diarrhœa stage, a measure of the dose or the virulence of the poison, just as, in the case of diabetes, the flux of urine is equivalent to the amount of sugar that has to be discharged through the kidneys. During the stage of collapse, the liquid discharges cease to be an index of the severity of the disease; for the reason that the partial arrest of the circulation impedes, more or less, the excretion of the

poison and its products through the alimentary canal, and in the same degree increases the risk of a fatal result.

Bleeding from the bowel sometimes occurs; and it is especially apt to occur when choleraic diarrhœa has been kept up for some days by continued exposure to the exciting cause, whether contaminated air or water. It is a very unfavourable and usually a fatal symptom. The best treatment consists in giving twenty drops of oil of turpentine in mucilage every two hours, and iced water may be taken freely. *In such a case no castor oil should be given.*

After reaction has occurred, an occasional laxative dose is required—about once in the twenty-four hours during the first two or three days.

It is worse than useless to attempt to *feed* a patient during collapse. The secretions of the stomach are utterly deranged; and the power of digestion is suspended. The mildest nourishment administered at this time only adds to the feeling of oppression and general distress, from which the act of vomiting often gives immediate relief.

After reaction is established, and when the normal secretions are restored, the most easily digestible food should be given frequently, but in small quantities—such as milk, gruel, or rice, or arrowroot, with a small quantity of brandy, soup or beef-tea, or chicken-broth. After an attack of cholera the stomach is sometimes long in recovering its tone and the power to digest solid food. When this is the case, a grain of quinine, with three minims of liquor strychniæ, ten drops of dilute hydrochloric acid, and an equal quantity of spirit of chloroform, may be taken with each meal.

Venesection has often afforded great relief during the stage of collapse, and in section iii. I have quoted some remarkable instances of great and instantaneous relief from the operation.

The symptom which appears especially to call for venesection is rapid breathing, with a feeling of impending suffocation and pain in the region of the heart. When, with these symptoms, there is a cessation of vomiting and purging, which is probably a result of the almost entire arrest of the circulation through the lungs, venesection may prove a life-

saving remedy. It is difficult to obtain a stream of blood in these cases; not, as many suppose, because the blood is too thick to flow, but because, in consequence of the block in the lungs, the blood in the veins is nearly stagnant.[1] The bleeding appears to be beneficial, partly by relaxing spasm and withdrawing some of the poisoned blood, but chiefly by lessening the distension of the right cavities of the heart, and so increasing their contractile power. Repeated doses of ammonia may help to quicken the circulation, and so favour the flow of blood and prevent its coagulation.

Hot saline injections into the veins have unquestionably saved life in some apparently desperate cases. This is an operation the performance of which requires great skill and care. I have already explained its mode of action (p. 128). In the condition of pulseless collapse with arrest of the discharges and extreme dyspnœa, and especially when venesection has been found to be impracticable, an injection of warm fluid is the only possible means of rescuing the patient, and therefore it ought to be resorted to and sometimes even repeated.

Consecutive Fever.—Reaction from collapse is frequently followed by a febrile condition—a hot skin, quick pulse, coated tongue, hurried breathing, often a scanty secretion or even a complete suppression of urine, with drowsiness tending to pass into coma. These unfavourable symptoms are more common when, during the earlier stages of the disease, opium and alcoholic stimulants have been freely given; but they may occur when no such means have been employed, and especially when the state of collapse has been long continued.

[1] Scot, referring to the difficulty of obtaining blood in some cases, says (*Report on the Epidemic Cholera*, p. lix): 'When, however, the operation is performed with the moral conviction that if successful in obtaining blood the life of the patient will most probably be saved, the operator will persevere, undiscouraged, in his efforts; he will call in every suitable aid, such as frictions, bathing the arm in hot water, re-opening the orifices of the vessels, administering stimulants, and applying external warmth. He is not deterred and induced to desist by any intermediate accession of debility or collapse, nor is he tempted to rest satisfied with any temporary amelioration of the pulse; his object goes beyond the present moment, and he feels satisfied that if he can fully unload the internal vessels he will save his patient, and if he fails that he will most probably lose him.'

The best treatment consists in a scanty liquid diet *without alcohol*, copious diluent drinks, with saline effervescing draughts, an occasional aperient, castor oil, sulphate of magnesia or a seidlitz powder, counter-irritation over the lungs and kidneys, and sometimes local bleeding to relieve congestion of those organs.

When the urine is suppressed or scanty, one of the safest and most efficacious diuretics is the 'Imperial Drink,' made as follows:—A quarter of an ounce of cream of tartar, the juice of one lemon, the outer peel of half a lemon, a pint of boiling water, and sugar *ad libitum*. One or two pints of this may be taken cold in the course of the twenty-four hours. Alcoholic stimulants should be withheld until the function of the kidneys has been completely restored.

In some cases there is complaint of pain in the region of the stomach during convalescence. This may be relieved by the application of a few leeches over the seat of pain, or sometimes by a mustard poultice. If, after reaction, the stomach remain irritable, with frequent vomiting, iced water is an agreeable and efficacious remedy.

It will be seen that the treatment which I recommend, and which has been practised by myself and others with a most encouraging amount of success, is not a mere system of drug-giving, but a definite plan based upon a careful study of the natural history of the disease. I am confident that anyone who, having an intelligent appreciation of the pathological conditions to be dealt with, will carry out this method of treatment with the same determination to arrive at a definite result as that with which he would treat a case of poisoning by opium, will soon have better reasons for continuing the practice than any with which I can furnish him. For the full success of the treatment it is necessary that the practitioner himself should have that confidence in the method, without which it can never be fairly tried, and that he should inspire his assistants, and above all his patients, with hope and confidence.

Preventive Measures.—The choleraic discharges from the bowels, although not poisonous when fresh, rapidly become so when they begin to decompose. If they have been long

retained within the bowel, they may have reached the poisonous stage of decomposition before they escape, and they should be disinfected and got rid of as soon as possible. Every vessel and article of clothing or bedding soiled by the discharges should be carefully cleansed and disinfected. The attendants on the sick should be warned of the necessity for thorough ventilation and for extreme personal cleanliness. The hands should be frequently cleansed with the aid of disinfectants, and always immediately before taking food.

If these simple measures be adopted, nurses and other attendants on the sick run little risk from infection.

The chief disinfectants are chloride of lime, Burnett's liquid, Condy's fluid, and Calvert's solution of carbolic acid. These disinfectants are sold with full printed directions for the use of each. Condy's fluid is well adapted for cleansing the mouth and hands before taking food; and carbolic acid for cleansing bedding and clothing, which would be damaged by mineral disinfectants. Any one of the above may be used for disinfecting the stools.

A concentrated solution of sulphate of iron is also a cheap and efficient disinfectant for the discharges.

The meals should be taken at regular and not too great intervals. Long fasting and over-fatigue should be carefully avoided.

Great moderation both in food and in drink is essential for safety during an epidemic of cholera. A single act of indiscretion has been followed by a severe attack. Intemperance at such a time is fraught with extreme danger.

Unwholesome articles of food, more especially tainted meat and fish and decayed vegetables, are to be carefully avoided. It is a mistake to suppose that entire abstinence from fruit and vegetables is necessary or wholesome during a cholera season. Ripe fruit and fresh vegetables may be taken in moderation with safety and advantage.

Especial attention should be paid to ensure the cleanliness and thorough ventilation of dwelling-houses. All vegetable and animal refuse should be removed as speedily as possible. Care should be taken to prevent the escape of sewer gases into the interior of dwellings.

The purity of the water employed for drinking and cooking should be most carefully provided for. A few drops of Condy's fluid may be used as a test for the purity of water. Organic impurities in the course of an hour or two decolourise the fluid; which is not only a test, but also a purifying agent, by oxidising the organic impurities. It is certain that a diarrhœa is often perpetuated by the daily drinking of impure water; and it is equally certain that to arrest a diarrhœa having such an origin by opiates and astringents is a most dangerous practice. I have seen instances in which a diarrhœa, probably caused by drinking foul water, having been arrested by opium, was quickly followed by an attack of cholera of the most formidable character.

No unnecessary medicines of any kind should be taken. When opening medicine is required, the mildest should be selected, such as castor oil or rhubarb. Saline purgatives, such as Glauber's salts and Epsom salts, are objectionable on account of their tendency to cause profuse watery purging. On the other hand, the common belief that prolonged constipation should not be interfered with during the prevalence of cholera is an error. An accumulation of offensive materials within the bowels may be itself a source of irritation and of danger.

Many persons, during an epidemic season, have an unpleasant feeling of commotion with a rumbling noise in the bowels and a sense of sinking. This discomfort is often removed by a tonic, such as a teaspoonful of tincture of quinine in water, twice or thrice a day before food. Sometimes ten drops of aromatic sulphuric or dilute hydrochloric acid may be usefully combined with each dose of the quinine. Another useful tonic in these cases, more especially when there is a tendency to looseness of the bowels, is a combination of twenty drops of the muriated tincture of iron with a teaspoonful of tincture or syrup of ginger in an ounce of water; and this dose may be taken *after* each meal. The slight bowel derangement just mentioned will certainly be made worse by laxative medicines; and I repeat that, *during an epidemic of cholera, no unnecessary medicine of any kind should be taken, and as a rule, none without medical advice.*

Some practitioners believe and assert that cholera collapse may be caused by a dose of purgative medicine. This is quite incredible. It must have happened frequently that an individual, experiencing the malaise which results from the cholera poison in the system, takes a dose of opening medicine; the symptoms of cholera develop themselves, and the disease may be supposed to have been caused by the medicine; yet such a conclusion would be as inconsistent with facts and with analogy as to attribute the outcoming of the rash of scarlatina to a mustard poultice applied to the neck for the relief of the sore-throat which commonly marks the commencement of that disease. Cholera and scarlatina are both specific contagious diseases: one could no more be caused by a purgative than the other by a mustard poultice.

With military authorities in India it is now an established practice, when cholera breaks out amongst troops in a cantonment, to march the men to a distant encampment. In this way the local causes of the disease in the contaminated soil and water are escaped from, and the spread of the malady through a regiment is arrested. A similar practical result is not unfrequently arrived at when, an outbreak of cholera having occurred in a native village, the whole of the inhabitants desert their homes and go for some days on a pilgrimage to worship at the shrine of some saint or deity. Whatever we may think of this proceeding from a religious point of view, there can be little doubt that as a sanitary measure it is often successful, and the natives appeal to the satisfactory results as a justification of the practice and a reason for its repetition when the occasion arises.

It is important to know that cholera may be imported into a district by a person coming from an infected locality while he is suffering from diarrhœa. The diarrhœa may be a mild form of cholera, and the discharges contain the specific poison of the disease. If these discharges are permitted to contaminate the soil, the water, and the air, the disease may spread with destructive rapidity. To prevent an outbreak of cholera, the diarrhœa patient should, if possible, be kept in quarantine, the discharges should be disinfected, and then buried, and all

soiled clothing and bedding should be carefully disinfected and cleansed, or burnt.

Public latrines in a district where cholera prevails are highly dangerous, and a fruitful source of infection. The use of latrines by British troops in India has often led to a great mortality, while the native troops, who rarely resort to latrines, have either escaped entirely or have suffered in a much less degree at the same time and place.

The early burial of those who have died from cholera is imperative as a sanitary measure, more especially in a tropical climate, where decomposition is extremely rapid. Of course care must be taken to ascertain that life is really extinct before burial takes place. And here it may be well to repeat that there are two *post-mortem* phenomena which the inexperienced might mistake for signs of vitality. The internal heat of the body comes to the surface after death from cholera, so that for an hour or two the temperature of the skin may be higher than it was for some hours before death. Again, it not unfrequently happens that for half an hour or so after death some muscles, probably stimulated by the poisoned blood which excited the painful cramps during life, are affected by irregular contractions which may be sufficiently powerful and concerted to move a finger, and even a limb. These purely physical *post-mortem* movements have often excited a vague terror, and a suspicion that life remains in the moving corpse.

In conclusion, let me once more emphasise the statement that there is no fact in the history of cholera more certain than that the disease is bred and nourished in filth. A foul leaven enters the body, either with the air or with the food and drink, and then excites mysterious fermentative changes in the fluids and tissues of the organism.

The most successful preventive measures are those which are the earliest applied, and which are the most far-reaching in their operation. To purify the air that we breathe and the water that we drink is often an easier and always a more effective proceeding than the endeavour to free the body from the loathsome ferment when once it has gained access to the circulating fluids.

The knowledge that we now possess of the conditions which engender the poison, and the mode in which it enters and operates on the human body, can scarcely fail to convince every reflecting mind how vain is the hope of discovering a specific cure for cholera—how mischievous the indiscriminate administration of active drugs, and the reckless treatment of symptoms without reference to their origin and their significance.

The chief object of rational medicine is, while avoiding all violent means and all mischievous medication, to make the treatment of cholera, as of other diseases, conformable to what is known of the nature of the malady and of the natural process of cure. For in this, as in many other acute and fatal diseases, there *is* a natural curative process; and the highest art of the physician consists in learning with respect to each disease what that process is; so that he may be enabled to assist, and, if need be, to regulate it, while he carefully avoids all such means as tend directly to repress or to oppose the curative efforts of nature.

EPILOGUE.

Since this chapter was in type I find that the late Dr. Fagge[1] maintained that my theory of cholera collapse must be erroneous, because suppression of urine and other symptoms of collapse occur in cases of perforation of the stomach and other allied conditions. I have already shown (p. 139) that the two forms of collapse are essentially different, notwithstanding their superficial resemblance. It is obvious that the arrest or failure of the circulation, whether caused by a fibrinous clot in the trunk of the pulmonary artery, as in Dr. Carpenter's case before quoted (p. 122), by contraction of the pulmonary arterioles, or by cardiac weakness from nervous shock, would, in proportion to its degree, be associated with the same chemical and physiological results of defective oxidation and suspended secretion. But to argue that, therefore, the pathological conditions must be, in all respects, the same, is about as convincing as would be the contention that retention of urine from paralysis of the bladder is identical with that from stricture of the urethra.

[1] *The Principles and Practice of Medicine*, vol. i. pp. 298–9.

CHAPTER VII.

LECTURE ON DELIRIUM TREMENS, ITS SYMPTOMS, PATHOLOGY, AND TREATMENT.[1]

Symptoms—Dreams—Spectral Visions, &c.—Occasional Violence—The Delirium often begins after Alcohol has been Withdrawn—Traumatic Delirium—Tendency to Suicide—Sudden Death from Struggling with Attendants—Heart often Fat and Weak—Urine often Albuminous—Delirium from Exhaustion apart from Use of Alcohol—Various Modes of Treatment in Accordance with Pathological Theories—General Rules for Treatment—Guard against Suicide—Best Means of Restraint—Feeding is of Prime Necessity—Cautious Use of Opium—Danger of Large Doses—Alcohol—Chloroform—Chloral—Tincture of Digitalis—Preventive Measures.

Those who suffer from this disease have a peculiar form of delirium, accompanied with spectral visions and a general unsteadiness and trembling of the voluntary muscles. They are obstinately wakeful. In a large proportion of cases the patient has habitually or fitfully indulged in excessive quantities of alcoholic stimulants, taking at the same time a comparatively small amount of solid nutriment. In some few exceptional instances, however, over-work, grief, disappointment, and anxiety have been the exciting causes of a similar form of delirium in persons of strictly temperate habits.

I purpose now to describe the symptoms of this disease, its essential causes, the peculiar dangers which attend it, and the means of cure.

Delirium tremens is very common amongst sailors, who give it the significant name of the 'horrors,' and the history of a case is often somewhat of this kind. A sailor who has been long at sea comes on shore with a plentiful supply of money in his pocket, and determines to enjoy himself in his

[1] Published in the *Lancet*, April 21, 1886.

own peculiar fashion. This consists principally in keeping himself incessantly drunk for several days in succession, during which time he eats scarcely any solid food. At the end of a week or two, having spent all his money and perhaps pawned his watch, the further supply of drink is stopped. He now recovers from the stupefying effects of the alcohol, and, reflecting upon his folly and the emptiness of his pockets with feelings of vexation, shame, and remorse, he becomes more and more fretful and excited, and soon, with an enfeebled body and an irritated mind, he comes thoroughly under the influence of 'the horrors.' He becomes very wakeful—in fact, he cannot sleep at all; he is delirious, but the delirium is usually not of a violent or mischievous character; he is talking incessantly and incoherently on a variety of subjects, sometimes to persons who are actually with him, but very commonly to persons who have no existence except in his diseased imagination, or who, at any rate, are not within sight or hearing. Usually he will answer questions correctly when they are put to him, will put out his tongue, or do whatever you bid him to do, in a tremulous and agitated manner, then quickly he wanders away into a region of shadows and fictions. He is troubled by spectral visions of various forms —rats and mice and reptiles are creeping over his bed; dogs and devils are dancing about the room, and frequently threatening him with teeth and claws. If by chance he fall to sleep for a time, his distress appears only to be increased: he is disturbed by terrific dreams, he moans incessantly, and not unfrequently wakes himself with a scream of terror, and he may then rush frantically towards the door or window to escape from some imaginary pursuer.

After a patient's recovery he often remembers distinctly the nature of his past dreams and delusions. One man, who had screamed loud enough to disturb the whole hospital, told me that while sleeping he thought he was in hell, and that he was being tortured by having red-hot irons thrust through his body. Another fancied that he was drowning, and that the devil came to help him out of the water. The alternative of drowning or falling into the hands of such a helper drew from him a scream which aroused the entire household.

The patient is often troubled and anxious about business matters. He will be incessantly getting out of bed and attempting to find his clothes, in order that he may start on some affair which requires his immediate presence; and if he cannot find his clothes, he will attempt to go without them. Some time since I had under my care—a lady I was about to say—at any rate, a woman, who, one Sunday morning, just as people were returning from church, rushed out into the street in her night-dress. One peculiarity in these cases is, that a patient, when not actually in terror of some spectral enemy, frequently appears to be amused at his own perplexities. Thus I have seen a man making ineffectual attempts to pick up money which he thought he saw on the bed, and occasionally remarking upon the extraordinary fact that he could not feel the coin which he so distinctly saw—a state of mental perplexity like that of Macbeth when he could not clutch the dagger which he plainly saw before him. The sight is not the only sense which is thus mocked and deluded. The patient often hears voices calling from the wall or the ceiling, and not unfrequently replies to imaginary addresses.

I have said that the delirium is not usually of a violent or mischievous character; but occasionally, when a patient is opposed in his attempt to get up or to leave the room, he becomes angry and violent. When I was house physician, a powerful man contrived to slip out of a strait-waistcoat by which he had been restrained during the night; and with a determination to escape he armed himself with a long metal tube. He then banged open the door of a room where some of the hospital servants were sleeping, and floored the nurse and one patient before he could be secured.

Some features of the disease yet remain to be mentioned. There is a peculiar tremulousness of the voluntary muscles; the hands are unsteady; the muscles of the face and of the tongue quiver, so that the voice is tremulous like that of a man suffering from fright or just emerging from a very cold bath. The tongue is usually moist and coated, the face is often flushed, and the eyes are injected—'red and ferrety,' as the term goes (white ferrets commonly having pink eyes). The pulse is quick, but soft and compressible; the skin is usually

moist, and not unfrequently bathed in a profuse sweat. Very rarely does the patient complain of headache, or, indeed, of pain of any kind. While he is haunted by imaginary evils his sensibilities are blunted, and he appears to have but little consciousness of physical suffering. This is an important fact to bear in mind, and it shows the necessity for watchfulness lest some serious disease escape detection. In one case which came under my observation, extensive hepatisation of one lung was not discovered until after death. There had been no cough or dyspnœa or complaint of pain to direct attention to the chest; and a physical examination, by which alone the disease could have been discovered, was not made.

Our typical sailor, you will remember, became delirious when he had *ceased* to drink; and this is the fact in a large proportion of instances. The habitual drinker learns from experience that he can ward off a threatened attack by a continual recurrence to the bottle. His nervous system is composed and his muscles are steadied by a frequent repetition of the customary dram. An enforced abstinence is quickly followed by an attack of delirium tremens. In some instances the unhappy man has continued to drink until the stomach—fretted and inflamed by excess of alcohol—will no longer retain either food or stimulants; then follows extreme bodily exhaustion with great mental excitement and delirium. In some cases, however, the attack is *not* preceded by a withdrawal or diminution of the stimulus; it seizes upon men who continually drink to excess; but rarely when the appetite and digestion have continued good. Sometimes we find that the immediate exciting cause has been some sudden anxiety or disappointment; in other cases the delirium follows quickly upon some injury or illness which is at once a source of exhaustion and of irritation and distress. For instance, an habitual toper breaks his leg, and, in the course of a day or two, he becomes delirious. With apparently an almost entire insensibility to physical pain, he will, if not carefully watched, tear off the bandages and dance about upon the broken limb. This is what the surgeons call *traumatic delirium*. It is, in fact, delirium tremens. The traumatic element is not an essential one in the causation of the disease. An attack of

pneumonia, or pleurisy, or pericarditis, or of almost any other acute disease, would just as surely as the wounded limb have acted as the exciting cause of the delirium in a subject predisposed by habitual intemperance.

I have told you that a patient with delirium tremens is rarely mischievous or violent to *others*; much more commonly he is dangerous to *himself*. A tendency to suicide is very common; and this danger we have always to remember, and to guard against.

Another fact important to be borne in mind is that any violent exertion on the part of the patient, such as running fast or struggling with an attendant, or such violent efforts as he will sometimes make to free himself from the restraint of a strait-waistcoat—any violent exertion of this kind is apt to induce rapid and great exhaustion, and even sudden death.

Dr. Budd was in the habit of mentioning in his lectures a tragedy which occurred at the 'Dreadnought' when he was physician to that hospital-ship, and which serves as a good and impressive illustration of the two dangers to which I have alluded—namely, the danger of suicide, and the danger resulting from any violent exertion on the part of the patient. Three sailors suffering from delirium tremens were in the 'Dreadnought' at one time. One of them suddenly cut his throat; and a second, excited and alarmed by the commotion in the ward which this event occasioned, jumped through a port-hole into the river, and swam away vigorously towards the shore. They immediately put after him in a boat, and picked him up. Having brought him to the ship's side, they were about to take him up the ladder, when he made a violent resistance, and in doing so suddenly fell dead in the boat.

The tendency to sudden death from exhaustion is accounted for by the condition of the *heart*. In nearly every fatal case of delirium tremens which has occurred since the attention of the morbid anatomist has been directed to the subject of fatty degeneration of the heart, it has been found that this change in the muscular structure of the organ exists to a greater or less extent, and not unfrequently in an extreme degree. The explanation of this fatty degeneration of the walls of the heart is probably to be found in the peculiar diet of these

patients—a diet abounding in hydro-carbon, and deficient in the nitrogenous materials which are contained in the fibre of meat, and which are required for the continued renewal of the constantly acting muscular tissue of the heart.

This degeneration of the muscular tissue of the heart is the most constant and the most important structural change discernible after death. The brain and its membranes are usually quite healthy. Sometimes there is an appearance of increased fulness of the blood-vessels, and there may be some serous effusion beneath the arachnoid in the meshes of the pia mater. Occasionally, too, though less frequently, the arachnoid is found more or less opaque and thickened. These, however, are only occasional and accidental appearances; they are not of the essence of the disease: for they are found when there has been no symptom of delirium tremens; and, on the other hand, they are wanting in the great majority of fatal cases of the disease.

Delirium tremens does not depend on inflammation of the brain and its membranes, as it was formerly supposed to do. It may, however, be complicated with inflammatory changes; and it is surprising that this inflammatory complication is not more frequent than it is actually found to be. If we consider the vast amount of alcohol consumed by an habitual drunkard; the obvious influence which it has in disturbing the *functions* of the brain; and the great affinity which appears to exist between it and the cerebral tissue, as shown by the fact (which Dr. Percy was the first to discover) that alcohol may be obtained in considerable amount from the brain of a dog that has been poisoned by it: these facts would naturally lead one to anticipate that inflammation of the brain and its membranes would be a frequent result of alcoholic intoxication. The reverse, however, is the case. I remember to have seen only one case in which, after death with symptoms of delirium tremens, there were decided indications of meningitis in an effusion of lymph as well as serum beneath the arachnoid.

How are we to tell during life that there is inflammation of the brain or its membranes in addition to the symptoms of delirium tremens? Not very easily, I can assure you. There are certain symptoms which would show the existence of

something more than an ordinary attack of delirium tremens: fixed pain in the head; convulsions, either general or partial; paralysis; inequality of the pupils; strabismus; unusual slowness of the pulse. You will get no help from the state of the skin or tongue, whether moist or dry; they may be one or the other in either disease. Neither will the colour of the countenance assist you in the diagnosis; it is almost always as flushed and red in delirium tremens as it is possible to be in cases of inflammation of the brain. You are not to suppose that a delirious patient has inflammation of the brain merely because his face is flushed.

You should never omit to examine the *urine* in cases of delirium tremens. Drunkards are frequently the subjects of renal disease; and an attack of convulsions, or symptoms of great cerebral oppression, which might be indicative of inflammation of the brain, may also be a result of uræmia consequent on degeneration of the kidney.

Delirium tremens is more common in men than in women, in proportion as they are more exposed to its exciting causes, but it is by no means uncommon in women, and the symptoms are essentially the same in the two sexes. Although, as I have before said, the sufferers from delirium tremens are, in the great majority of cases, persons of intemperate habits, so that the disease has with good reason been designated *delirium e potu*, yet we sometimes meet with cases of delirium of essentially the same character, and requiring precisely the same plan of treatment, in the persons of those who have never drunk to excess. I could give several illustrations of this doctrine from my own experience, but I prefer to quote from Dr. Abercrombie a case which you will find fully recorded, under the head of Meningitis, in his work on *Diseases of the Brain*. The outline of the case is briefly this.

A lady aged 38, after the birth of her eleventh child, had a deep-seated painful swelling in the pelvis. This was treated by repeated topical bleeding. The result was, that after three or four weeks her strength was much reduced, and she was still confined to her bed. At this time she was alarmed and agitated by some family occurrence, and immediately she began to talk wildly and incoherently. She con-

tinued to talk incessantly the whole night. The next day the symptoms rapidly yielded to treatment by stimulants. A glass of wine was given every hour. After the fourth glass she was perfectly composed and rational.

Some pathologists would deny that this was really a case of delirium tremens; they would call it delirium from exhaustion, which it unquestionably was, as the history of the disease and the result of treatment clearly show. But I maintain that delirium tremens is also essentially an instance of delirium from exhaustion, and that the direct action of alcohol is *not* an essential element. What is common to all forms of delirium from exhaustion, and what therefore is essential, is this, that there is a mental and a bodily element—some grief or disappointment, vexation, anxiety, or terror, acting upon an enfeebled body. The delirium of intoxication is entirely distinct from that form of delirium which we call delirium tremens. The delirium of intoxication is a direct effect of the presence of alcohol in the blood; whereas alcohol is only *indirectly* concerned in the causation of delirium tremens. An habitual excess of alcohol tends to impair the nutrition of the brain and to exhaust the powers of the body by excluding wholesome food and deranging the digestive process, and in this way, rather than by any direct toxæmic influence, it acts as a predisposing cause of delirium tremens.

The *treatment* of delirium tremens has passed through various phases, and undergone various modifications, in accordance with the theories which have at different times prevailed with respect to the nature of the malady. Until within the last seventy years, it was generally looked upon as an inflammatory disease. Many of the cases which the older writers called 'phrenitis' were, in fact, cases of delirium tremens. The treatment then was by bleeding and other antiphlogistic remedies, and the result was most unfavourable. In the year 1813, Mr. Thomas Sutton, of Greenwich, published a tract in which he distinguished these cases of delirium, so commonly occurring in persons of intemperate habits, from cases of inflammation of the brain and its membranes. He first applied the term 'delirium tremens' to the former class of cases, and remarked upon it that the name, though not perhaps the

most appropriate, would serve to convey a notion of an essential symptom of the disease, and was sufficiently explicit for his purpose. He states that from the year 1798 to 1807 he resided on the eastern coast of Kent, first as physician to the forces, and afterwards as a private practitioner. He there found that delirium tremens was treated in two very distinct ways by different practitioners; the one party employing antiphlogistic remedies, the other using narcotics: and, as he says, he 'soon perceived that the latter practice carried with it all the success.' He adopted the successful mode of practice, and, after giving it a fair trial, published a very sensible treatise on the disease. He states that in the course of three years he had treated twenty-two cases of delirium tremens, and that only four of these had died. He estimates the mortality under the antiphlogistic treatment as two out of three.

Since that time the treatment of delirium tremens by narcotics, and especially by opium, has been gradually established as the orthodox practice; and it is, on the whole, a successful practice. But it does not always succeed. The doctrine that 'opium is to be given in large doses, and repeated fearlessly until the patient sleeps, is a dangerous doctrine, and must often have led to a fatal result. There are many patients who would die from the depressing effect of opium on the heart without being made sleepy by the drug. I had this fact deeply impressed upon my mind many years ago, when I was house-physician to the hospital. A man who had taken repeated large doses of opium sank rapidly exhausted without a symptom of narcotism, consciousness remaining until within a few minutes of his death.

Of late years Dr. Peddie,[1] of Edinburgh, Dr. Laycock, and some other practitioners have attributed the symptoms of delirium tremens to the direct poisonous influence of alcohol upon the brain, and in accordance with this view they have treated the disease without opium or alcohol, and have given diaphoretics, such as tartar emetic, which is supposed also to exert a sedative influence on the brain. The successful

[1] *Monthly Journal of Medical Science*, 1854.

result of this treatment, at any rate, proves that opium and alcohol are not necessary for the cure of delirium tremens.

The three chief modes of treatment, therefore, which have been pursued in this disease are :—

(1) The antiphlogistic, now by general consent abandoned.

(2) The treatment by opium, which is in very general use, and often highly successful; but which is attended with peculiar risks, the nature of which I will presently endeavour to point out to you.

(3) The eliminative treatment, with abstinence from alcohol and opium.

I will now tell you what, in my opinion, are the main points to be attended to in the treatment of delirium tremens, and what are the peculiar dangers which have to be guarded against.

And first, bearing in mind the tendency to suicide, the patient must be constantly and carefully watched by a judicious attendant who has the power to exercise some moral control over him. The doors and windows should be secured, and razors, knives, and other means of mischief, put out of sight and reach. He should be kept quiet and undisturbed by noise or excess of light. It is better, if possible, to avoid direct physical restraint. If the patient be violent and difficult to control, secure the services of two attendants rather than have recourse to a strait-waistcoat. I have known more than one instance of sudden death from exhaustion induced by violent struggling against the restraint of the strait-waistcoat, and I have also seen more than one case of fatal exhaustion from struggling with injudicious attendants. Not long since a medical friend called one morning and desired me to go with him immediately to visit a publican whom he had just before seen with an attack of delirium tremens. On entering the room we found the patient gasping for breath and evidently dying. We learnt that immediately after my friend had left his patient, less than half an hour before, the man got out of bed and wished to leave the room. Three strong men who were present resisted his attempt to go out; a struggle ensued, and in the midst of it he suddenly fell exhausted as we saw him, and he quickly ceased to breathe.

You see, then, how great is the risk resulting from violent exertion on the part of these patients, and how important it is to avoid any restraint which is likely to provoke a struggle. In some instances a patient who cannot otherwise be kept in will remain perfectly quiet under the restraint of the waistcoat; but while he is thus restrained he must on no account be left alone. He may begin to struggle at any moment, and this struggle may cause rapid and fatal exhaustion.

To sum up, then, what I have said on the subject of restraint, the guiding principle is to prevent any violent exertion on the part of the patient—any struggle either with the strait-waistcoat or with the attendants. A padded room, when it can be had, is the safest and most effectual means of restraint for a violent patient.

Having ensured the safe custody of the patient, we have next to consider other methods of treatment. Now, in the first place, bear in mind that delirium tremens is one of those diseases from which recovery may take place without any assistance from drugs. Medicines are of great use in the treatment; but they are not essential. It is, however, of essential importance that the delirious patient should be *fed*. The history of the disease teaches us that the immediate cause of the delirium is the exhaustion consequent on a deficiency of wholesome nutriment; and, until this deficiency be supplied, all attempts to procure the wished-for sleep, which is so generally followed by a cessation of the exitement, are often useless and worse than useless.

Whatever else you do or leave undone, never neglect to feed a delirious patient. If he will take food when he first comes under treatment, give it immediately, and let it be repeated as soon as he will take it. If there be, as there often is, a disinclination for food, with nausea and a coated tongue, an emetic of ipecacuanha, followed by a dose of calomel and colocynth, or a saline laxative, will be of use as a preparation for food and for the opiate, which may best be given at bedtime. You may then give thirty or forty minims of tincture of opium, which I believe to be a better soporific in these cases than the salts of morphine, and a smaller dose may be

given in three or four hours if necessary. The first object is to secure sleep. But here I wish to impress upon you the necessity of great care and watchfulness. Do not make rash attempts to force on sleep by repeated large doses of opium. These attempts will often fail to procure sleep, and they may kill the patient. It is an undoubted fact, that opium in many of these cases has no soporific effect whatsoever. And not only in cases of delirium tremens is opium uncertain in its operation. When we give opium in ordinary cases of disease as an anodyne or soporific, we can never be sure that it will cause sleep. In a considerable proportion of cases opium *prevents* sleep, and makes the patient more wakeful than he would have been without it. Another not uncommon effect of an opiate is to cause nausea and faintness. These two effects of opium should always be borne in mind in the treatment of delirium tremens. Remember that when opium fails to act as a soporific in delirium tremens it is not inert, and must not be given in repeated large doses as if it were. While it fails to procure sleep, it may be exerting a powerful depressing and paralysing influence upon the heart. The symptoms indicating that opium is thus acting on the heart are these:—The patient continues wakeful, excited, and delirious, but grows rapidly weaker; the pulse becomes quick, small, and feeble; the pupils are contracted; the skin is bathed in a profuse sweat; and if the opium be continued in large and frequent doses, the patient rapidly sinks, but remains wakeful and conscious until perhaps within a few minutes of the end. The opium in these cases acts as a powerful sedative on the heart, and in proportion as it does this it fails to exercise any soporific influence. If you find that opium is acting thus injuriously, you must immediately discontinue it, and give liberal doses of brandy, or the stimulant to which the patient has been accustomed. Full doses of quinine, too, will be of use as a tonic; and nutriment, either in the liquid or solid form, should be freely given.

It is an undoubted fact that the soporific effect of opium is often much more certain and decided when the medicine is given *with some alcoholic stimulant* than when it is given alone: and I have seen not a few cases of delirium tremens

in which, after repeated large doses of opium have failed to procure sleep, a liberal allowance of the patient's accustomed stimulant, more especially when combined with food, has been followed by a long sleep, and this by an entire freedom from delusions and delirium. I scarcely need impress upon you that these unquestionable facts are quite irreconcilable with the hypothesis that the proximate and essential cause of delirium tremens is the presence of alcohol in the blood. If the patient refuse to take the opiate or other medicine which you are anxious to give, he may sometimes be induced to take it mixed with his beer or other beverage. Both food and stimulants may be given by the rectum in case of need; and morphine may be introduced hypodermically; but this mode of giving morphine requires at least as much caution as the administration of opiates by the mouth.

Chloroform vapour has the immediate effect of quieting the delirium and excitement; but the quiescence in most cases is only temporary. Occasionally, however, after one or two repetitions of the anæsthetic the patient sinks into a quiet slumber of some hours' duration, and when he awakes the delirium and excitement have passed away. The fat and flabby state of the heart in these cases calls for extreme care in the administration of chloroform.

The hydrate of chloral in doses of from twenty to forty grains often acts very speedily and satisfactorily.

When there is much flushing of the face and heat of the scalp, cold lotions or iced water in a bladder may be applied to the head with advantage.

You may have heard or read of large doses of tincture of digitalis having been given for the cure of delirium tremens. I consider this a very dangerous remedy. I have no doubt that some patients have recovered in spite of the treatment. When half-ounce doses of the tincture have been given, it is to be hoped that the digitalis was of bad quality: the patient would then, at any rate, have the benefit to be derived from the stimulant action of the rectified spirit. I have heard of several instances of sudden death after the administration of large doses of digitalis, and it is only natural that these calamitous results should not, as a rule, obtain equal publicity

with the cases of apparently successful treatment. A consideration of the natural history of delirium tremens suffices, I think, to show that the treatment by large doses of digitalis is irrational and dangerous.

To sum up, then, the main practical points which I have endeavoured to impress upon you. Bear in mind that an attack of delirium tremens, as a rule, subsides in a few days without help from medicines of any kind. Let the patient be carefully watched, and guarded, and fed, and the excitement and delirium will gradually subside. In many instances opium is a valuable aid in the treatment, and it will often cut short the disease; but remember that it is powerful for evil as well as for good. Its use, therefore, requires much care and discrimination. The hydrate of chloral often acts very speedily and satisfactorily, and may therefore sometimes be preferred to opium. Some form of alcoholic stimulant will often be of great use in calming the nervous excitement and procuring sleep.

Lastly, when the patient has recovered from his attack, do not fail to impress upon him the moral lesson which his sufferings have been designed to convey. The tortures of delirium tremens are calculated to exercise a wholesome deterring influence on the self-indulgent and the intemperate, and in that light he should be taught to view them. The poet Wordsworth has given the true interpretation of the spectral visions which form so marked a feature of delirium tremens, in the following lines from his *Sonnets on the Punishment of Death*:—

> Ye brood of conscience—Spectres! that frequent
> The bad man's restless walk and haunt his bed,—
> Fiends in your aspect, yet beneficent
> In act, as hovering angels when they spread
> Their wings to guard the unconscious Innocent.

The only hope for him who has once been the subject of delirium tremens is in total abstinence, which for such a man is easier than temperance. Some years since I had under my care in the hospital a man with delirium tremens whom I persuaded to become a teetotaler. Seven years afterwards he was re-admitted with the same disease. He then de-

cleared—and I believe that he spoke the truth—that since he left the hospital he had been a total abstainer until a fortnight before his re-admission, when, on the occasion of his son's emigrating, he began to drink a little, and this led on to continued excess for several days, ending in an attack of his former malady.

CHAPTER VIII.

ON SOME NERVOUS DISORDERS THAT RESULT FROM OVERWORK OR MENTAL SHOCK AND ANXIETY.[1]

SECTION I.

CASE 1. Distressing Nervous Symptoms of a Year's Duration, the Result of a Carriage Accident, removed by a Few Doses of Chloral at Bedtime.—CASE 2. Epilepsy and other grave Nervous Disorders of Seven Years' Duration, the Exciting Cause having been the Shock of a Fatal Accident to a Child.—CASE 3. Nervous Symptoms of Four Years' Duration, the Result of the Patient's House having been struck by Lightning, removed by the Temporary Use of Chloral and Change of Air.—CASE 4. Nervous Symptoms the Result of intense Business Anxieties removed by the Temporary Use of Opium.—CASE 5. Nervous Disorder the Result of Overwork removed by the Temporary Use of Chloral.—CASE 6. Insomnia, with Hemi-hyperæsthesia removed by Four Small Doses of Chloral—The more common Sources of Mental Shock, Anxiety, and Nervous Disorder.

I WISH to direct attention to a very numerous and interesting class of cases, in which a great variety of so-called nervous symptoms are traceable to the influence of overwork, anxiety, or mental shock. For many years I have taken an especial interest in cases of this kind, and so long ago as the year 1853, in a course of lectures which I delivered at the Royal College of Physicians, I described their general features, and gave a considerable number of examples illustrative of their etiology, their symptoms, and their successful treatment by a cautious temporary use of opium at bedtime. Up to that time my experience of these cases had been gained chiefly as a dispensary physician and in the out-patient rooms of the hospital. Since then I have met with them in all classes of society, and I know of few forms of disease in which a treatment based upon the recognition of the true nature and origin of the symptoms

[1] *Lancet*, July 1875.

is, in a large proportion of cases, attended with more entirely satisfactory results. My treatment of these cases is essentially the same now as it was then, but the substitution of chloral for opium as a temporary soporific is a great practical gain. I say emphatically, as a 'temporary soporific;' for I shall hereafter explain that I resort to the chloral only to tide over a temporary difficulty. Few drug evils can be greater than that involved in the habitual employment of soporifics of any kind.

I shall endeavour to convey an accurate idea of the diseases in which I now desire to excite interest, partly by a brief history of a few typical cases, and partly by a general description of their leading and characteristic features. In order to give greater variety and completeness to my illustration of these maladies I shall select some cases from those which I observed in my hospital practice many years ago, and others from the more recent records of private practice.

I beg it to be understood that in using the terms 'mind' and 'mental,' as I shall frequently have to do in the course of these lectures, I mean by these expressions no more than is implied in their use by scientific psychologists. We know nothing of mind as a separate entity—apart, that is, from cerebral organisation and function. Mind is an abstract conception, like heat or motion, and it is conveniently used to designate a set of complex psychological energies. It comprehends volition, reason, affection, &c.—phenomena which are inseparably associated, so far as we know, with a material organisation. As physiologists, in our study of mental phenomena we make no reference to theological doctrines or metaphysical theories. We have no sympathy either with those who charge physiologists with materialism or with those who contemptuously speak of theologians as superstitious. With this preliminary explanation I will give my first illustrative case.

CASE 1.—Mrs. L——, aged 65, the wife of a farmer, consulted me on January 8, 1872. She had been ill and under medical treatment without benefit for a year. She was a woman of great energy and spirit, but since her illness she had been very weak and nervous, and unable to attend to her

household affairs. Her appetite had gone, and her sleep was constantly disturbed by distressing dreams, during which she often fancied that she was falling down a steep precipice or into deep water. After such a night she got up feeling more tired than when she went to bed. She perspired profusely during the night. Of late there had been some œdema of the legs, apparently from debility. There was no evidence of renal, cardiac, or pulmonary disease.

I now inquired for the cause of this nervous disorder, and it was not difficult to find. She had been in perfect health up to a year before, when one day, as she was driving with her husband in a dog-cart, the horse ran away. Her husband was first thrown out, and after a gallop for some distance along the bank of the Thames, at the risk of being pitched into the river, she was thrown out, and her arm was hurt, but not very seriously. The arm soon got well, but the effect of the mental shock continued.

Having heard this history, I said to her, 'If I can make you sleep quietly for a few nights I shall cure you quickly and completely.' I then prescribed twenty grains of chloral hydrate to be taken every night, and a mixture, containing quinine, tincture of nux vomica, and hydrochloric acid, to be taken three times a day after food. I saw her again on January 16. She had taken the chloral for seven nights. She had slept well without dreaming, she had therefore got up refreshed, her appetite and her spirits had returned, and she declared herself quite well. Her sister, who came with her, said to me, 'Her recovery is quite a miracle.' I replied that it was a simple and natural result of a few nights of quiet sleep and freedom from the harassing terror of the distressing dreams which had so long troubled her. I advised her to take a half-dose of chloral for a few nights longer. Her sister called with another patient on February 26, and reported that Mrs. L—— had continued her medicine for a week after her second visit, that her appetite, her spirits, and her sleep were good, and that she was quite well. I have several times heard since from the friends and relatives of this lady that she has continued in good health.

This case is a good example of a numerous class in which

the effect of a sudden, violent mental shock is continued and intensified by nightly recurring dreams, during which the brain is worried and exhausted, and the patient gets up feeling more tired than on going to bed. This condition of nervous system, if not removed by remedies, is often continued for an indefinite time until some more serious disorder results.

The following case is an illustration of the terrible consequences which may result if the state of dreaming restlessness, which was so speedily removed in the previous case, is permitted to continue unchecked.

CASE 2.—A. S——, a warehouseman, aged 29, came to me at the hospital in July, 1851, and said that he was subject to epileptic fits. His intellect was impaired, and he could give but an imperfect account of himself, so I desired him at his next visit to bring his wife with him. She was a very intelligent woman, and from her I obtained the following remarkable history. Her husband had always been a sober and steady man, and he never had any serious illness until he became subject to fits, the origin of which she thus accounts for. Seven years ago he was returning with his wife and some friends from Epsom races, when a child in the road accidentally fell under the wheel of his chaise, and was killed. The man was much horrified at the sight of the child's crushed head. That night he started up in his sleep, and called out, 'Oh! save the child.' He continued thus to call out in his sleep almost every night, and sometimes two or three times in the course of the night. When he started up in this manner he often screamed so as to awaken and terrify his children. At the same time he struggled and clutched with his hands; but when his wife aroused him (which she sometimes had a difficulty in doing) he usually appeared to be quite unconscious that he had been dreaming or making a noise. At other times he complained of having frightful dreams, and, putting his hand to his head, would say that he felt as if he were going out of his mind. Sometimes he exclaimed, 'That child haunts me night and day;' at other times, 'There is that child again;' and often, when in the street he has seen a child near a horse, he has been in great

terror, and his hands have been convulsively clutched. The state of things which I have described continued unchanged for about two years; then he was seized one morning soon after getting up with a sudden and violent fit of convulsions. About two or three weeks after he had two similar fits while in bed, and in about six or seven weeks after this he had another attack. The fits, which were evidently epileptic, frequently recurred up to the date of this history being taken. He has had three or four, and on one occasion, five fits in a day. The longest interval between the fits has been eight or nine weeks. One of the most remarkable features in the history of this poor man, is the fact that he had continued to be haunted in his dreams almost every night by the same horrible vision of the child, exciting the same startings up with screams and exclamations. This had occurred, as his wife assured me, so lately as the night before these notes were taken, seven years after the fatal accident. After his epileptic attacks he had sometimes been maniacal, and more than once he had attempted suicide. He had a well-formed head, but his expression was heavy and fatuous, and his memory and his mental faculties generally were much impaired. This sad case had gone beyond the reach of remedies; but what a different history might not this have been if at any time during the two years before the occurrence of the epileptic attacks he had taken for a few nights in succession some soporific pill or draught, which might have broken the habit of dreaming, and thus restored rest to his distracted brain! The history of this case affords a good illustration of the tendency which the habit of dreaming restlessness, when once established, has to perpetuate itself, and to induce other serious consequences.

Case 3.—The following case, in which distressing nervous symptoms of four years' duration, were removed by the temporary use of chloral, is one amongst the most recent that have come under my observation.

The Rev. J. D——, æt. 29, a country clergyman, consulted me first on May 22, 1886. Four years ago he received a severe nervous shock in consequence of the lightning having struck his house and nearly destroyed the roof. Since then his sleep had been habitually disturbed by distressing

dreams, during which he fancies that someone is trying to get the better of him. In his dreams he often calls out, clutches his wife by the throat and attempts to drag her out of bed. He has sometimes walked about the room in his sleep. He has frequent fits of mental depression, and has much difficulty in doing his work. His legs are feeble, and he fears that he shall lose the use of them.

There was no discoverable organic disease. I encouraged him to hope for a cure, and I prescribed chloral hydrate with ammon. bromide, of each twenty grains, to be taken every night, and a drachm of Fellows' syrup hypophosp. comp. twice a day. On June 1, his wife wrote that 'he seems much better since taking the sleeping draught, and he managed to take his duty on Sunday better than he has done for several months.' At first I had some difficulty in persuading him to take the night dose regularly; he took it only occasionally, and when he omitted it his nights were bad. On July 26 he called on his way to Switzerland, where he intended to stay for two months. He was still very nervous, and he complained that the draught made him drowsy during the day. I advised him to persevere with a twenty-grain dose of chloral without the bromide. He called again on his way home on September 24. His wife reported that on his way out he did not take the chloral, and that he was very restless and excited, but on his arrival at Arolla, which is 6,500 feet above the sea, he resumed the chloral and took it regularly for three weeks. During this time he slept well, and since then has continued to sleep well without the draught. He considers himself quite well, and says, 'I feel a different man.'

On December 27 his wife wrote to me that, in spite of hard work during Advent, 'he is really wonderfully better in every respect. His voice is so much stronger the people are quite surprised at the change in him.' I have no doubt that the rest in high mountain air materially aided his recovery; but I believe that without the temporary help of the chloral he would not have got free from his nocturnal disturbance and excitement, and the ultimate result might have been as disastrous as that of Case 2.

I will next give some particulars of a case in which

distressing symptoms of very long standing were completely removed by the temporary use of opium at bedtime.

CASE 4.—R. M——, a tailor, was 32 years of age when he came under my observation, in October, 1850. He had been a delicate child, but between the ages of 9 and 23 he had been healthy and strong. At the latter period his father, who had a good business, absconded, taking his money with him and leaving his family destitute; and the son now had to provide for thirteen persons besides himself, including his own wife and one child. From this time he was fearfully harassed, and suffered the usual horrors of intense mental anxiety, restless nights, fearful dreams, spectral illusions, and confusion of thought to a degree which had often rendered it impossible for him to attend to business. He had often been tempted to commit suicide, and has been able to resist the temptation only, as he says, by the force of religious principle. At one time, for a period of two or three years, he was so nervous as to be incapable of taking care of himself, and he had a man to watch him. His habits have always been strictly temperate. This history was obtained partly from himself and partly from his wife, who is a very intelligent and apparently truthful woman.

When I first saw him he was tolerably well nourished, but his face was pale; his expression was torpid rather than anxious; his sleep was disturbed; he complained of great confusion of thought and of 'losing himself;' he had a slight cough and a great dread of consumption. I did what I could to encourage him. I assured him of his freedom from bodily disease, and of my hopes that he might recover. I advised him to take a walk daily in the open air; and on October 28 I prescribed compound soap pill, five grains every night; and tincture of perchloride of iron, twenty minims, with infusion of quassia, one ounce, three times a day.

On November 11 the report is that he sleeps soundly, and his appetite has been much better since taking the medicine.

November 18.—He is looking much better and brighter; he now feels that he can do his work, and he is confident that he will get well. He has had no confusion of thought the last few days. *Repet.*

January 13.—He has continued to improve since the last report, and considers himself quite well; his countenance is natural, cheerful, and intelligent; he sleeps well and has neither dreams nor fears; 'life is now a pleasure to him instead of being a constant dread.' He has no work at present, but he feels quite fit for working. He has continued to take the mixture regularly, but the night pills only occasionally.

March 27.—Two days after my last report he undertook the management of an extensive tailor's business, which he conducts with ease and comfort to himself, and to the entire satisfaction of his employers. He declares that he never was better in his life, and his wife thinks the same. He looks well and strong. Since the last report he has taken no medicine except an aperient pill occasionally.

In no other case that has come under my observation have symptoms so serious and of such long duration disappeared so speedily and completely as they did in this instance. The improvement, too, was permanent. I saw the man two years afterwards, in March, 1853, when he told me that he was in the same situation, and although the hours were long and the work heavy, he had not been one day absent on account of illness. He slept and ate well, and his countenance was cheerful and intelligent.

I will next give a brief report of a case in which mere overwork of brain was the exciting cause of nervous symptoms.

CASE 5.—Mr. H. C——, aged 23, articled clerk to a solicitor, consulted me on March 7, 1872. For several weeks he had suffered from headache, confusion of thought, and inability to work. His appetite was bad, and he complained of uneasiness after taking food. He slept badly, and was much distressed by dreams. The cause of the symptoms in this case was overwork of brain. He had been reading most injudiciously, sometimes from eight o'clock in the morning until two the following morning. When he came to me he had been away for a month's holiday, during which he abstained from all mental application, but he was no better for the change. He had continued to sleep badly, and he was therefore unrested. I prescribed twenty grains of chloral

hydrate to be taken every night. He was on his way home when I saw him. He wrote to me on March 16 that 'for the first four nights he had the most profound sleep, without any dreams; since then he had not quite such unbroken sleep, but he was thankful to say that he was wonderfully better.' I advised him, in reply, if the dreams continued, to take thirty grains of chloral for a few nights, and then gradually to reduce the quantity and leave it off. I heard from him again on March 26 that his sleep was now very good, the pain in his head had ceased, and he intended to return to work that day week.

I wrote afterwards to this gentleman asking for a report of his progress since I last heard of him. He stated that he remained, with quiet work, in pretty good health for some five months, when he had a slight renewal of his old complaint. He took a few doses of chloral and a little rest from work, and then went to Switzerland, where he did some good mountaineering. He returned, read up for and passed his examination, and has stuck well to work ever since, feeling very little of his 'old enemy' except 'after sticking too closely to work.' He goes on to say that he finds plenty of exercise and early to bed the great preventive, but he thinks 'the chloral an invaluable remedy.' He adds that 'after taking the chloral for some time he found no difficulty in leaving it off.'

In the next case excessive brain-work with anxiety caused painful symptoms, which quickly yielded to treatment.

Case 6.—I was consulted some time since by my friend Dr. ——, 35 years of age, who complained of a painful hyperæsthesia over the whole left side of his body; so sensitive was the skin that even the touch of his shirt gave him pain. He was apparently in robust bodily health, but his nights were disturbed by dreams, and he got up unrefreshed. I knew that he was overworked, and that he had much cause for anxiety. I advised him to take a small dose of chloral at bedtime for a few nights, and I said, 'A few nights of quiet sleep will rest your brain and remove your nervous symptoms.' A fortnight afterwards I received from him the following note:—'Here is an interesting clinical fact. I suffered from hyperæsthesia of exactly one-half of the body—the left side—for nearly a

month. Even Brighton failed to cure it. After ten grains of chloral I was much relieved, and after four doses of ten grains on alternate nights *it has entirely disappeared.* My brain, which has for months felt queer, has got a new lease of activity; I feel ridiculously well, and am amazed at the change. I see now that my ailments have all been due to want of sleep.' My distinguished friend has had no return of his unpleasant symptoms; he is now free from anxiety, and his energetic brain is working as hard as ever.

The cases which I have thus briefly described will suffice to convey a general idea of the symptoms which result from overwork, mental strain, and nervous shock. I proceed now to point out some of the more common sources of nervous disorder, with a view to facilitate their recognition by those who are investigating the history of these diseases. It will be unnecessary to dwell upon those sources of mental anxiety whose influence is immediately and universally recognised, such as the illness or the death of relations and friends; or, what is often worse than illness or death, the misconduct of those in whom a deep interest is felt, reverses of fortune, failures and disappointments in business, &c. The operation of these causes is so obvious and so powerful as to be seen even by the least observant. We may commonly notice that an overworked man, whether his work be mechanical or mental, becomes an over-anxious, nervous man. The overtaxing of the strength is attended by a sense of fatigue and exhaustion, loss of appetite for food, and often by inability to sleep soundly; with this there comes an anxious dread of breaking down, and the combination of fatigue, anxiety, and unrefreshing sleep is a common cause of mental and bodily collapse. So long as a hard-worked man can obtain refreshing sleep he is safe, but danger begins when, his sleep being disturbed by distressing dreams, night brings no rest to his overwrought brain. And here it may be observed that habitual indolence and neglect of duty often occasion as much mental disquietude as excessive labour and over-fatigue. Those who 'kill time' are not unfrequently

> Haunted much by visions strange,
> And sore perplexity of roaming dreams,
> The spectres manifold of murdered hours.

The infringement of moral laws brings discomposure and anguish upon the mind as a physical injury gives bodily pain. Some of the most distressing cases of nervous disorder are those which result from the excessive indulgence of the baser animal passions. This is partly a result of the nervous exhaustion and disorder which too often follow upon excessive nervous excitement; partly it is a result of the remorse and self-reproach which torture the man who is conscious of degrading habits. It is scarcely necessary to remark that alcoholic excess will aggravate the effects of overwork, anxiety, and other disturbing influences.

We frequently meet with instances of severe and long-continued nervous derangement as a result of a momentary but violent mental shock. Cases of epilepsy in which the fits have been the result of fright are by no means uncommon. It is a well-known fact that some young and nervous subjects have been frightened into epilepsy by seeing another in a fit. Some time since I heard of two young brothers, one of whom became epileptic. They continued to sleep together in the same room, and the result was that the second soon became epileptic too.

The causes of sudden and violent mental shock are very numerous, and most of them are so obvious as to require no comment. One of my hospital patients (a woman) was excessively nervous long after receiving a fright from the collision of two steamboats, and a false alarm that the boat in which she was a passenger was sinking. I have had several patients who have suffered long and seriously from the nervous shock of a railway collision. One gentleman, who was much bruised and shaken in the collision at Harrow Station, in November, 1870, was excessively nervous, and had his sleep disturbed by frightful dreams for several months after he had recovered from the serious physical injuries which he sustained in that terrible crash.

The alarm of fire is a frequent cause of long-continued nervousness. Many years ago the Olympic Theatre was burnt down, and for two or three years after the fire I had several hospital patients, both men and women, who had lived near the theatre at the time of the accident, and who had con-

tinued to dream of fire, and to be excessively nervous and timid.

In directing attention to these various sources of nervous shock and mental anxiety, I have acted in the spirit of a suggestion which is contained in the following remarks by Dr. Latham:[1] 'Prior to diseases, to their diagnosis, their history, and their treatment, prior to them and beyond them, there lies a large field for medical observation. It is not enough to begin with their beginning. There are things earlier than their beginning which deserve to be known. The habits, the necessities, the misfortunes, the vices of men in society, contain materials for the inquiry, and for the statistical systematising study of physicians, fuller, far fuller of promise for the good of mankind than pathology itself.'

The kind of inquiry here referred to, and commended to our notice by Dr. Latham, is, in my opinion, an essential element of a truly scientific pathology, and with reference especially to diseases of the nervous system its paramount importance is indisputable. It would not be difficult to show that many serious errors which have prevailed with regard to both the pathology and the treatment of some of these affections are attributable to a disregard of that portion of the patient's history which precedes the occurrence of actual disease, and a study of which is requisite, as well for the discovery of the true cause as for directing the successful treatment of the malady.

SECTION II.

Symptoms and Results of Overwork, Mental Shock, and Anxiety—Insomnia—Distressing Dreams—Somnambulism—Sense of Exhaustion—Anxious Expression of Countenance—The 'Anxious Eye'—Loss of Appetite and Weight—Dyspeptic Disorders—Glycosuria—Palpitation, with a Dread of Heart Disease.

In the present section I propose to describe some of the consequences or symptoms which may result from overwork, anxiety, or mental shock and terror.

[1] *Clinical Lectures on Diseases of the Heart.*

The effects of mental anxiety vary greatly in different cases, the difference depending partly upon the original mental and bodily constitution of the patient, partly upon education, position in society, and habits of life, and very much, too, upon the kind of treatment to which the anxious patient is subjected. But amidst all the variety of circumstances to which I have alluded there are certain general features which characterise nearly every case of overwork and mental worry. These general characters I now proceed to describe.

Amongst the effects of anxiety of mind, whatever may have been the exciting cause, there is not one which is more frequent or more important than that which manifests itself by sleeplessness or by disturbed and therefore unrefreshing sleep. It is very interesting to note the various accounts which different sufferers give of their sleeping hours. One of the most frequent complaints is that the sleep is disturbed by distressing and frightful dreams, which assume a great variety of forms in different cases. The sleeper often fancies that he is falling down a precipice or into deep water, or that he is pursued by some fierce animal in brute or human form, or that he is in some situation of difficulty, perplexity, and danger from which he cannot escape; these are amongst the mildest forms of unpleasant dreaming. In other cases the dreamer has visions of dead relations and friends, or he is haunted by spectres of every form which a disordered imagination can conceive. No physiologist can doubt that in dreams such as these we have the explanation of most stories of ghosts and apparitions. Samuel Taylor Coleridge, who, as we know, at one period of his life was a large consumer of opium, when asked if he believed in ghosts, replied, 'No, I have seen too many of them myself.' The distress of the dreamer is often manifested to those who are watching his slumber by a pained or terrified expression of countenance, sometimes by profuse perspiration, with violent action of the heart, and laborious or hurried and gasping breathing; sometimes, too, by suppressed moans, or by frequent talking, and not uncommonly by a loud cry of terror. In some cases the dreamer starts out of bed and walks about the room, and he

may awake in doing this, or he may be aroused by his own cries; but in other instances, as in the case (No. 2) which I related in the previous section, it is very difficult to bring him back to a state of complete consciousness. On awaking, he often has a vivid recollection of all the circumstances of his dream, and he fears to go to sleep again lest in his sleep he should have a return of the dreadful thoughts and visions; but in other cases he is quite unconscious that he has been dreaming or talking or making a noise; he has only a vague sense of something terrible, and he finds his skin bathed in perspiration and his heart beating violently. This violent palpitation is sometimes a source of great alarm to the patient.

When a patient awakes after a night of disturbed sleep, such as I have described, he usually complains of exhaustion, and will often say that he is more tired in the morning than when he went to bed; a feeling which we can easily understand if we bear in mind that not only has he had no rest—a dreaming brain being an unresting brain—but he has been exposed to the disturbing and exhausting influence of terror during the greater part of the night. This sense of languor and unrest is in most cases combined with loss of appetite, inability for exertion, depression of spirits, a sensation of pain or weight in the head, and sometimes with an oppressive feeling of drowsiness during the daytime, particularly after meals.

When this restless condition has continued for some time it is almost invariably associated with a peculiar, anxious expression of countenance, and, in particular, with a remarkable appearance of the eyes, which will often enable one at once to detect the general character of the case with which one has to deal, and so will suggest such further inquiries as may be necessary for its complete elucidation. The appearance of the eyes to which I refer is difficult to describe, but it may be immediately and easily recognised when once it has been pointed out and clearly perceived. The anxious eye loses that brilliancy and liveliness upon which so much of its natural beauty depends, and it assumes something of the dull and inanimate appearance of a snake's eye. The iris often appears to be in a more or less fixed and inactive condition, so that the size of

the pupil varies but little and slowly under the influence of light or mental emotion. If you watch the bright eyes of an intelligent child under the influence of some pleasing emotion, you will see that there are frequent and rapid changes in the size of the pupil. The dull and anxious eyes of the overworked and harassed man or woman are in striking contrast with the brilliant eyes of the happy child. In cases of long standing the pupil is often very small; and, in some of the worst cases, I have seen the pupil contracted almost to the size of a pin's point. It is seldom that even in these extreme cases there is any intolerance of light; but there is often, in a greater or less degree, dimness or defect of sight, without any appearance of structural change in the eye. I believe, however, from what I have seen, that atrophy of the optic nerve and retina is one of the more serious consequences of long-continued overwork of brain. Quite recently I have seen a very intellectual girl, aged 19, who, as a result of excessive mental application, in addition to other signs of nervous disorder, suffered from severe *pain* in the eyes, which prevented her from reading. An eminent oculist had examined her eyes, and pronounced them to be free from all appearance of structural change.

I have already intimated that the anxious expression of the patient's face will often give a clue to the general character of the case, and serve as a guide to the true source of the symptoms. I may add, too, that the information which we gain by observing the patient's physiognomy will sometimes induce us to persevere with our inquiry when the answers to our first questions are not such as we expected to receive. For instance, I have often been told by patients, who have presented the anxious expression of face which I have described, that their sleep is not only not bad, but indeed they 'sleep too well.' Now in these cases it will often be found, on inquiry, that although they sleep so heavily that they can with difficulty be aroused in the morning, yet their sleep is constantly disturbed by frightful dreams; consequently they awake unrefreshed, and they are languid and drowsy during the day. The cure for this too heavy but unrefreshing sleep is a dose of chloral or some other soporific at bedtime, repeated for a few nights until the habit of dreaming is broken; the result will

often be refreshing sleep at night, and, as a consequence of this, the cessation of the languor and drowsiness which had before continued during the day, after a night of harassing dreams.

I have sometimes been told, in answer to the question whether a patient has been disturbed by dreams, that he does not get sleep enough to render dreaming possible. He declares that he lies awake all night. I believe, however, that a patient is very rarely so wakeful as this statement would imply; and that often when he supposes that he has not once closed his eyes he has really slept, but his slumbers have been so frequently broken and disturbed by dreams that he can scarcely distinguish between his sleeping and his waking hours. I scarcely need say that sleep of this imperfect character is almost as unrefreshing as complete wakefulness.

One of the consequences of mental anxiety, which is as often as any other associated with disturbed sleep, is a more or less complete loss of the appetite for food. In only a very small proportion of the many cases that I have seen have I been told by an overworked, anxious, dreaming patient that he could take his food as usual. In many instances the digestive powers are much impaired; there is pain or a sense of weight or fulness, with flatulence, after eating. In some cases severe stomach pain, with water-brash and occasional vomiting, are complained of; in others diarrhœa is a troublesome symptom.

It is obvious that the combined influence of loss of sound refreshing sleep and loss of appetite, with imperfect digestion, must tend to impair the nutrition and lessen the strength. Loss of flesh and strength to a greater or less extent are observed in almost every case when the symptoms have been of long duration. One man who had suffered much from restlessness and frightful dreams, in consequence of business anxieties, assured me that during a period of two years his weight had been reduced from 15 st. to 11 st.; and this statement appeared quite consistent with the loose fit of his skin and his clothes. Another man, who had suffered in a similar way, in consequence of misfortunes, had lost, as he informed me, from 2 st. to 3 st. weight in two years. This was one

of the cases in which, after a long period of nearly total inability to eat or to digest food, a voracious appetite became almost a matter of complaint after refreshing sleep had been obtained by the temporary use of opium.

The derangement of digestion, to which I have referred as one amongst the most common results of mental anxiety, not unfrequently gives rise to symptoms referable to the urinary organs—symptoms which often excite an unfounded dread of serious organic disease. Frequent micturition and pain in the back are the symptoms usually complained of, and these are often supposed by an anxious patient to depend on disease of the kidneys or bladder, more especially when the urine is turbid and deposits a copious sediment. On examination it is often found that the urine is excessively acid and deposits lithates on cooling. The acid urine irritates the kidneys and bladder, and thus occasions the pain and the frequent micturition. In other cases the urine is neutral or alkaline, with a copious phosphatic sediment. These states of urine are, of course, not peculiar to nervous and anxious patients; but since any pain or any unusual appearance of the urine is a source of alarm to these timid subjects, we must be prepared to give a correct explanation of the symptoms and to apply the appropriate remedies.

In some instances the pain in the back is very severe, and, being much increased by motion, its seat is probably in the lumbar muscles; but I have met with several cases of this kind which have been mistaken for inflammation of the kidneys and treated as such.

I have seen several cases in which saccharine urine and other symptoms of diabetes have apparently resulted from excessive brain-work and mental worry. This would seem to be analogous to the result of Bernard's well-known experiment on rabbits, in which the secretion of saccharine urine was directly caused by mechanical irritation of the medulla oblongata.

There is, probably, no form of functional disturbance which is a more frequent source of anxiety and alarm to a nervous patient than the palpitation of the heart which is so often associated with mental anxiety and restlessness. I have already referred to palpitation in connection with the frightful

dreams by which the patient is harassed at night. A similar disturbance of the heart's action is scarcely less distressing in the daytime. The cause of this is probably in part purely physical and in part mental. The nutrition of the heart suffers, in common with that of the rest of the body, in consequence of broken sleep and the associated disorder of the digestive organs. The tendency to palpitation thence resulting is similar to that which exists in cases of anæmia and chlorosis. The powerful influence of mental emotion upon the heart's action is so well known that it is unnecessary to insist upon it. It will commonly be found that the disturbance of the heart's action bears a relation, first, to the weakness and consequent irritability of the organ itself, and, secondly, to the degree in which the patient's attention is anxiously directed towards the heart. The two main objects of treatment, therefore, are to strengthen the muscular tissue of the heart by sleep, food, and tonics; and to divert the patient's attention from his heart by the assurance that there is nothing in the state of that organ about which he need be anxious. The effect of a favourable opinion and a hopeful prognosis is often very beneficial by calming the patient's fears and restoring his confidence; while, on the other hand, an erroneous diagnosis or an unguarded expression with reference to heart-disease may be a source of great additional suffering to the patient, and may greatly retard or entirely prevent his recovery. A poor woman whose natural irritability and nervousness had been increased by great domestic troubles, and who, in consequence, suffered much from palpitation, was told by her medical attendant that she had a diseased heart, and an issue was established over the supposed seat of disease. She had endured this addition to her sufferings for several weeks when she came to the hospital. The impulse of the heart was sharp but feeble, and the sounds were quite normal; there was evidence of a weak and irritable heart, and of nothing more; but it was long before she lost the dread of sudden death, which had taken complete possession of her mind, and which had doubtless been confirmed by the erroneous diagnosis and the injudicious treatment to which I have referred.

It is but seldom that an irregular and excited action of

the heart is unassociated with some degree of difficult breathing, and when this is attended with cough an anxious patient will often be tormented by a dread of consumption.

I make frequent allusion to the fears of this class of patients because they constitute so very important a feature of their history, and because it should be one of the chief objects of the physician to convince the anxious man that his fears are groundless, and that the disease which he so much dreads has no existence except in his own terrified imagination. If, after a careful examination of the chest, we can assure him that his lungs and heart are sound, we shall remove a weight of anxiety from his mind which will greatly contribute to the recovery of equanimity and bodily strength.

SECTION III.

Some of the More Formidable Disorders of the Nervous System the Result of Mental and Emotional Influences—Epilepsy.—CASE 7. Delirium and at length Epilepsy the Result of Anxiety caused by a Drunken Wife—Delirium from Fright—Temptation to Suicide.—CASE 8. Mania and Epilepsy the Result of Suppressed Pecuniary Anxieties.—CASE 9. Mania and Fatal Epilepsy the Result of Anxiety caused by an Unhappy Marriage.—CASE 10. Chorea the Result of Fright.—CASE 11. Hysterical Gloom from Overwork and Anxiety.—CASE 12. Distressing Choking Attacks removed by Amputating an Elongated Uvula.

I described in the last section many of the symptoms and signs of functional disturbance in various organs which may have their origin in mental and emotional influences. I now wish to point out that some of the most formidable disorders of the nervous system may result from the same causes. A study of the more serious consequences of excessive brain work and of mental anxiety and shock will serve to show the practical importance of an exact diagnosis and appropriate treatment in the early stages of these disorders of the nervous system.

There are few diseases more terrible than epilepsy, and this dreadful malady is one of those which are often traceable to causes such as we are now considering. Many years ago, when I was physician to the Public Dispensary in Carey

Street, and afterwards when seeing out-patients as assistant physician of the hospital, I obtained the histories of a large number of cases of nervous disease, and I will now give, as briefly as possible, the results of that inquiry, so far as relates to the exciting causes of epilepsy. On analysing the histories of 37 cases of epilepsy, I found that no fewer than 28 were probably the result of what are commonly called and understood as mental influences. In 5 the fits were the result of some obvious bodily disease or of intemperance, and in 4 no probable cause could be assigned. The cases of epilepsy to which I have referred as having their origin in influences acting primarily on the mind naturally divide themselves into two classes, distinct, though closely allied. 1. The first class includes those cases in which epilepsy has resulted from great terror or excessive and sudden grief, and in which the first fit comes on almost immediately after exposure to the exciting cause without being preceded by warning symptoms. 2. In the second class are included those cases of epilepsy which arise from excessive mental work or continuous grief and anxiety, and which are preceded for a variable period by warning symptoms. Out of the 28 cases to which I have referred, 11 belonged to the former class and 17 to the latter.

Amongst the exciting causes which have acted suddenly in producing epilepsy, I may mention the sight of a supposed ghost which had been dressed up in order to terrify. In one case the ghost was a boy covered by a sheepskin. One girl attributed her fits to the fright of hearing a knocking, which she believed to be supernatural. A woman became epileptic on hearing of the sudden death of a friend; she felt, as she said, 'a sudden turn and agitation,' and within an hour she had a convulsive fit. A man walking along the street saw a workman killed by falling headlong from a scaffold close to his feet. Instantly the horrified passenger fell in an epileptic fit, and he had been epileptic for years when I saw him and obtained his history. A boy was playing in the street, when a policeman terrified him by threatening to take him to the station for making a noise. He ran a few yards, and fell in a fit at the door of his home. The fits recurred, he became paralytic, and died two years afterwards.

When epilepsy results from a sudden mental shock, the first fit usually occurs immediately or within a few hours; but in some cases an interval of days, weeks, and even months, may intervene, the patient meanwhile manifesting signs of nervous disorder. The most remarkable example of a mental shock renewed and aggravated by nightly recurring dreams, and at length, after an interval of two years, culminating in epilepsy, is afforded by the second case, whose history I gave at p. 201 of this chapter. From what I have seen of analogous cases, I believe that the habit of dreaming might have been checked and the chain of morbid phenomena broken by a judicious use of soporifics at any time during the two years before the occurrence of the epileptic convulsions.

Those cases of epilepsy which result from excessive brain work, from anxiety or continued grief, are usually preceded by a train of nervous symptoms; and it is during this period of what we may call the incubation of the disease that preventive measures may be resorted to with a reasonable hope of success. The symptoms which precede the full development of epilepsy are similar to those which I described in my last lecture as resulting from mental shock and anxiety: terrific dreams and visions, starting, struggling, talking, moaning, and sometimes screaming during sleep; a sense of fatigue in the morning, the patient often declaring that he is more tired when he gets up than when he goes to bed; often there is a sense of pain, weight, or constriction in the head, loss of appetite, disorder of digestion, and general weakness. These symptoms continue for a variable period, sometimes for many months, but in other cases only for a few weeks or even days, before the occurrence of the first epileptic fit, which generally takes place in the night and during sleep. The nocturnal disturbances then continue, sometimes in an aggravated form; and after an interval, which varies from a few hours to several months, a second fit of epilepsy occurs, and so by degrees the disease is established.

Now, let me here guard against a possible misconception. I do not maintain or believe that every overworked or anxious man whose sleep is disturbed by terrifying dreams, or who

talks, screams, or starts up in his sleep, will necessarily become epileptic or insane. Such an opinion would not only be very unreasonable, but, expressed in the hearing of one of these nervous patients, it might contribute much to bring about its own verification. The symptoms which I have described indicate, as the doctor says of Lady Macbeth, 'a great perturbation in nature,' which if neglected, may lead on to epilepsy, or an outburst of delirium or mania. Obviously, then, they demand the most earnest attention, with a view to ascertain their cause, and then to find a remedy.

The following case presents some interesting and instructive features :—

CASE 7.—In March, 1851, I was called to see a merchant's clerk (J. R. S——), aged 28. I was told that he had been delirious for three days. I found that his head had been shaved, his temples leeched, and his neck blistered, and he had been kept on a rigidly low diet. The result of this treatment had been an increase in the violence of the paroxysmal delirium, but at the time of my visit he was quiet and answered questions rationally. His skin was cool and moist, his pulse moderately quick, and his pupils natural. I ascertained, not only from his family, but also from his employers, that he was a very steady and temperate man. His wife told me that for several weeks before his illness his nights had been very disturbed ; that in his sleep he had talked almost incessantly of his business ; that in the morning he appeared unrefreshed ; that his appetite had been bad ; and that he had been gradually losing strength. When I inquired for the cause of all this disturbance, I was told that the fatiguing and harassing nature of his employment was the only cause for anxiety of which his family were aware. It seemed evident that the case should be treated as one of delirium from exhaustion ; and accordingly I prescribed a mutton chop with a glass of porter, and at bedtime forty drops of laudanum. The next day I found that he had slept well and there had been no return of the delirium. He rapidly recovered, and at the end of a week he returned to his employment. About three weeks after he had returned to his work I was asked to see him at his employer's warehouse, where I found that he had

suddenly fallen in an epileptic fit whilst engaged in washing his hands. I again inquired particularly for sources of anxiety, and I was again told by his family that overwork was his only trouble; but his employers, who were friends of my own, assured me that there was nothing in his work to trouble a healthy man. The fits recurred again and again. His wife told me, in the month of October, that his nights were constantly disturbed by dreams, and that he talked, moaned, and frequently called out in his sleep. He became a confirmed epileptic, was soon compelled to give up his situation, and about two years afterwards he died. It was not until the month of February, 1852, nearly a year after I first saw the patient, that I learnt the real cause of his nervous disorder. His father then told me that my patient's wife was a confirmed drunkard, and that when drunk she was extremely violent; that she had more than once threatened to stab her husband, and that on one occasion she had actually wounded his hand with a knife. My informant had from the first been aware that this was the cause of his son's misery and restlessness; but he was unwilling to speak of it, even to me, until matters had become desperate. It is evident that the information thus tardily given is the central fact in the history of the case, and until its sad domestic history was revealed there appeared no satisfactory explanation of the poor man's continued and increasing nervous disorder.

I have seen other cases similar to this within a comparatively recent period in private practice, but for obvious reasons it would be inexpedient to publish them.

The delirium which occurred in the early period of this case is a good example of the kind of delirium which is sometimes brought on by intense mental anxiety in persons of strictly temperate habits. Great and sudden terror may be followed almost immediately by maniacal delirium. Quite recently we have had in the hospital a boy aged 15, who became violently delirious immediately after being terrified and assaulted by two men as he was returning from his work in the neighbourhood of London. But the delirium which results from overwork and anxiety rarely if ever occurs without being preceded for a variable period by disturbed sleep

and the other signs of nervous disorder which I have before described. Sleep-talking and somnambulism are amongst the most frequent precursors of delirium. When an anxious patient is talking in his sleep he will sometimes answer questions as if all his faculties were alive, and yet, when he awakes, he may have no recollection of what he has said or heard. In other cases he appears, while sleeping, to be carrying on a conversation with someone whom he imagines to be present. I was once told by a very intelligent woman that her husband, who ultimately became insane, was of what she called 'a very close disposition,' so that he would never willingly speak of his troubles to her or to anyone; but whenever he had been unusually worried, he talked so much in his sleep that she heard from him then, not only that he was anxious, but also the cause of his anxiety. 'Infected minds to their deaf pillows will discharge their secrets.' The transition from a state of delirious sleep to delirium in the waking condition may be direct and rapid, but there sometimes occurs a transient intermediate stage, during which a man starts up in a state of confusion and terror, looking about him distractedly, and perhaps talking wildly, and recovering complete consciousness and self-possession only after being frequently spoken to and roused by being touched or roughly shaken.

In some instances the excitement on first awaking is accompanied by a temptation to commit suicide, or to do violence to others; and the patient will afterwards speak of these feelings as most distressing and terrible. In other cases he begs that he may not be left alone lest he should destroy himself, or that those whom he most loves should remain away lest he should injure them. These symptoms, if permitted to continue unchecked by judicious treatment, may be quickly followed by an outburst of maniacal delirium, or by an epileptic seizure, or sometimes, as I have seen in several instances, by both these terrible consequences in succession. In no case whose history I have investigated has an outbreak of delirium of this kind occurred without being preceded for a longer or shorter period by some of the premonitory symptoms which I have described. I have heard and read of men becoming suddenly delirious and mad without warning

or any discoverable cause. I have never seen such a case, but I have met with more than one which, without a careful examination, might have passed for such—cases in which there was a deep and abiding cause for anxiety which was not revealed, even to the nearest relative or friend, until it was discovered, after it had wrought irremediable mischief on the nervous system of the patient.

Case 8.—One such case was that of a clergyman whom I had known for some years as a man of powerful mind and body, until, to the surprise and grief of his family and friends, he suddenly, as it appeared, became insane, and subsequently epileptic, when he was just over thirty years of age. His habits were strictly temperate, but he was disposed to be over-anxious about his parochial work. His wife observed that his appearance and manner were changing; he became unusually reserved and sometimes irritable. He was at a distance from all other members of his family, and his anxious wife had no suspicion either of the real cause of the change which had come over him or of the serious consequences which were threatened. At length he became suddenly and furiously delirious in the middle of the night. The mental disease continued and became complicated with epilepsy; and after nearly four years of suffering he died. Now the cause of this terrible calamity was not discovered until the time for prevention had passed. His income was limited, and insufficient for the wants of an increasing family; he was in pecuniary difficulties of which no one but himself knew until the state of his affairs was disclosed after his illness. He was naturally reserved, sensitive, and proud; and he had concealed his difficulties from his wife, probably from an unwillingness to distress her with a knowledge of them. Of course his own anxiety was intensified by this reserve, and by his not seeking that relief which might have been obtained if he had imparted his troubles to his family and friends.

Case 9.—In September 18—, I was consulted by an officer, aged 46, in a high position in the army and in society, on account of an attack of loss of consciousness, which had occurred while he was in bed three days before I saw him. I inferred from his description that he had had an epileptic

attack. I observed that he had what I have described as the 'anxious eye' (p. 211); but, in reply to my particular inquiries, he assured me that he was not overworked, and that he had nothing to trouble him. I saw him again in a few days, when I repeated my inquiries and received the same answer. In November his sister called to tell me that he was in the country acting very strangely, and evidently insane. I inquired for sources of mental anxiety, and was told that it had recently come to the knowledge of his family that some years since he had formed a *liaison* with a lady, by whom he had had several children, and whom he had recently married. For reasons which need not be mentioned here, this affair had for some time caused him the most intense anxiety, and was without doubt the cause of his mental derangement. He came up to town in a state of maniacal excitement. I had a consultation with Dr. Monro and with the surgeon of the regiment. He was sent to an asylum, where he was generally happy in the belief that he possessed boundless wealth. He had occasional epileptic fits, and after some months he had a rapid succession of epileptic attacks and died.

I could give the details of a considerable number of cases of whose history I have an intimate knowledge, in which consequences equally disastrous, though differing in different cases, have resulted from similar causes, and especially from the influence of suppressed grief, care, or anxiety, the cause of which has been neither known nor suspected by the patient's nearest friends, until it has been brought to light after the onset of some alarming illness—an attack of mania, or epilepsy, or paralysis.

Amongst the nervous diseases which are frequently the result of mental shock, *chorea* or *St. Vitus's dance* is well known to be thus excited in a very large proportion of cases. One of the most interesting problems in pathology is to explain the fact that chorea may result from such apparently different causes as the mental influence of terror and the mechanical plugging of minute cerebral vessels by fibrinous particles detached from an inflamed endocardium. I do not propose to discuss that question now, neither shall I attempt to account for the fact that a similar mental shock will excite

in one patient delirium, in another epilepsy, and in a third chorea; but I wish to insist upon one fact of practical importance with reference to the preventive treatment, which is this: that as in cases of delirium and of epilepsy excited by mental shock, so in cases of chorea, the symptoms of the special nervous disorder may not commence until some days after exposure to the exciting cause; but on careful inquiry it will generally be found that in the interval the patient's sleep is broken and disturbed by dreams, as a result of which the effect of the original mental shock is perpetuated and intensified until the nervous disorder reaches its full development.

CASE 10.—In December, 1874, I had in the hospital a boy 15 years of age, in whom a severe attack of chorea was excited by fright. As he was walking in the dark a large black dog jumped over a fence by the side of the road, and came so close to him as to knock a basket out of his hand. He was very much frightened, but a week elapsed before the symptoms of chorea appeared. In the meantime, however, his sleep had been disturbed every night by terrifying dreams, which made him more and more nervous. The symptoms after his admission rapidly yielded to the soothing influence of ten grains of chloral hydrate taken three times a day.

Now, the practical lesson which I wish to convey is this, that in all probability if he had taken a dose of chloral at bedtime for three or four nights after his fright he would have slept without dreaming, his brain would have been soothed, and the mental shock therefore would not have developed into an attack of chorea.

The nervous symptoms which result from mental shock and emotional excitement sometimes assume the form of *hysteria*.[1]

I have no doubt that in some cases the nervous sensation of globus is excited by some local irritation of the throat. When a nervous woman, or even a very nervous man, gets a common sore-throat, the distress and alarming sense of impending suffocation are often quite disproportionate to the physical changes in the throat which we can see with the laryngoscope.

CASE 11.—A poor woman who was very anxious and much

[1] See the chapter on Hysteria.

overworked had a slight choking fit while she was eating, and afterwards she felt sure that some substance was still lodging in her throat. She went to one of the hospitals, where the house-surgeon passed a probang, but that rather increased her distress. When she came to me I found the mucous membrane of her fauces and pharynx congested, but her intensely anxious face and nervous manner, and her description of the 'rising' in her throat, were the most significant facts. It was not until after many days, and then very gradually and with occasional relapses, that she escaped from the conviction that some solid mass was sticking in her throat and threatening suffocation.

CASE 12.—I was once consulted by an unmarried lady, about 45 years of age, who, for several months, had been disturbed nearly every night by attacks of choking, which compelled her to sit up in bed. She had a pallid and delicate, but not an anxious look. On examining her throat I found that she had a very long and slender uvula, which was always in contact with the back of her tongue. I cut off about two-thirds of the uvula, and from that time she had no more attacks of suffocation. In this case I have no doubt that the distressing attacks of suffocation were excited during sleep by the elongated uvula.

SECTION IV.

Principles of Treatment—Ascertain and remove the Cause of the Symptoms.—CASE 13. Insomnia and Nervous Disorders of Many Months' Duration removed by a few doses of Opium—Chloral a More Valuable Soporific than Opium when there is no Pain—It sometimes flushes the Face—Precautions to be observed—Cases Most and Least Amenable to Treatment.—CASE 14. An Intemperate Man assisted to abstain by the Temporary Use of Opium.—The Careful Use of Alcoholic Stimulants sometimes Beneficial.—CASE 15. Maniacal Excitement successfully treated by the Wet Pack after Chloral had failed.

In previous sections I have had frequent occasion to refer to principles of treatment, and to the influence of particular remedies on the various forms of nervous disorder which I

have brought under notice, and I purpose to devote the present lecture to a more detailed consideration of the important subject of treatment.

The observations which I am about to offer upon the subject will have reference chiefly to the earlier stages and the less serious forms of nervous disorder—sleeplessness or disturbed sleep, with its usual attendants and consequences, lassitude, loss of appetite, palpitation, and the other symptoms which were described in the second section of this chapter.

The first point obviously is to ascertain the cause of the symptoms, and to inquire whether or not it be still in existence and operating. When the apparent cause is overwork, whether of brain or of muscle, the patient must be warned to abandon his work for a time, or to do less work. All nervous patients whose occupations are sedentary, or who are disposed to mope within doors, should be urged to take active walking exercise in the open air for at least an hour daily. To this it is often objected that they cannot afford the time; but the reply is that if they will perseveringly act upon the advice they will preserve their health and economise their time. They will actually do more work and they will be less fatigued and distressed by their labours. I have before referred to the importance of relieving the patient from any groundless fear as to the state of his health. The anxious patient will often be half-cured at once by the confident assurance that he has no disease which is not remediable; and to give this assurance is one of the most agreeable duties that we can ever have to perform.

When all that is possible has been done to discover and avoid the causes of nervous disorder—when bad and unwholesome habits have been corrected, and when all needful advice and encouragement have been given, we have next to direct our attention to the consequences, some of which will continue long after the cessation of their exciting cause, while others are perpetuated by some persistent and unavoidable source of brain trouble.

Now, as we have seen, by far the most frequent consequence of overwork and anxiety, the one, too, which oftener

than any other is productive of further disorder and mischief, is sleeplessness, or some form of disturbed and unrefreshing sleep; and the chief remedy for this, after the discoverable causes have been as much as possible removed, is a soporific at bedtime.

Before the introduction of chloral hydrate I was in the habit of treating these cases with some preparation of opium or morphine; and the result, in a large proportion of cases, was highly satisfactory. I have already given some cases showing the striking benefits resulting from the temporary use of opium at bedtime. The following case is another illustration of the same principle of treatment.

CASE 13.—L. H——, a widow, 41 years of age, was first seen at the dispensary on January 6, 1851. For many months her sleep had been disturbed by frightful dreams and spectral visions. Her friends told her that she talked as much in her sleep as when she was awake. She complained of a painful sense of weight on the top of the head, and her countenance was expressive of great anxiety. She drank neither beer nor spirits. She had been a widow eighteen months, and for many months before that she had suffered much from anxiety in consequence of the intemperate habits of her husband, who had squandered his money and failed in business. She had friends who were well off, so that she had never been in want; but her appetite was very bad, and she suffered much from flatulence and constipation. Before I saw her she had been cupped and leeched, and several times blistered, without benefit. On January 6 I prescribed five grains of compound soap pill, to be taken every night; and a laxative in the morning, to counteract the constipating effect of the opium. I told her to walk out daily, and I gave her hopes of speedy amendment. She came to me on January 13, and said that she had slept better the first night after taking the pill, and the second night better still; she had rapidly improved in every respect, and had a bright and cheerful expression of countenance. To continue the medicine. My last report of her, on January 17, is as follows:—She considers herself quite well: she sleeps well without dreaming; the sense of weight on the head is gone; her appetite is good,

and she has no discomfort after eating. She 'feels quite a different person.'

The simple explanation of this rapid recovery appears to be, that ten nights of sound and refreshing sleep had sufficed to remove the wearying effects of many months of anxiety and restlessness. Since the introduction of chloral hydrate as a soporific I have generally employed this valuable medicine as a substitute for opium in this class of cases, for reasons which I will now briefly explain.

One of the most serious objections to the use of opium as a soporific is its notorious tendency, in some cases, to produce an effect the direct opposite of that which we desire—to cause wakefulness and excitement instead of sleep and composure. This may sometimes be obviated by changing the form of the medicine or the mode of administration: by giving morphine hypodermically, or opium by the rectum; in other cases, by combining the opium with a small dose of antimony (the tartarated antimony) or James's powder—a combination which was strongly recommended by the late Dr. Graves to procure sleep and check delirium in some cases of fever. There are, however, some patients on whom opium or morphine, in any form or in any dose, has the effect of causing troublesome constipation, nausea, vomiting, distressing prostration and faintness, and sometimes severe headache.

The advantages of chloral hydrate are chiefly these. It is a far more certain soporific than opium, so that when given in a safe but sufficient dose of fifteen to thirty grains it rarely fails to procure sleep. Then it comparatively seldom gives rise to any unpleasant symptoms. Its nauseous taste sometimes, though rarely, excites vomiting; and in some few cases I have known it cause headache. It disturbs the digestive organs much less than opium; and it rarely, if ever, causes constipation. One of the most common and most disagreeable effects of chloral is its tendency, in some cases, to cause redness of the eyes and flushing of the face—a tendency which is much increased by even a small quantity of wine or alcohol in any form. Some years since I was consulted, for the first time, by a nervous lady, who complained to me that for the last fortnight she had been distressed by finding that

if she took a single glass of wine at lunch her usually pale face became suffused with a crimson flush. I at once suspected the cause, and asked if she had been taking chloral. She had been taking twenty grains of chloral every night for a fortnight, the exact time during which the flush of the face had occurred. The chloral was discontinued, and her face soon ceased to be flushed.

The general result of experience is, that while in suitable cases of nervous excitement and wakefulness, chloral hydrate as a soporific is far superior to the preparations of opium, it is much inferior to the latter as an anodyne in cases of neuralgia.

The average dose of chloral for an adult is twenty grains. I have rarely given more than thirty grains. The rule is to give the smallest dose that will suffice to procure refreshing sleep, and so to break the habit of wakefulness or dreaming restlessness. In most cases this object will be attained by a nightly repetition of the dose for a week, with perhaps a diminished dose for another week. It is seldom necessary or desirable to continue the medicine for more than a month, though, in some cases, it may be necessary to extend the period considerably. As to this, it is difficult to prescribe a general rule. In many cases I have found that the beneficial effects of the medicine have been immediate ; the patient has slept soundly, the distressing dreams have ceased, the appetite has returned, and the patient has rapidly regained strength and spirits. After a few nights of sound sleep have been thus procured, the chloral should be discontinued, and in most cases the patient will sleep as well without the medicine as with it. There is probably no method of treatment which has the power of rapidly removing such a number and variety of distressing symptoms as chloral or opium when the soporific action is really favourable in this class of cases, the benefit being due, not to the direct effect of the narcotic, but to the marvellous influence of sleep in refreshing both body and mind. In some cases, when chloral alone has failed as a soporific, I have found a combination of chloral with morphine successful, while in other cases the chloral has been usefully combined with from ten to twenty grains of bromide of potas-

sium or ammonium. In others, again, the bromide alone, in twenty-grain doses at bedtime, has had a soothing and beneficial effect.

But may not the frequent repetition of a soporific dose of chloral or opium become a necessity for the patient? This is a question which we are bound to face. There are few results of medical practice which I should more regret than the reflection that I had, in any way, contributed to make recourse to narcotics or stimulants habitual or necessary to a single patient. I believe, however, that a cautious use of chloral is attended with little danger of leading to so terrible an abuse of the drug. As I have before said, the medicine should be discontinued as soon as it can be dispensed with; as soon, that is, as restlessness and frightful dreams have ceased to harass and exhaust the patient. The rapid convalescence, and the renewed health and strength and spirits, which are so wonderfully promoted by securing sound and refreshing sleep, will generally enable the patient at once and without difficulty to dispense with artificial aid. I should strictly forbid narcotics of every kind for a patient who neglects directions which have been given him as to exercise, diet, &c., and whose nervous restlessness appears to result from such negligence. In other words, I would not encourage a patient to trust habitually to chloral or opium for the removal of discomforts which might be avoided by the exercise of self-control and obedience to sanitary laws.

The chloral hydrate, which, with the precautions before mentioned, I look upon as a safe and most valuable remedy, is subject, at the present time, to two kinds of *abuse*: while, on the one hand, it is indiscreetly, and therefore injuriously, employed by some practitioners, and perhaps by some patients, without medical advice; on the other hand, it is decried by some physicians as a pernicious and dangerous drug. It is our duty to avoid these two unreasonable forms of abuse. If the right use of a remedy is to be condemned because it is liable to be wrongly and mischievously employed, then with the disuse of hydrate of chloral must follow that of opium, alcohol in its various forms, and, indeed, of all our most valuable remedial agents; for of each and all of them the

maxim is true—'Nullum remedium pro auxilio est nisi tempestivo usû tale fiat.'

After all that I have said of the value of choral and of opium in suitable cases, we must not fall into the routine practice of giving narcotics to every patient who complains of inability to sleep. Our first care is to discover, and then, if practicable, to remove, the cause of the insomnia. We meet with some indolent patients for whom the best soporific is some regular useful occupation and daily active exercise in the open air; for others, who are feeble, tonics and nutritious food, with a moderate allowance of wine, will be the appropriate remedies; and, again, in other cases dyspeptic symptoms will cease, and refreshing sleep will return, under the influence of a scantier and a carefully regulated diet, with an occasional purgative. In cases such as these, narcotics would not only be unsuccessful, but would probably be injurious.

The cases of nervous disorder in which the treatment by hypnotics is most rapidly and completely successful are those in which the symptoms are the result of some bygone grief or anxiety, the impression of which remains and is perpetuated by inability to obtain refreshing sleep.

Another class of cases in which equal benefit is often derived from a similar plan of treatment are those in which the nervous symptoms have been induced by continued overwork, whether mental or bodily. In such cases it is obviously desirable that the patient should have a complete rest, or, if possible, that he should lessen the amount of his work. It is a remarkable fact that when overwork has induced that disordered condition of nervous system which I have described, the mere cessation of work does not bring with it the required rest so long as the sleep continues to be disturbed by dreams. This habit of dreaming requires to be broken before rest and refreshment come. The overworked solicitor's clerk (p. 205) was more rested by a few nights of unbroken sleep than by a month's holiday; and Dr. —— (p. 206) obtained by the same means, while still going on with his work, relief from distressing symptoms which rest at Brighton had failed to afford him. I have seen a considerable number of cases of overworked

men in which a few nights of refreshing sleep obtained by chloral or opium at bedtime have effected a greater restoration of brain power than had previously been obtained by a long abstinence from work while the sleep continued to be broken by dreams.

The cases which are the least likely to be benefited by this or by any other plan of treatment are—1st. Cases of confirmed hypochondriasis or melancholy of very long duration, and especially when they assume the form of so-called religious despondency. This pitiable state of despair is not unfrequently the result of alarm excited by the frantic ravings of some half-educated and wholly irrational preacher, whose caricatures of the Creator and the moral government of the world are as frightful to contemplate as the most hideous figure of any bogie that was ever dressed up for the purpose of exciting terror. 2nd. Cases in which extreme nervousness has resulted from great terror or a sudden violent mental shock, which has left a deep and indelible impression upon the mind and nervous system. 3rd. Lastly, cases in which the symptoms of nervous disorder are perpetuated by some present unavoidable and irremovable source of anxiety or sorrow. Every practitioner of large experience knows that cases of this kind are only too common.

When insomnia and nervous disorder are the result of excess of alcohol, with a deficiency of nutritious food, the main point is to withdraw or lessen the poisonous alcohol, and to substitute wholesome nutriment. In these cases, after the nervous system is calmed and tranquillised, sleep returns without the aid of narcotics; but I look back with much satisfaction to some cases in which, by a temporary use of a soporific at bedtime, I have greatly assisted a patient to break through a habit of chronic intemperance. I will give some brief particulars of one case of this kind.

CASE 14.—F. R——, aged 40, a carpenter, came to me at the dispensary on May 27, 1850. For some years he had been in the habit of drinking rather freely both beer and spirits; he had worked hard, and had suffered much from anxiety consequent upon family misfortunes and disagree-

ments. For many months past he had been troubled by vertigo and headache; his sleep was disturbed by frightful dreams and spectral visions: his appetite was bad, his digestion disordered, and he often suffered from vomiting. He had been leeched about a dozen times for the vertigo. Latterly he had been almost incapacitated for work. His countenance was anxious, his eyes suffused, and his tongue coated and tremulous. I prescribed two grains of opium in a pill at bedtime every night, and an aperient of rhubarb and magnesia occasionally; and I advised him to take no beer or spirits. Four days after (on May 31) the sleep had been somewhat quieter, and he felt rather better. On June 3 the dose of opium was reduced to one grain, a mixture of the sulphate of quinine and iron was prescribed, and an aloetic pill occasionally. On June 18 the report runs thus:—He has continued to take the medicine; he sleeps well without dreaming, awakes refreshed, and feels a pleasure in commencing his work at five or six o'clock in the morning, whereas before he had some difficulty in being up by nine o'clock. He feels much stronger and less nervous; has lost the dizziness; his countenance is much improved; his tongue is clean and not tremulous; his appetite is good. He has taken no stimulants, and has resolved to abstain entirely. The opiate was then omitted, but the tonic was continued.

There can be no doubt that in this case the vertigo and other symptoms were aggravated by the repeated leeching, and by the continued drinking of beer and spirits while the appetite was bad and the digestive powers impaired. He was quickly restored to health by sleep and by the recovery of the power to eat and digest his food.

As the habitual abuse of alcohol is notoriously a frequent cause of nervous disorder, so its cautious and judicious employment is in many instances a most important means of cure. We not unfrequently meet with cases of obstinate restlessness, either with or without delirium, the result of intense grief, or long-continued anxiety, or of watching and fatigue, or of some exhausting illness in which there is mental excitement proportioned to the general bodily weakness. In these

cases, often the usual narcotics entirely fail to procure sleep, even when they do not increase the excitement and distress; and the surest mode of arresting the collapse and calming the nervous system is to give wine or brandy more or less liberally but cautiously, with frequent supplies of beef-tea, milk, and eggs.

I once had the opportunity of seeing a case of acute mania successfully treated by a method somewhat out of the usual course.

CASE 15.—G. H——, aged 16, a bookfolder, was admitted into Twining ward on October 20, in a state of maniacal excitement, having been found in the street very scantily clothed and unable to give any account of herself. When placed in bed she constantly threw herself about, pulled off the bedclothes, and laughed and talked incessantly. Her friends stated that for the past four days she had complained of headache and giddiness, but she had continued her work until the day before her admission. The only assignable cause of her illness was that for the past fortnight she had been removed from a room in which she had been in the habit of working to one in which machinery was at work. On the day of her admission she had twenty grains of chloral hydrate at bedtime. She slept after this for a few hours, but when she awoke she was as noisy and violent as ever. She was removed to the refractory ward, and ordered to take twenty grains of chloral every three hours. After several doses of the medicine had been taken, it was evident that it had no beneficial soothing influence; the excitement and maniacal delirium were unabated. The house-physician, Mr. Philip Birch, now asked my permission to have her packed in a wet sheet and blankets. To this I assented, the more readily from my recollection of a similar case thus treated by my friend and colleague, Dr. Sheppard, and recorded in his able and interesting *Lectures on Madness* (p. 58). Accordingly, on the evening of the 24th, the chloral having been discontinued, she was packed, and, as the excitement was evidently lessened, she was kept in the wet sheet and blankets for eight hours. On the following day (the 25th) the excitement was much less, but not entirely subdued, and in the evening the packing was repeated for about three

hours. On the 26th, having had a good night and being quiet and rational, she was allowed to return to Twining ward; and on the 28th she was discharged cured.

In this case, the chloral having entirely failed to subdue the excitement and delirium, the wet pack effected a complete cure with great rapidity.

CHAPTER IX.

THE PATHOLOGY AND TREATMENT OF EPILEPSY.[1]

Prologue—Epilepsy not the Result of Congestion, but of Sudden and Extreme Anæmia of the Brain—Experiments of Kussmaul and Tenner—Convulsions from Sudden Arrest of the Pulmonary Circulation by Air or Salts of Soda in Veins, by Embolism, by Acute Apnœa—Evidence as to Cerebral Anæmia resulting from Arterial Spasm—Two Classes of Cases, Nervous and Toxæmic—Epilepsy is Cerebral Collapse, Cholera Collapse is Pulmonary Epilepsy—Partial Epilepsy a Result of Spasm in Limited Arterial Regions—Comparison of Epilepsy and Syncope—Rational Principles of Treatment in the Two Classes of Epileptic Cases—The Action of Chloroform, of Chloral, and of Bromides.

PROLOGUE.

Since this lecture was published, nearly twenty years ago, the extremely complex and difficult subject of which it treats has been laboriously investigated by many able physiologists and physicians; amongst whom Dr. Hughlings Jackson is, on all hands, acknowledged to occupy a foremost position. The result of these inquiries has been to modify some of the theoretical views which were generally accepted when my lecture was written. For instance, not only are not all convulsions of an epileptiform character now looked upon as examples of true epilepsy—this distinction has always been admitted—but even true epilepsy is not now considered to be a clinical unity. Thus Dr. Jackson says:[2] 'There are really many different epilepsies (I mean what would be called "varieties" of genuine epilepsy), each dependent on a "discharging lesion" of some part of the highest centres;' and he goes on to explain that 'epilepsies are only one class of fits ("highest level fits"). There are, besides, different epileptiform seizures from "discharging lesions" of the middle motor centres ("middle level fits"),' and also, as he thinks, 'different

[1] *British Medical Journal*, March 21, 1868.

[2] *The Journal of Mental Science*, April 1887.

fits depending on discharges beginning in different parts of the lowest level of central evolution ("lowest level fits")." It is impossible by brief extracts to do justice to Dr. Jackson's views. He would confer a great benefit upon the profession and upon the public if he could find time to collect, arrange, and codify the numerous valuable papers of his which, being scattered through various journals, are now difficult of access.

To return to my lecture. If I were now to rewrite it I should modify some of my expressions. For instance, I should in some parts use the more general term 'convulsions' instead of 'epilepsy.' But inasmuch as the experimental results obtained by Kussmaul and Tenner, and the recorded clinical facts which prove that sudden and extreme anæmia of the brain has for one almost invariable result the occurrence of epileptiform convulsions, are unquestionably true, I have thought that the lecture would still have sufficient interest, and to many readers be sufficiently instructive, to warrant its republication. I have, therefore, determined to reproduce it in its original form, with the addition of an occasional paragraph or a note which I have marked as recent.

I purpose upon the present occasion to attempt an answer to the following question—To what extent do the phenomena of an epileptic fit admit of explanation? In a fully-developed epileptic fit, there are two chief phenomena to be explained: these are loss of consciousness and convulsions. The loss of consciousness was formerly supposed to be a result of congestion of the nervous centres, and especially of the cerebrum. But this explanation is inconsistent with the fact, that the epileptic loss of consciousness comes on in a moment, at the very commencement of the attack, when there is no evidence of congestion and when the face is usually pale from anæmia. The congestion follows the loss of consciousness; and the explanation of its occurrence appears to be this. The convulsion implicates the respiratory muscles; the blood, therefore, being imperfectly aërated, is impeded in its passage through the lungs; it consequently accumulates in the right side of the heart and in the veins. The congestion is a

secondary venous congestion, and, at the time when this congestion has reached its greatest height, there is often a commencing return of consciousness. Obviously, then, this congestion is not the cause of epileptic loss of consciousness. This retrograde venous engorgement is the cause of the ecchymoses beneath the skin and the conjunctiva which often occur during a fit, and of the hæmorrhage into the substance or upon the surface of the brain, which happily is a much less frequent accident.

A number of facts point to the conclusion, *that both the loss of consciousness and the convulsions of epilepsy are the result of sudden and extreme anæmia of the brain.*

In man, and in most, if not in all, warm-blooded animals, a rapid and very copious hæmorrhage usually causes convulsions. Kussmaul and Tenner state[1] that, in numerous cases of dogs, cats, and rabbits, they observed, without a single exception, violent and general convulsions preceding death from loss of blood. In order to produce this result, the hæmorrhage must be rapid. If it occur slowly, so that the vital powers are gradually consumed, death then occurs with swooning, drowsiness, and delirium, without convulsions.

The same observers found that an interruption in the supply of blood to the head of a rabbit, by ligature or compression of the arteries, produces epileptic fits as surely as hæmorrhage does. In about one hundred rabbits they ligatured or compressed the carotids and subclavians, from which, be it remembered, the vertebrals proceed, and in every instance except that of one very old, lean, and feeble rabbit, convulsions occurred. In order to produce convulsions it was necessary to close all the four arteries which supply the brain. If but one carotid or one subclavian remained pervious, the animal was enfeebled and more or less paralysed, but not convulsed. And, again, if during the height of a convulsion the ligature is removed from one carotid, the convulsions generally cease immediately, and there is a sudden change from the most frightful spasm to complete relaxation of the muscles. The description of the convulsions thus artificially produced in these animals shows that they were essentially the same as

[1] *On Epileptic Convulsions from Hæmorrhage*, New Sydenham Society, 1859.

epileptic convulsions in the human subject. There was the dilated pupil, the tonic spasm, quickly succeeded by clonic convulsion, so violent as to throw the animal forcibly forwards to a distance of one or two feet, and sometimes even over the shoulders of the experimenter. These experiments suffice to show the fallacy of the explanation which Dr. Brown-Séquard and others have given of the clonic convulsions in man. It has been supposed that the clonic convulsions are a consequence of the circulation of black blood which results from the tonic spasm of the respiratory muscles. Now it is manifest that, in these animals with ligatured arteries, no black blood could reach their brain. In them, therefore, the clonic convulsion, as well as the preceding tonic spasm, must be due to want of blood, and not to the altered quality of blood in the brain.

These experiments obviously cannot be repeated on the human subject; but Drs. Kussmaul and Tenner describe the effects of compressing the carotids in six men. In all, the face turned pale; the pupils first contracted and then dilated; the respiration became slow, deep, and sighing; then there was giddiness, staggering, and unconsciousness, and the patients would have fallen had they not been supported. 'In two subjects, of weak intellect and moderately anæmic, in whom, notwithstanding the above symptoms, the compression was continued, a choking sensation, attended by vomiting and general convulsions, came on, which, however, did not attain an aggravated form, for, on withholding the compression, they disappeared in a few seconds.' [1]

Compressing the carotids does not, of course, entirely cut off the supply of arterial blood to the brain; but these experiments render it probable that sudden occlusion of all the arteries of the head will as certainly excite epileptic convulsions in man as in the lower animals.

There is a class of cases in which a sudden arrest of the blood in its passage through the lungs causes convulsions and speedy death. I mean cases in which the circulation is arrested by the admission of atmospheric air into the veins; cases of embolism of the pulmonary artery; again, cases in which the flow of blood through the lungs is stopped by the

[1] *Op. cit.*, p. 28.

injection of certain salts into the veins; and, lastly, cases of acute apnœa. When animals are killed by blowing air into the veins, the breathing becomes hurried, the animal falls down, and usually dies in convulsions; the contents of the bladder and rectum being frequently expelled at the time of death. Dr. John Reid states that 'in a very few cases only is death from this cause not preceded by convulsions.' [1] The immediate cause of death in these cases is the arrest of the frothy mixture of air and blood in its passage through the minute pulmonary arteries (the air rarely reaches the left side of the heart); and, as a result of this arrest, there is, of course, anæmia of the brain and of every other organ supplied by the systemic arteries.

In man, it appears that death from the admission of atmospheric air into the veins has been less frequently preceded by convulsions. Probably the chief reason of the less frequent occurrence of convulsions in the human subject is, that the amount of air accidentally admitted is less, and death, consequently, is less rapid than when air is forcibly driven into the veins of an animal. It would probably be found, on a careful inquiry, that the occurrence of convulsions in these cases depends upon the circulation being suddenly and completely arrested.

Convulsions are mentioned in only 5 out of 15 cases collected by Amussat; but, Dr. Reid remarks, 'several of the cases, as we might have expected, are very imperfectly reported; for it is not to be supposed that the surgeon or his assistants should possess the coolness and time to watch narrowly the phenomena, when their minds were agitated by the threatened sudden dissolution of their patient, and their attention distracted by anxious attempts to save him.' For the same reason, the record of cases of pulmonary embolism is very imperfect; but in some instances it has been observed that death was preceded by violent convulsions; and Virchow noted, amongst the results of artificial embolism of the pulmonary artery in animals, convulsions and dilatation of the pupils.[2]

In a subsequent chapter on *Thrombosis and Embolism*

[1] *Physiological, Anatomical, and Pathological Researches.*

[2] *Des Emboles Pulmonaires*, par B. Ball, p. 129.

I have recorded a case (Case 1 in that chapter) in which a thrombus detached from the wall of the right auricle completely occluded the tricuspid orifice and caused convulsions and death in a few seconds.

Again, Blake found that an injection of a solution of soda or its salts into the veins of a dog destroys life by arresting the flow of blood through the lungs. The left side of the heart is found empty, and the right distended. Death occurs in about forty-five seconds, and is preceded by violent opisthotonos.[1]

Blake attributes the nervous symptoms to the venous pressure on the brain; but they may, with much more reason, be attributed to the arrest of the arterial supply to the brain. When, from any cause, the blood is arrested in its passage through the lungs, it is obvious that distension of the systemic veins must have, as its necessary correlative, comparative emptiness of the systemic arteries; the one will be an index and a measure of the other.

Lastly, we have the convulsions which occur in almost every case of acute apnœa or sudden suffocation. It is generally supposed that the convulsions of apnœa are excited by the circulation of black blood through the brain; but they are more probably due to the rapid and extreme anæmia of the brain consequent upon the impeded transit of blood through the lungs. When the air is excluded from the lungs, the circulation is rapidly arrested by the contraction of the minute pulmonary arteries. That this is the true explanation of the convulsions of apnœa is rendered highly probable by an observation of Kussmaul and Tenner,[2] to the effect that 'the approach of convulsions in strangulation can be accelerated if the arteries are simultaneously compressed.' It is obvious that, if the presence of black blood in the brain were the cause of the convulsions, their approach would be retarded, and not accelerated, by compression of the arteries which supply the brain. The facts are consistent only with the theory that the immediate cause of the convulsions in cases of suffocation is a rapidly increasing cerebral anæmia, resulting from the arrest of the pulmonary circulation. Unaërated venous

[1] *Edinburgh Medical and Surgical Journal*, vol. liv. p. 343.

[2] *Op. cit.*, p. 75.

blood, in so far as it is deficient in oxygen, is equivalent to no blood. Probably it is rather by its negative quality of being unoxygenised, than by any positively noxious properties, that it is unsuited to maintain the functions of the brain. It is probable, too, that the minute cerebral arteries resist the passage of black blood, and so increase the anæmia of the brain. If the circulation of dark blood through the cerebral vessels would excite convulsions, we should expect to find this symptom of common occurrence in cases of emphysema with bronchitis.

The epileptiform convulsions which occur in a large proportion of cases when nitrous oxide gas is inhaled until the pulse at the wrist disappears are explained in part, perhaps, by the deoxidation of the blood, but mainly by the sudden diminution of the blood-supply to the brain consequent on the arrest of the circulation through the lungs.[1]

I have now referred to instances of epileptiform convulsions occurring under a considerable variety of circumstances, but all agreeing in this one condition—namely, that the convulsions are associated with a defective supply of arterial blood to the brain. Let us now proceed to inquire whether the phenomena of epileptic convulsions, as they ordinarily occur in the human subject, are consistent with the theory of anæmia. It is a matter of general observation that, at the very commencement of an epileptic fit, the face is pallid. There is anæmia of the superficial vessels; and with this there is probably associated anæmia of the intracranial vessels which supply the brain itself. The pallor is in most cases soon succeeded by lividity, owing to venous engorgement consequent upon the impeded respiration and pulmonary circulation. It is very remarkable that, while the face is pallid, the heart and the carotids are beating strongly. It is probable, therefore, that there exists some impediment to the flow of blood through the minute branches of the arteries. To explain this impediment, Kussmaul and Tenner suggest that the minute arteries, both the superficial and the intracranial branches, contract so as to bar the passage of blood. Hence arise the pallor of the face and the epileptic convulsion. In some cases it is said that the

[1] See Chapter II. *On the Physiology of the Circulation*, p. 33.

face is more or less livid at the very commencement of the fit. The probable explanation of this is, that the respiratory muscles are convulsed, and there is a consequent venous turgescence before the spasm affects the facial arteries. The spasm of the *facial* arteries, though usually present, is obviously not the cause of the fit. The early implication of the respiratory muscles is clearly shown in those cases in which the 'epileptic cry'—a result of spasm of the glottis—is the first indication of the fit.

Kussmaul and Tenner endeavoured to support the theory of arterial spasm by experiment, and to some extent they succeeded. In each of three white rabbits they ligatured the two subclavians and one carotid; the cervical sympathetic was then exposed and galvanised, with a view to excite contraction of the arterioles by the stimulus conveyed through the vaso-motor nerves. In two animals, no effect was produced: but in the third rabbit the background of the eye became completely pale; the pupil dilated, so that the iris could scarcely be seen; the neck was drawn back; and violent convulsions occurred. The electrodes being removed, the spasms ceased, the pupil contracted, and the background of the eye became red; but the animal continued in a swooning condition. After some minutes, electricity applied to the sympathetic nerve produced the same effects as at first. A third attempt did not succeed.

The authors suggest that these experiments deserve repetition, with the view of rendering certain what at present is probable — namely, 'that epileptic convulsions can be brought about by contraction of the blood-vessels induced by the vaso-motor nerves.'

[It is to be observed, however, that the arterial spasm, assuming it to exist, is a secondary result, and not the starting point of the epileptic fit. The theory in question supposes that a 'discharge' from some 'unstable' local brain-lesion acts, not directly through the musculo-motor nerves upon the convulsed limbs, but immediately through the vaso-motor nerves upon the muscular arterioles, the cerebral anæmia which results from their extreme contraction being the direct cause of the convulsion.

The experiments of Kussmaul and Tenner have conclusively proved that extreme anæmia of the brain suffices to cause violent convulsions, and their results receive confirmation from the various forms of arrested circulation before referred to, which are found to have for their common result similar convulsions. These facts, therefore, seem to afford strong support to the theory that, however diverse may be the remote exciting causes of convulsions, whether epileptiform convulsion or the convulsion of so-called 'true epilepsy,' they all have this result in common, that they cause anæmia of those parts of the nervous centre which are implicated.]

According to this theory, then, epilepsy is a result of sudden anæmia of the brain; and this anæmia, when not caused by hæmorrhage or by a mechanical impediment to the circulation outside the cranium, is due to an extreme contraction of the minute cerebral arteries. With reference to this theory of arterial contraction, all cases of convulsions may be arranged in two distinct classes: 1, cases in which the arterial contraction is the result of a purely nervous or reflex influence; 2, cases in which the arterial spasm is a result of blood-poisoning.

In the first class are included all cases of epilepsy which are due to emotional influence, sudden terror, or anger, or long-continued anxiety and sorrow, perpetuated and intensified often by nocturnal dreams and frequently recurring nightmare; also cases associated with those disordered states of the nervous system which are the result of vicious sensual habits; cases, too, in which the disease is hereditary. This class also includes cases of epileptic convulsions from the irritation of the gums during dentition; of the kidney, or the ureter, or the gall-duct, by a calculus; and of the alimentary canal by worms. Again, the cases in which convulsions result from tumours or other gross organic disease of the brain are included under the head of epilepsy from a reflex influence. The structural change in the brain is not the proximate cause of the epilepsy; it excites the epileptic convulsions through a secondary reflex influence upon the blood-vessels. Dr. Brown-Séquard's guinea-pigs, rendered epileptic by injury to the spinal cord, are also included in this class of cases. The

injury to the cord probably acts by increasing the reflex excitability of the nervous centres, so that a trifling external irritation suffices to excite a fit.

In the class of toxæmic convulsions are included those cases in which noxious materials in the blood are the immediate exciting cause of the arterial spasm—uræmic convulsions, and all cases of convulsions from retained excreta; convulsions resulting from the admission into the circulating blood of unwholesome and imperfectly digested food; the convulsions which result from alcoholism; the convulsions which sometimes occur during the initiatory stage of certain of the acute febrile exanthemata, more especially small-pox; the convulsions which are occasionally associated with pyæmic infection; and the convulsions resulting from a poisonous dose of prussic acid. In each and all of these cases, it is probable that the immediate cause of the convulsion is anæmia of the brain, resulting from contraction of the cerebral arterioles; the arterial spasm being excited by the presence of morbid blood in the vessels.

In the hypertrophy of the muscular walls of the arteries of the pia mater, which we find in cases of Bright's disease, we have evidence of a continual resistance to the passage of the deteriorated blood through these vessels. This resistance probably explains some of the cerebral symptoms of Bright's disease; but a convulsive seizure must be due to a sudden temporary increase of arterial contraction—this sudden contraction differing as much from the continuous tonic contraction of the vessels as the cardiac spasm of angina pectoris differs from the regular strong contractions of a hypertrophied ventricle. With reference to the action of prussic acid upon the blood-vessels, it is noteworthy that Blake, having killed a dog by injecting prussic acid into the jugular vein, observed that, after the animal had ceased to struggle, the dynamometer in the femoral artery still indicated a considerable increase of pressure. And W. Preyer[1] has shown that prussic acid robs the blood of its oxygen. The high pressure in the systemic arteries is probably caused by contraction of the systemic

[1] *Die Blausäure, Physiologische Untersucht.* Bonn, 1868. Reviewed in the *Practitioner*, August 1868.

arterioles excited, not by the direct action of the acid upon the vessels, but by the influence of the deoxygenised blood—an influence analogous to that exerted by the inhalation of nitrous oxide gas; and this contraction affecting the cerebral arterioles would account for the convulsions in cases of prussic-acid poisoning. Again, the symptoms which result from an over-medicinal dose of prussic acid are such as might be occasioned by a less degree of obstruction to the cerebral circulation. These symptoms, as described by Pereira, are the following: 'Disordered and laborious respiration (sometimes quick, at others slow and deep), pain in the head, giddiness, obscured vision, and sleepiness. In some instances faintness is experienced.' These symptoms are remarkably like those of the epileptic vertigo, or *petit mal*, as it is called; and, like them, they are probably due to a temporary and partial interruption of the cerebral circulation by arterial spasm. The poison, being very volatile, is quickly exhaled by the lungs; and the symptoms soon cease. In a future chapter I shall record several cases of poisoning by a so-called homœopathic solution of camphor, in most of which violent epileptiform convulsions occurred. It seems probable that in these cases the camphor, precipitated from the spirituous solution in a state of minute subdivision, enters the circulation and excites the contraction of the cerebral arterioles, which is the immediate cause of the convulsion. In accordance with this theory of arterial contraction, epilepsy might be designated *cerebral collapse*; and, on the other hand, the arrest of the circulation by the contraction of the pulmonary arteries in the collapse of cholera may be looked upon as a form of *pulmonary epilepsy*. It would seem, then, that the true key to the pathology of both these awful diseases, epilepsy and cholera, is to be found in this doctrine of arterial spasm.

The two classes of epileptic cases—the purely nervous and the toxæmic—have their analogues in the two varieties of laryngeal spasm. Spasm of the larynx in children may result from irritation of the brain, or of the gums, or of the alimentary canal; in short, it may be a purely nervous reflex *laryngismus stridulus*, which is closely allied to epilepsy. On the other hand, laryngeal spasm may be excited by a crumb of bread or

a grain of salt, or other irritant, within the larynx; and this is analogous to the arterial spasm which is excited by toxæmia, and which may result in an epileptic fit.

There are cases of convulsions in which the purely nervous and the toxæmic elements are combined in varying degrees—cases of so-called idiopathic epilepsy, in which the immediate exciting cause of a fit is some blood-contamination consequent upon disordered digestion or retained excreta; and again, cases of toxæmia in which a paroxysm of convulsion is provoked by some emotional or other nervous excitement. In many cases of puerperal convulsion there is a combination of the toxæmic and the purely nervous influences—a scanty secretion of albuminous urine and consequent uræmia, together with the exalted excitability of the whole nervous system which often accompanies the puerperal state, and which is highly intensified during the pains of parturition.

There are various forms of what may be called partial epilepsy: sudden and transient impairment of motor power, or irregular spasmodic movements limited to a particular set of muscles; various disordered sensations in limited portions of the skin; derangements of the special senses; sudden perversions of taste, or smell, or sight, or hearing; sudden impairment of speech; vertigo; confusion of thought; temporary delirium, and mental excitement. One or more of these symptoms may occur singly or variously combined in different cases, the onset and the departure being often equally sudden. These phenomena may probably result from a sudden temporary interruption of the blood-current through one or more branches of the cerebral arteries by spasm of their muscular walls; so that the brain-tissue within a circumscribed 'arterial region,' having its nutritive supply arrested or limited, would suffer a suspension or impairment of its proper functions. This appears to be a very probable explanation of the curious phenomena in question. It must be borne in mind that the brain is not one organ, having a simple function, like a lung or a kidney, but that it is a congeries of complex organs, having very diverse functions. It seems probable that the physiological co-operation of all these cerebral organs may require that the blood-supply to the various regions of the

brain should be specially regulated by certain branches of the arterial tree, under the guidance of the vaso-motor nerves; and this regulating power residing in the arteries probably renders them liable to disorderly action under the disturbing influences of disease.

During the disorderly convulsive movements of epilepsy nerve force is converted into motion, and this is followed by a state of exhaustion; so that for a time the reflex excitability of the nervous centres is lessened. When fits recur at short intervals they are usually less violent; on the other hand, a very violent paroxysm often follows an unusually prolonged immunity from the attacks. It would appear that the nerve force, which is stored up during the intervals of the attacks, is discharged during the convulsive seizure. The convulsive movements are probably, in part, at least, a result of the suspension of the functions of the cerebral hemispheres, whereby the controlling influence of the will is cut off from those lower centres which more directly influence muscular movements. There appears to be some analogy between epileptic convulsions and the involuntary reflex movements which are common in paralysed limbs, and which cease with the return of voluntary power over the limbs. Cases of hemi-convulsion, without loss of consciousness, may on this theory be explained by supposing that while the circulation through the hemispheres remains uninterrupted, there is a sudden temporary occlusion of the arteries which supply the corpus striatum; and the irregular movements then result from the controlling influence of the hemispherical ganglia being thus cut off from a portion of the motor tract. There is an immense reserve of latent force stored up in the nervous centres. This force is normally under the control of the will; but it is liable to sudden and explosive discharges when the machinery of volition is out of gear, as it is during an epileptic fit. The influence of the will in warding off a fit is shown by the fact that, in most cases, the attacks are more frequent during sleep, while, in some instances, they occur only during sleep when the will is off guard.

An arrest of the cerebral circulation from any cause, of necessity, involves a suspension of the functions of the brain.

For this suspension of function it is not necessary that the vessels should be empty; they may be full, and even gorged with blood; but, if the blood be stagnant, the effect is essentially the same as if they were bloodless. Great venous turgescence may so retard the current of blood in the capillaries as to suspend the functions of the brain. Some time since I saw two patients, in each of whom a sudden and complete loss of consciousness occurred during a violent fit of coughing. One man fell and cut his temple during the brief loss of consciousness. The explanation of such an attack is, that during the violent expiratory efforts of coughing the blood is driven back into the veins, which consequently become so turgid as to check the capillary circulation within the brain. So that, whether the suspension of the cerebral functions be the result of the arrest of the arterial or of the venous current, in either case the symptoms are immediately due to defective capillary circulation. In like manner, the functions of the kidney are suppressed by ligature of the renal vein no less than by ligature of the renal artery. The functions of every organ require for their discharge a continual supply of moving blood. Great confusion has often resulted from a disregard of the undoubted fact that the activity of the circulation through an organ is often in an inverse ratio to the 'congestion' of its small vessels. In other words, the vessels are gorged because the blood cannot readily pass through them.

Epileptic loss of consciousness differs from ordinary syncope in the suddenness of its occurrence. We have already seen that anæmia of the brain, to cause convulsions, must be sudden and extreme. In cases of syncope the circulation *gradually* fails, in consequence of diminished cardiac power, and there may be complete loss of consciousness without convulsions. Syncope, in proportion to its suddenness, approaches in its character to epilepsy, and there are certain cases of fainting, with semi-convulsive shudderings, which show that the boundary between epilepsy and syncope is sometimes ill-defined and difficult to trace.

During a severe and prolonged convulsion there is usually much rattling noise over the chest, resulting from the mixture of air and mucus in the bronchial tube, and frothy mucus is

forcibly driven from the mouth and nose. This mucus is an exudation from the bronchial mucous membrane, and the explanation of it appears to be this. While the respiratory muscles are in a state of spasm—respiration being imperfectly performed—the movement of blood through the lungs is impeded by the contraction of the pulmonary arterioles; there is consequently an accumulation of blood in the right side of the heart and in the veins; and this venous stasis, taking a retrograde course, distends the bronchial veins and capillaries, which relieve themselves by a mucous exudation into the bronchial tubes. Sometimes the capillaries are ruptured, and the mucus is tinged with blood, even when the tongue is not bitten and bleeding. Precisely similar phenomena occur in all the forms of apnœa; for example, during a fit of spasmodic asthma, during the death struggles of drowning and suffocation, during the coma and the consequent apnœa from brain disease or narcotic poisoning; and what is vulgarly called the 'death-rattle' is a noisy mixture of air and mucus in the bronchial tubes, the mucus being a passive exudation from the bronchial capillaries, the result of an increasing accumulation of blood in the right side of the heart and in the systemic veins, while respiration and circulation are simultaneously failing.[1]

Extreme dilatation of the pupil appears to be a constant phenomenon at the commencement of the epileptic seizure. This is usually attributed to spasm of the radiating fibres of the iris under the influence of the sympathetic nerve. It is a positive fact that galvanising the cervical sympathetic dilates the pupil at the same time that it excites contraction of the minute arteries, and a similar influence is supposed to operate in epilepsy. It is, however, conceivable that the dilatation of the pupil at the commencement of an epileptic fit is a secondary result of the extreme anæmia of the retina, just as the pupils of a chlorotic girl are dilated until her natural colour has been restored by iron. The pupils are dilated in cases of ordinary syncope, when the retina is anæmic from failure of the circulation, and when there is no suspicion of spasm of the radiating

[1] See Chapter IV. *On Some Results of a Retrograde Engorgement of the Blood-vessels.*

fibres of the iris excited by a stimulus being conveyed through the sympathetic. And again, in Kussmaul and Tenner's experiments upon animals and upon men, it was found that when the brain, and of course, the retinæ, were rendered anæmic by ligature or compression of the arteries, the pupils dilated as in an ordinary epileptic fit. Yet here there is no reason to suppose a primary influence of the sympathetic acting simultaneously upon blood-vessels and iris. On the other hand, it may be argued that the dilatation of the pupil in these cases is a result of spasm of the radiating fibres of the iris, and so constituting part of the general convulsion, consequent on extreme anæmia of the nervous centres. The question is one of physiological interest, but it has no practical importance.

Treatment.—It has been too much the custom to look upon epilepsy as a separate entity without reference to the physiological history of the disease, and the treatment has, for the most part, been a blind empiricism. The sources of epilepsy may be as numerous and as varied as the sources of apnœa, with which, both as a cause and a consequence, it is so closely correlated. To prescribe for epilepsy without reference to its cause is as unscientific as to prescribe for loss of voice without first ascertaining upon what conditions within the larynx or elsewhere the aphonia depends. As aphonia has for its immediate cause a defective vibration of the vocal cords, so the proximate cause of epilepsy is a defective blood-supply to certain parts or to the whole of the brain. The problem in each case is to ascertain the antecedent conditions and the circumstances which have led to the functional defect.

A priori considerations might have taught us what experience amply confirms—namely, that in the two classes of epileptic cases very different remedies are required. In the purely nervous cases, the indication is to remove all sources of irritation, whether mental or bodily, to soothe the nervous system by anæsthetics, and to impart vigour by a well-regulated nutritious diet and by tonics. On the other hand, in the toxæmic cases, the primary object is to free the blood from noxious impurities. In effecting this object, active eliminative and depletory measures may be required ; as, for instance, in

cases of uræmic poisoning. A local anæmia, the result of a toxæmic influence upon the cerebral arteries, may co-exist with a general vascular plethora. The argument that epilepsy cannot be a result of cerebral anæmia because it sometimes occurs in plethoric subjects and is sometimes relieved by depletory treatment, appears to me to be based upon a misapprehension of the terms of this most interesting pathological problem.

Convulsions of the mixed character before referred to may require a combination of the evacuant and the sedative treatment.

It is not now my intention to enter into details as to the treatment of epilepsy; but there are three remedies upon which I desire to say a few words—namely, chloroform, chloral, and the alkaline bromides. It is a well-known fact that chloroform inhalation has a remarkable power of arresting epileptic convulsions. Its action in warding off a threatened fit, and in cutting short a violent and prolonged paroxysm, is uniform and certain; as uniform and as certain as the influence of sudden and extreme anæmia in exciting convulsions. I, for a time, supposed that the chloroform acts by relaxing the cerebral arterioles; but Kussmaul and Tenner have shown that animals when etherised get no convulsions while being rapidly bled to death, or when their arteries are ligatured. It is probable, therefore, that anæsthetic vapours prevent or stop convulsions by lessening the reflex excitability of the nervous system; so that convulsions do not occur in etherised rabbits, even though the brain be rendered extremely anæmic by hæmorrhage or by arterial obstruction.

A full dose of chloral (thirty grains for an adult) is most efficacious in putting a stop to a rapid succession of fits. Its action in soothing the nervous centre is probably analogous to that of chloroform, but the effect is more durable.

And here I may mention that chloral is a most invaluable remedy in cases of laryngismus stridulus, which, in children, is so often associated with epileptiform convulsions. I feel justified in saying that by means of this drug I have saved the lives of several children.

My practice has been to give to a child a year old one

grain of chloral, and to repeat the dose about every eight hours. The dose may be increased one grain for each additional year of age. For the permanent cure of laryngismus, tonics, such as cod-liver oil, and especially country air, in the case of children living in towns, are requisite.

Chloral appears to have a special soothing influence upon the nerves of the larynx, and so it is a most efficacious addition to diaphoretic and other remedies, when in a nervous child, catarrhal laryngitis is associated with spasm of the larynx, constituting the *laryngite striduleuse* of French authors.

Experience has proved that the alkaline bromides, in full and frequent doses and sufficiently long continued, are of great value in the treatment of epilepsy. Some years since I prescribed for a gentleman, aged 25, twenty grains of bromide of potassium three times a day. He had been epileptic and hemiplegic from infancy. For many years he had not passed a week without an attack, and the average number of fits was from seven to fourteen in a week. After commencing the bromide he had no attack for eleven months, and for a long time the frequency of the fits was greatly lessened.

The known physiological action of the bromides renders it probable that their beneficial action in epilepsy is a result of their soothing, sedative influence upon the nervous centres, whose reflex excitability they lessen.

In short, their action in preventing convulsions would seem to be analogous to that of chloroform and choral; differing, however, from them in being less powerful and rapid in operation, while by frequent and prolonged use their influence is rendered more permanently beneficial.

Some of the nervine tonics, such as quinine and zinc, and in anæmic cases iron, may be given with great advantage to some epileptics.

CHAPTER X.

THE PATHOLOGY AND TREATMENT OF RIGORS.[1]

The Phenomena of Rigors—The Various Circumstances under which they occur—Their Relation to Epileptic Convulsions—Both Rigors and Convulsions may result from Toxæmia or from a purely Nervous Reflex Influence—As Epilepsy has for its Proximate Cause Anæmia of the Cerebrum, so the Proximate Cause of Rigors is Anæmia of the Spinal Cord—Rigors are Spinal Epilepsy—Comparison of the Cold Stage of Ague with the Collapse of Cholera—Explanation of the Rigors which follow Hot Saline Injections into the Veins of Cholera Patients—The Prognosis and the Principles of Treatment—Chloroform stops Rigors as it stops Convulsions—Case of Constant Rigors and Coldness from Pressure of a Dislocated Axis on the Cord—Another Case of Frequent Cold Chills from Pressure of an Enlarged Odontoid Process on the Cord.

The subject of rigors as a symptom of disease is one of considerable interest both to the physician and the surgeon. Rigors occur under a great variety of circumstances, and their significance is very diverse in different cases. In one case, they may result from a formidable blood-infection; while, in another case, they may indicate nothing more than a transient nervous irritation. Whenever they occur, they demand immediate and careful attention; and it is always of the highest importance that their true significance be ascertained.

The phenomena of rigors are mainly these. The patient has a sensation of cold, which is sometimes positively painful, and which is often referred especially to the back. A common expression is, 'I feel as if cold water were running down my back.' With this sensation of cold there is associated a general tremulousness of the muscles; the teeth chatter; the whole body is shaken and agitated by the muscular shudder-

[1] *British Medical Journal*, vol. i. 1868.

ings; and the limbs are the seat of aching pains, of greater or less severity. The surface of the body often appears shrunk and livid; the hands shrivel; and the nails are blue. The pulse is usually small and feeble; the breathing is often quick and difficult. With all these appearances of coldness of the body, and with the subjective sensation of cold, the thermometer commonly indicates that the temperature is some degrees above the normal standard. After a time, the shivering ceases, and the patient begins to feel hot; while it may be that the temperature is no higher than it was before. The features now become full and florid; the pulse is full, as well as quick; there is dryness of the mouth, and thirst; then a moisture begins to appear on the skin, and soon this becomes a profuse sweat. With the commencement of the perspiration, or even before it, the temperature begins to fall, and soon reaches the normal point, or even passes below it. This is the usual course of an attack of rigors; but they vary in degree from a transient feeling of chilliness to semi-convulsive shudderings.

Now, in order that we may be enabled to give a physiological interpretation of the phenomena, we must consider the chief circumstances under which they occur.

First, then, a paroxysm of ague is a typical example of rigors followed by what is called the hot and the sweating stage of the fever.

Then the onset of many diseases, which are with good reason believed to result from a blood-infection, is usually marked by rigors occurring once or oftener: for example, small-pox, typhus, erysipelas, pyæmia, puerperal fever, &c.

Again, rigors occur at the commencement of some inflammatory diseases in which there is reason to believe that not only the distribution of the blood is altered, but that the quality of the blood is also abnormal. I refer to such diseases as acute rheumatism, pneumonia, and pleurisy. And rigors are not unfrequently associated with the passing into the circulation of crude and undigested materials from the alimentary canal.

Then there is a totally distinct class of cases, in which rigors of great severity result from a purely nervous impres-

sion. Thus rigors may be excited by the passing of a catheter through the urethra, or by the passing of urine over the abraded surface of a torn urethra. Again, severe rigors often occur during the passage of a renal calculus through the ureter, or of a gall-stone along the bile-ducts. Frerichs [1] states that, in one case of supposed ague that occurred in his practice, he tried quinine for a long time without benefit; and the cause of the rigors was not discovered until after death, when numerous calculi were found in the hepatic duct.

If we can ascertain that any one essential condition is common to all these diverse cases, we shall probably have arrived at the true physiological interpretation of rigors. Let us consider this question as we recently discussed that of the pathology of epilepsy.

Now, in the first place, I would remark that there is certainly some not very remote analogy and relationship between epileptic convulsions and rigors. This is shown by the fact that, in certain cases, they merge into each other and replace each other. Thus the initiatory fever of small-pox, which is commonly heralded by severe rigors, is not unfrequently, in children especially, associated with convulsions, which may recur again and again, and then finally cease when the eruption appears on the skin. Convulsions occasionally occur during the cold stage of a severe ague-fit. Pyæmic infection is usually marked by the occurrence of severe rigors; more rarely it is associated with convulsions. Sir James Paget has remarked upon this relationship; [2] and he states that once, having cut a gentleman for stone, there occurred soon after the operation a terrible rigor, followed by heat and swelling, and then by extensive suppuration in the cellular tissue over the chest. Again, some days after, there occurred another rigor; and this was followed by similar suppuration, and by other symptoms of pyæmia. Then, some days later, he had a severe epileptic seizure; and this was followed, in about the same time and in the same way as the rigors had been, by another suppuration. Then after phlebitis and other mischievous results of pyæmia, he ultimately recovered.

[1] *Diseases of the Liver*, New Sydenham Society, vol. ii. p. 516.

[2] *British Medical Journal*, August 16, 1862.

Another case referred to by Sir James Paget is that of a woman at St. Bartholomew's who had relapsing erysipelas. The earlier relapses were preceded by rigors of various degrees of severity; the last was preceded by violent epileptiform convulsions, and these were followed by three days of complete coma, which seemed to be relieved when the erysipelas appeared. During the rest of her life she showed no sign of brain-disease, and she died exhausted.

In the cases to which I have just now referred, the alternating rigors and epilepsy have been the result of toxæmia; but the same close relationship between rigors and convulsions is shown by the fact that both may be excited by a purely nervous influence. We have before seen that rigors are frequently excited by the irritant action of biliary and renal calculi. Convulsions are a less frequent result of the same irritation. Thus Frerichs states[1] that, as a result of the passage of gall-stones, 'in irritable individuals, reflex cramps occur, which may become aggravated into the most violent convulsions, resembling epilepsy.' And Dr. Prout says[2] that he has known the irritation and pain attending the passage of a renal calculus 'accompanied in one or two instances by epilepsy.'

Convulsions have sometimes, though rarely, been excited by the operation of catheterism. My friend Mr. Barnard Holt tells me that, in three or four instances, he has seen men of nervous temperament, but not previously epileptic, thrown suddenly into a state of unconsciousness and convulsions by the passing of a catheter through the urethra.

It is plain, then, from these facts, that rigors and epilepsy have this feature in common—that both the one and the other may result from a blood-infection (toxæmia), or from a purely reflex nervous influence. In the previous chapter I have referred to a number of facts which point to the conclusion that the immediate cause of epileptic convulsions is sudden and extreme anæmia of some part of the brain. Now, a careful consideration of the phenomena of rigors renders it probable that they result from *anæmia of the spinal cord.* The

[1] *Diseases of the Liver, New Sydenham Society Translation*, vol. ii. p. 518.
[2] *Stomach and Renal Diseases*, 5th ed., p. 322, *note*.

evidence upon this point is not demonstrative, but it amounts to a high degree of probability. There is an *à priori* probability that convulsions and rigors—distinct as they commonly are, yet sometimes associated in the same case—result from an identical or a similar condition of the nervous system; but that different portions of the nervous centres are concerned in the production of each. In epilepsy, it is certain that the encephalon is mainly affected; and loss of consciousness is one of the most constant phenomena of the disease. On the other hand, during the severest rigors, the cerebral functions are usually quite unimpaired. Then, in the case of rigors, there is positive evidence of the implication of the spinal cord in the fact that the sense of coldness is very commonly referred to the spine, which is often also the seat of severe pain. The severe backache which is associated with the rigors during the initiatory fever of small-pox is a familiar symptom; and a similar spinal pain frequently accompanies the rigors which mark the onset of other febrile diseases. Not long since, I attended a young lady with what proved to be only a catarrh; but the attack commenced with rigors and backache of so severe a character, that I feared lest the disease might be small-pox.

A superficial view of the subject might suggest the erroneous conclusion that rigors result from an interruption of the function of respiration and a diminished circulation of blood through the pulmonary and systemic arteries. The phenomena of an ague-fit, apparently, are consistent with this theory. During the cold stage of ague there is undoubted evidence that the functions of respiration and circulation are both impeded; yet that the respiratory functions are not necessarily implicated in the phenomena of ague is proved by a curious case referred to by Sir Thomas Watson, in which a pregnant woman 'had tertian ague, which attacked her, of course, every other day; but on the alternate days, when she was well and free, she felt the child shake, so that they both had tertian ague, only their paroxysms happened on alternate days.'[1] Now it is certain that, in the causation of intra-uterine rigors, an impeded respiration could have had no share.

[1] Watson's *Lectures*, 5th ed., vol i. p. 765.

Let us, for a moment, compare the cold stage of ague with the collapse stage of cholera. On a hasty view, the two pathological conditions appear strikingly similar; but in some most important particulars they are extremely unlike, and even opposite to each other.

In the cold stage of an ague-fit, and in the collapse stage of cholera, there is evidence of impeded pulmonary circulation and of embarrassed breathing. In these respects the two diseases resemble each other; but now let us consider the differences between these morbid states. A patient in the collapse of cholera complains of burning heat in the epigastrium, and tosses away the bedclothes; yet the thermometer shows that the temperature in the axilla is several degrees lower than normal. On the other hand, in the so-called cold stage of ague, the thermometer indicates a considerable elevation of temperature; yet the patient, shivering with a sense of cold, begs for additional clothing and warmth.

And now observe what happens to the cholera patient after the injection of a hot saline liquid into his veins. In Chapter VI. p. 128, I have endeavoured to prove that the hot liquid overcomes the spasm of the minute pulmonary arteries, thus allowing a free passage of blood through the lungs, and then through the systemic arteries. At any rate, the result is, that the symptoms of collapse rapidly pass away; the pulse becomes full, and the temperature normal, or even higher than normal. But now it is most remarkable that, in a large proportion of cases, coincident with this greater freedom of the circulation and with this rise of temperature, the patient begins to feel cold, and has severe rigors. Upon this point, Dr. Mackintosh, who, in the year 1832, injected the veins of 156 patients, says that 'rigors, severe rigors, almost invariably followed the saline injection. They generally commenced a few minutes after the completion of the operation, sometimes during its performance.'[1] The same phenomena have been observed by others. Thus Mr. Little, who injected twenty patients in 1866, states that rigors occurred in the most promising cases; and that all those who recovered had one or more severe rigors, during which 'the teeth chattered, the bed shook, and the

[1] Mackintosh's *Practice of Physic*, vol. i. p. 369.

patient complained bitterly of cold. At the same time, the temperature rose several degrees in the axilla; and, but for the cold, the patient expressed himself as infinitely better, and was so.' [1]

What, then, is the explanation of this very remarkable phenomenon? The most probable theory appears to be this. During the collapse stage of cholera, a large amount of morbid blood accumulates in the right side of the heart and in the venous system, while comparatively little blood is circulating through the systemic arteries; hence arise the thirst and the dyspnœa—a cry from the tissues for liquid and oxygen. When the pulmonary obstruction has been overcome by the hot venous injection, an abundance of morbid blood is suddenly conveyed through the systemic arteries; and, reaching the spinal cord, this contaminated blood excites contraction of its arterioles; hence spinal anæmia, a sense of cold, and rigors.

It is manifest, from a consideration of these facts, that rigors do not depend upon a partial arrest of the pulmonary circulation, or upon any impediment to the respiratory changes. For, in the collapse of cholera, rigors do not occur until this arrest and impediment have been suddenly removed; and it is, in a high degree, probable that rigors depend upon an anæmic condition of the spinal cord. The sense of heat in a tissue is closely connected with the amount of blood in its vessels. The sensation of burning in the epigastrium experienced by a cholera patient is probably dependent on the large afflux of blood to the stomach and bowels; and, on the other hand, during the occurrence of rigors, it is probable that the anæmic spinal cord conveys to the sensorium the impression that the whole body is cold, when, in reality, it is several degrees above the normal temperature. During the so-called 'cold stage' of ague, the temperature of the body is usually as much increased as it is during the subsequent hot stage; but the brain, receiving through the anæmic spinal cord its information as to the state of the tissues, appears to be deceived by the abnormal condition of the conducting medium. Then, during the succeeding hot stage, the spinal arterioles

[1] *Medical Times and Gazette*, April 1867, p. 355.

relax; a fuller supply of blood reaches the cord at the same time that it reaches the skin and the other tissues; and a sensation of heat is conveyed upwards to the cerebrum. The sensation of heat and of cold in various morbid states of the system apparently depends, not upon the actual temperature of the body, but upon the condition of those parts of the nervous system through which impressions reach the sensorium.

This, then, is the theory which I submit for your consideration: that, as epilepsy is a result of anæmia of the brain, so rigors result from anæmia of the spinal cord; that the immediate cause of the anæmia of the nervous centres is the contraction of the minute arteries; and, further, that this arterial contraction in both classes of cases—namely, in cases of epilepsy and of rigors—may be a result of blood poisoning (toxæmia), or of a purely reflex nervous influence. If, then, this theory be well founded, rigors might be designated *spinal epilepsy*.

This doctrine, if true, has important practical applications, with reference especially to prognosis and treatment. The purely nervous rigors are usually of far less serious import than those which are of toxæmic origin; and they require a different method of treatment. In the nervous cases, narcotics and anæsthetics are of great value; and additional evidence of the analogy between convulsions and rigors is afforded by the fact that both the one and the other may surely be arrested by chloroform, which is especially useful in cutting short the distressing symptoms of reflex nervous rigors.

On the other hand, in cases of toxæmic rigors, we have to study the natural method of cure, and to assist that if possible. The vomiting and purging which often precede or accompany toxæmic epilepsy, as well as toxæmic rigors, are probably the result of an effort to expel from the circulation the noxious materials which are exciting the arterial contraction. These symptoms, therefore, often afford valuable indications for treatment, and a prompt resort to evacuant remedies, such as emetics, purgatives, and diaphoretics, may sometimes cut short an attack which might have resulted in grave disease.

Shortly after the publication of the preceding lecture, Dr. Burney Yeo contributed to the *British Medical Journal*[1] an interesting paper, which I here reproduce:—

'THE PATHOLOGY OF RIGORS.

'Illustrated by a Case of Hemiplegia from Dislocation of the Atlas preceded and accompanied by constant Shivering and Sense of Coldness.

'The following case, extracted from the second volume of Cruveilhier's *Pathological Anatomy*, fell under my observation after reading Dr. George Johnson's lecture in the *British Medical Journal*, on the "Pathology of Rigors." As it appeared to me to afford very remarkable and, of course, entirely undesigned support to the explanation therein given of their phenomena, I called Dr. Johnson's attention to the case; and he agreed with me that it was of so singular a nature as to deserve translation.

'Femme La Croix, æt. 68, came under notice on October 13th, 1832, with hemiplegia of the right side: paralysis both of sensation and of motion. The hemiplegia came on suddenly, while she was in the hospital of La Charité, into which she had been admitted six months before for *severe shivering, which never left her* ("un grand frisson qui ne la quittait jamais"). Warm baths were prescribed for her; and it was after one of these baths that she became paralysed. One curious point is that at first she was uniformly paralysed, or rather enfeebled, on both sides, and it was only later that the hemiplegia came on. She stated that ever since the commencement of her illness she had felt frozen ("glacée"). The hottest applications could not make her warm. The cold "penetrated even to the marrow of her bones." The surface of the body was cold to the touch, like "touching a cold-blooded animal." She complained of severe pain behind the right ear, and suffered much from painful cramps of the lower limbs. She remained in this condition until January 30th, 1833, when she died.

'During all this time the coldness was constant. Cruveilhier observes: "La constance de l'algidité était toujours pour moi un nouveau sujet d'admiration."

[1] July 4, 1868.

'The *post-mortem* examination showed that the *raison anatomique* of the hemiplegia and other symptoms was a displacement of the atlas, producing compression of the cord immediately below the medulla oblongata. This compression had borne more on the right than on the left half of the cord, and had led ultimately to its disorganisation. In the right half the *débris* of the cord was grey, without a vestige of white substance. In the left, the *débris* contained still a little white substance. The annular ligament of the odontoid process was large and yellowish, with little chalky plates in its substance, much like the alteration undergone by the coats of arteries. The odontoid or check ligaments were much weakened, and their fibrous bundles separated by a reddish substance. They permitted the odontoid process to play backwards and forwards so as to produce an incomplete luxation.

'Cruveilhier observes that the displacement was due to disease of the ligaments uniting the odontoid process to the occipital bone and the atlas, probably of rheumatic origin.

'There is no evidence whether in this case, at the onset of the shivering, the sense of cold was simply subjective, as we know it to be in ague and in many other cases. It may have been so, although at the time when the patient came under Cruveilhier's observation, six months after the first appearance of these symptoms, and when she had become hemiplegic, the surface was unusually cold.

'There can be little doubt, when one takes into consideration the cause of the displacement in this case, that it must have been gradual; and the primary effect of such gradual displacement would be an interference with the vascular supply to the cord, either by direct pressure on the spinal arteries or by pressure and stretching of the vertebral arteries, which this partial dislocation of the atlas must of necessity produce. In either case, there would be a corresponding anæmia of the cord.

'This case, then, appears to offer something like a demonstrative proof that there exists a very close connection between the phenomena of rigors and an anæmic condition of the spinal cord. The singular character of the phenomena

presented by this case did not escape Cruveilhier's observation; but the only kind of explanation he attempts is vague and unsatisfactory in the extreme. He says: "The coldness so manifest and so considerable during the progress of the illness lends support to the opinion of those physiologists who consider the spinal cord as the principal centre of animal heat."'

It will be seen that the phenomena in this remarkable case differ from those observed in ordinary cases of rigors, in so far as there was not only a subjective sensation of chilliness, but the whole body was actually cold. It is to be regretted that no thermometric observations were recorded.

The case appears to afford support to the doctrine held by modern physiologists, that under the influence of the nervous system, the metabolic changes in the muscles or other organs, and, as a result of these, the production of heat, may be either increased or diminished.[1]

Dr. Bright [2] has recorded the case of a man, aged 20, in whom paralysis of all the extremities resulted from pressure on the spinal cord by an enlarged odontoid process. He was admitted into Guy's Hospital on August 18th, six weeks after the commencement of the symptoms, and he remained under observation until he died on February 8th; the immediate cause of death being an attack of erysipelas.

'Very few particulars of the case are given, but under the date of January 28th there is the following note: "He had several cold chills on that day, which, as *he had not unfrequently complained of the same feeling*, excited no particular attention; he also complained of constriction at his chest, and a difficulty of breathing. On the night of the 29th he was attacked with erysipelas, after experiencing some chills: this affection quickly extended over his face and scalp."'

No doubt the chills which immediately preceded the erysipelas were a part of that malady, but the cold chills of which 'he had not unfrequently complained' before (how frequently and for what length of time is not stated) may

[1] See Dr. M. Foster's *Physiology*, 3rd ed., p. 431.

[2] *Reports of Medical Cases*, vol. ii. pt. ii. p. 417.

probably have resulted from the pressure of the enlarged odontoid process on the upper part of the cord.

Without attaching undue importance to these two cases, their record may serve to direct particular attention to the symptoms, and especially to the temperature, in future cases of a similar kind.

Dr. Todd [1] has recorded a case (Case lxiii., Marianne Catlin) of enlargement of the odontoid process causing death by pressure on the cord. In the history of that case no mention is made of rigors, which probably, therefore, were absent.

[1] *Clinical Lectures on Paralysis, &c.*, p. 321.

CHAPTER XI.

A LECTURE ON HYSTERIA.

Various Forms—Hysterical Paroxysm—Distinction between Hysteria and Epilepsy—Epileptiform Hysteria—Exciting Causes—Globus Hystericus sometimes occurs in Men—Case—Mock Diseases—Peritonitis—Spinal Irritation—Pain in the Feet—Hysterical Breast—Paralysis—Durable Contraction of Limbs—Affection of Special Senses—Extreme Neurasthenia and Emaciation—Aphonia—Spasm of Larynx—Loud Cough—Dysphagia—Vomiting—Hysterical Joints—Feigned and artificially acquired Diseases—Hæmoptysis—Cases—Hæmatemesis—Cases—Retention and Suppression of Urine—Hysterical Ischuria—Artificial Skin Eruptions and Bleeding Ulcers—A Hair-pin and a Calculus in the Bladder—Artificial Gastric Fistula—A Finger destroyed by Caustic—Two Cases of Hysteria in Young Men—Treatment of Paroxysm—Compression of Ovary—Treatment in the Intervals—Remedies for Special Symptoms—Cases of Extreme Emaciation and Exhaustion successfully treated by Dr. Weir Mitchell and Dr. William Playfair.

A GREAT variety of forms of disease and nervous derangement are commonly, although perhaps with doubtful propriety, included under the term *hysteria*.

There is—1, the hysterical fit or paroxysm; 2, various neuralgic, spasmodic, and paralytic affections which have some resemblance to other and more serious forms of disease; 3, a morbid state of mind which leads the unhappy patient to *feign* a variety of diseases. I will presently relate to you some remarkable examples of this painful class of cases.

The word *hysteria* is suggestive of a disease having its origin in uterine disease or disorder; but the disease, though infinitely more common in females than in males, is not confined to them. And even in females there is no necessary, though a frequent, connection between hysteria and uterine or ovarian disease or derangement.

The *hysterical paroxysm* is of the convulsive character. Sometimes it is preceded by outward manifestations of grief

and mental disturbance. The patient has been observed to shed tears, or she has been unusually depressed, or excited, and irritable. In other instances, after perhaps a violent effort to repress some strong emotion, the fit comes on quite suddenly. The patient falls on the floor, or throws herself on the bed, and the trunk and limbs are then drawn into strong convulsions. These convulsions pull and twist the body into every possible position. Sometimes the head and trunk are bent backwards into an arch (opisthotonos); then the patient rises into a sitting position, and the body is bent forwards on the knees. Then she will twist round, and writhe like an eel; very commonly the hands clutch at the throat, which is usually full and swollen; sometimes they violently tear out the hair.

The jaws are often tightly closed, the face is hot and flushed from exertion, but there is none of the hideous distortion which is so constant during the epileptic paroxysm. The eyes are usually closed, but if the lid be raised the pupil is seen to be natural. The heart beats violently, and the pulse throbs. The breathing is deep, labouring, and irregular.

During the paroxysm consciousness is rarely, if ever entirely lost—though the patient often *seems* to take no heed of what is going on, and makes no reply when spoken to. The jaws being clenched, she cannot be made to swallow food or medicine. Sometimes there is constant moaning, or there are frequent loud screams and exclamations; at other times, there is wild and incoherent talking.

The paroxysm lasts for a very variable period—from a few minutes to many hours—with occasional remissions and periods of repose in some cases. The attack often terminates with an explosion of sobs and laughter, a copious discharge of tears and of pale limpid urine.

Occasionally, during the paroxysm, there is enormous development of *flatus* in the intestines; the abdomen becomes much distended on one side perhaps, in consequence of an accumulation of air in a limited portion of intestine; then suddenly the distension subsides, as the air is diffused through other parts of the bowel, with a loud rumbling sound.

When the paroxysm is over, the patient is left exhausted,

and she will sometimes sleep for many hours. The muscles, for some days, remain sore and tender from the effects of the straining and violent exertion.

A paroxysm of this kind having once occurred, the patient is liable to a relapse if, within a short period, she be exposed to any repetition of the exciting cause. And by recurring again and again the paroxysm may become habitual and return on the slightest provocation.

As a general rule, the distinction between hysteria and epilepsy is attended with no difficulty, but the two diseases may be combined in various ways. Thus hysteria may be grafted upon epilepsy. I have often seen an epileptic patient become hysterical on recovering consciousness after an epileptic seizure. The mental distress occasioned by the knowledge that he or she has been revisited by the terrible malady has sometimes been the apparent exciting cause of the hysteria. It very rarely happens that epilepsy occurs in a previously hysterical patient, the two diseases having a distinct history. But there is a class of cases to which the term *epileptiform hysteria* has been applied. There are cases in which, although the disease is essentially hysteria, yet it presents some epileptiform features, as fully described by Charcot. The attack perhaps begins with a sudden shriek, pallor, loss of consciousness, a fall and distortion of the features, then tonic rigidity, foaming at the mouth, the foam being sometimes blood-tinged from bitten lips or tongue—with these there is coma and stertorous breathing for a longer or shorter time. After this all the symptoms are hysterical—violent contortions and gesticulations, and paroxysmal delirium, the attack ending with sobs, tears, and laughter.

Notwithstanding the epileptiform mode of onset, Charcot maintains that the disease is 'only the highest degree of development of common hysteria.' One point insisted on by Charcot is that even the most intense epileptiform attack may be modified, and sometimes arrested, by compression of the tender ovary, which has no influence upon a true epileptic seizure. This, therefore, is an important means of diagnosis as well as of treatment.

Hysteria is much more common in females than in males.

It is oftener met with in single than in married women; and in married women it is more common in those who are barren than in those who have borne children. It is also more common between the age of puberty and what is called 'the change of life'—that is, between the ages of 14 and 45, than at earlier or later periods of life. It may occur in the robust and plethoric, but it is more frequent amongst those who are feeble and anæmic, and especially in those of nervous and excitable temperament.

The exciting causes of the paroxysm are very various. The hysterical fit may be excited by severe pain—for instance, the pain of dysmenorrhœa, or of parturition; for, although more common in those who have not borne children, parturient women are not free from its attacks; in other cases the agonising pain caused by the passing of a biliary or a renal calculus has been the exciting cause of a paroxysm.

In by far the greater number of cases the exciting cause is some *violent emotional influence*—a shock of terror, or a sudden overwhelming sorrow, or continued grief or anxiety or disappointment; and be it remembered that emotional influences of this kind are often the more powerful in their action in proportion as they are *concealed* and *suppressed*.

In every case of hysteria the condition of the various organs and functions should be investigated, more especially the catamenial function and other symptoms referable to the uterus and ovaries; and careful inquiry should be made of the patient and her friends for the existence of any emotional influences which may have tended to excite the disease.

The paroxysm, or fit, forms but a small part of the history of hysteria.

One of the most frequent and characteristic symptoms of hysteria is a peculiar sensation as of a *ball* rising into the throat and there causing a sense of choking or suffocation. This is the *globus hystericus*.

It is apt to be induced by any emotional disturbance, and commonly precedes the fully developed hysterical fit. It often distresses and alarms the patient at night, coming on as soon as the head is placed on the pillow, or awaking her with a sense of suffocation. Some time since I attended a lady of

nervous temperament, who told me that for three or four nights before I saw her, she had by a great effort kept herself awake, to avoid the dreadful choking sensation which came upon her as soon as she fell asleep.

I have often observed this symptom in *men*. One of the most remarkable examples of it occurred in a man 60 years of age, who came to me, some years ago, at the dispensary and said that, for nearly a year, he had scarcely passed a night without being disturbed by a frightful sense of suffocation, for the relief of which his son and daughter had to assist him to the open window. He had no cough or other symptom of disease within the chest. His countenance indicated great anxiety, and he spoke of having been much troubled by circumstances the nature of which he did not communicate. I gave him pil. saponis co. gr. v. om. n. He came again at the end of a week and told me that since taking the pills he had slept well and had not once been disturbed by the choking sensation which had so long distressed him. I have found in other cases of the same kind (*i.e. globus* occurring at night and during sleep) that an opiate, given for a few nights, has been very efficacious.

Hysteria may assume the form of a variety of serious diseases, and the diagnosis of these cases is sometimes extremely difficult. I will now refer to some of these *mock diseases*. *Peritonitis* is one of these simulated maladies. There is extreme pain and tenderness over the abdomen, the pain being much increased by motion as well as by pressure. In forming a diagnosis of these cases you must look to the entire history of the patient. You will often find that the tenderness is felt more on light than on firm pressure; the patient even cries out before she is touched, if she sees that you are about to touch her. On the other hand, when her attention is diverted she bears pressure without flinching. Usually there is no fever or vomiting, and never the anxious expression of countenance which is almost constant in cases of actual peritonitis.

This abdominal pain is often seated in the muscles. It may be excited by any unusual exertion; thus it may follow an hysterical fit—having its seat in the muscles which have been strained and injured during the convulsive seizure; or it may

come on after a feeble, bed-ridden patient has been sitting up for some hours. The weak and over-strained muscles become fatigued and painful. You may form an idea of this kind of pain from your recollection of the pain and soreness following your first game of cricket or football, or a long pull in a boat, or any other unaccustomed muscular exertion.

It is remarkable that the *skin* over the strained and painful muscles sometimes becomes exquisitely tender, so that even the contact of the clothes excites severe pain.

I am indebted to my friend and former fellow-student, the late Dr. Inman, for having first directed my attention to the subject of muscular pains. His work on *Myalgia* contains many original and most valuable observations on this subject.

Sometimes the abdominal muscles become cramped and drawn up into hard masses, which may be mistaken for *tumours*. One or more divisions of the recti muscles may be thus thrown into spasm and simulate a tumour. In many instances the pain is most felt at the tendinous insertions of the muscles into the bone. Thus, a pain at the insertion of the recti muscles into the pubes may excite a suspicion of inflammation of the uterus or bladder.

Charcot insists upon the fact that in a large proportion of cases one or other *ovary*, most frequently the left, is the seat of the abdominal pain, and that firm pressure over the tender ovary not seldom tends to excite the hysterical symptoms which, at other times, arise spontaneously.

The pain and tenderness of the spine, of which hysterical patients often complain, has its seat in the muscles and ligaments, which, being weak and relaxed, are fatigued and stretched in supporting the spinal column, and thus become the seat of a severe aching, burning pain, which is often increased by pressure. Cases of this kind have often been fearfully maltreated by leeching and counter-irritation. They are commonly spoken of now as cases of *spinal irritation*; they are really examples of pain in fatigued and over-stretched muscles and ligaments. A similar pain sometimes occurs in the ligaments and tendons of the foot in persons who are not hysterical, but who are flat-footed or in whom the arch of the foot has yielded under the weight of the body.

This pain in the feet may sometimes be relieved by putting a soft but firm cushion beneath the arch, so as to take off a portion of the weight and prevent the further stretching of the ligaments. Or the sole of the boot may be made and kept in an arched form by the insertion of a piece of bent steel between the layers of leather. By this device I once relieved a very heavy and paralysed gentleman from pain in the feet which had caused much suffering.

Hysterical Breast.—The breast is sometimes painful and tender, and sometimes slightly enlarged, but without induration or structural change. It is purely neuralgic.

Another so-called hysterical *pain* is the *clavus hystericus*—a pain above one or other brow, as if a *nail* were driven into the brain. It sometimes comes on every day at the same hour, and in this respect resembles hemicrania or brow ague. A severe pain in the head should always be carefully investigated. It *may* be something more than neuralgia or hysteria. It has sometimes happened that a fixed pain in the head, supposed for a time to be purely neuralgic, has ended in fatal coma, the result of a tumour or an abscess in the brain. In all cases of persistent headache the fundus of the eye should be carefully inspected with the ophthalmoscope.

Certain paralytic affections occasionally occur in hysterical subjects, assuming the form of *hemiplegia* or *paraplegia*; but they often come and go so suddenly that they are evidently not dependent on any structural change in the nervous centres, and they are frequently induced by some sudden emotion of the mind. One lady whom I knew, suddenly became paraplegic on hearing of the death of her daughter, but she soon regained the use of her limbs. The hysterically paralysed muscles, even in cases of long standing, are usually well nourished and retain their electric excitability. In comparatively rare cases there is not only paralysis but a durable *contraction* of one or more limbs, which may assume the form of hemiplegia or of paraplegia.

Charcot has clearly indicated the differences between hysterical hemiplegia with contraction and hemiplegia the result of central lesion of the brain. In the hysterical cases there is no facial palsy; but there is often complete hemi-

anæsthesia on the affected side, extending over the entire half of the body, and involving not only the skin, but the mucous membrane and muscles, and perhaps even the bones. The anæsthetic side is usually paler and colder than its opposite. There is a state of ischæmia, so that little blood flows from a prick or a leech-bite. The organs of special sense are often to some extent affected on the same side. Taste and smell on that side are lessened or lost, and the vision of one eye may be more or less impaired without discoverable change of structure within the eye. This combination of symptoms is rarely observed in hemiplegia of cerebral origin or in cases of spinal hemiplegia from a unilateral affection of the spinal cord. The anæsthesia, as Dr. Brown-Séquard has shown, is on the side opposite to that affected by the motor paralysis. Then, whereas the contraction which results from a cerebral lesion is always developed slowly, the hysterical contraction often comes on suddenly after a fit. Sometimes, even after a long continuance, it passes away as suddenly as it came, and that, too, perhaps under the influence of some powerful emotional excitement, such as, in other cases, has been the exciting cause of the contraction. In the lower extremity, the contraction sometimes assumes the form of club-foot (*talipes varus*). I once rescued a case of this kind from an orthopædist who wished to divide the tendo Achillis. The contraction had come on, with other hysterical symptoms, in a highly sensitive young lady, as a result of intense mental anxiety, and it was soon removed by a combination of sedatives and tonics. To divide the tendo Achillis in such a case would be little short of a crime.

In some not rare cases, without any special paralysis or contraction of the limbs, the power of walking is gradually lost. A delicate girl complains of weakness, backache, and inability to walk; she is allowed or perhaps advised to remain in bed. And there she continues to lie for months and even for years. At length, perhaps, some sagacious doctor discovers that the only impediment to her walking is the want of the will to do so. He resolutely determines that she shall walk; she violently protests, but the attempt is made and is successful. From that time she continues to walk about, as she might have done during the whole period of her confine-

ment. In some cases the power of walking has been suddenly recovered under the influence of some powerful emotion. Some reported miraculous cures of paralytics by pilgrimage to a religious shrine have without doubt been cases of this class. What is required to effect a cure is some impulse powerful enough to call into exercise the nervous and muscular force, which, although it has long been dormant, still exists.

Cases, however, not seldom occur in which there is something more than the want of the will to walk. In consequence of inability to take or digest or retain food, the patient is reduced to an extreme degree of emaciation, with nervous and muscular exhaustion, so that the slightest movement is attended with great pain, and walking is an impossibility. These distressing cases, after resisting for years all other methods of treatment, have, in many instances, been rapidly and completely cured by the combination of remedial measures of which Dr. Weir Mitchell, of Philadelphia, was the originator, and which has been very successfully followed up in this country by Dr. William Playfair. I shall presently refer again to this method of treatment.

Aphonia.—Loss of voice is another result of a local hysterical paralysis. The aphonia may come on suddenly as a result of fright or fatigue, or it may supervene upon an ordinary cold. The larynx, inspected by the mirror, is seen to be quite healthy, but when the patient is told to vocalise, the cords remain more or less wide apart. The patient can usually speak in an audible whisper, but in some cases even the whisper is abolished. The voice is often recovered as suddenly as it was lost. A sudden violent emotion, whether of fear or of joy, may elicit a loud cry, and the voice is at once restored. In most of these cases a cure is instantaneously effected by a shock of electricity applied to the interior of the larynx by means of Dr. Morell Mackenzie's laryngeal galvaniser; the patient utters a scream and the voice is recovered.

Spasm of the Larynx.—The larynx is sometimes *spasmodically* affected. The breathing is noisy (stridulous), and there is a mimicry of laryngitis.

Sir Thomas Watson mentions one case of this kind which he saw with Sir Charles Bell at the Middlesex. They discovered

its true nature, but the girl had twice before had tracheotomy performed for similar attacks, and there were the scars of the operations on her neck.

Some years ago I saw such a case in the hospital with Dr. Todd and Sir William Fergusson. There was loud stridulous and hurried breathing, but no lividity, and no distress in her countenance. I suggested to try the effect of chloroform. The breathing at once became quiet and entirely free from stridor, proving that the obstruction had been due to spasm alone.

A similar case was under my care in the hospital in March 1870, and in this case we had the laryngoscope to aid us in the diagnosis.[1]

Another symptom referable to the larynx and the respiratory muscles is a peculiar *loud barking cough*. Sometimes it is a single cough occurring at wide intervals; sometimes it is nearly incessant, or occurs in paroxysms. It appears to be almost entirely a *voluntary* cough, and can be restrained by a strong effort of the will.

A very loud cough is more likely to be the result of spasm than of organic disease in the larynx. A tumour or an inflammatory swelling of the mucous membrane within the larynx, by pressing on the vocal cords, impedes their vibrations and so renders a loud cough impossible. The only cough resulting from organic disease which at all resembles this hysterical cough is the frequent loud ringing cough which is excited by the pressure of an aneurysm upon the trachea.[2]

Dysphagia, the result of spasmodic constriction of the œsophagus or irregular action of its muscles, sometimes occurs; and a loud discordant *hiccup*, dependent on hysterical spasm of the diaphragm, is another symptom occasionally met with.

Vomiting is a symptom of common occurrence in these cases, and when it happens frequently after food it is injurious by depriving the system of nutriment.

Hysterical affections of the joints are common. Pain and tenderness are complained of in the knee or hip: the joint is kept immovably fixed in one position, at first apparently by a

[1] The particulars of this case are given in a subsequent chapter on *Spasm of the Larynx*.

[2] See the chapter on *Thoracic Aneurysm*.

voluntary effort, but gradually the muscles become automatically contracted, so that the limb cannot be moved without exciting great pain. Sir B. Brodie was one of the first to direct attention to cases of this kind, and he declared that a few years ago, at least four-fifths of the female patients in the higher classes of society who were supposed to labour under diseases of the joints were simply hysterical. These cases are now much more generally understood than they were when Sir B. Brodie made this statement, so that a wrong diagnosis is less probable, and would be more discreditable in the present day.

About the symptoms to which I have hitherto referred, there is, at any rate, a sort of half-reality; they are not entirely imaginary or fictitious, and they are often attended with much suffering. But there are cases of what is usually called hysteria in which the symptoms are *feigned or induced* with a deliberate intent to deceive. In some of these painful cases it is impossible to assign any rational motive for the strange conduct of the patient. A desire for sympathy is doubtless the original motive in some instances.[1] A girl who has thought herself neglected or uncared for finds that she becomes an object of great interest and attention when suffering from what appears to be a painful or an unusual form of disease. She begins by feigning some symptoms; one act of deception almost of necessity leads to others, in order to prevent detection. Having told one lie, she is compelled 'to endorse it with another;' and so by degrees all sense of shame and of truth is lost, and the whole moral character becomes deteriorated. Thus it sometimes happens that the hysterical girl, well born and well educated, and originally perhaps unselfish and amiable, may become so degraded as to derive a strange and morbid satisfaction from witnessing the anxiety and distress of her friends; and, though previously truthful, she seems actually to enjoy the idea of deceiving them. Cases of this kind are closely allied to insanity; and in fact hysteria and insanity sometimes merge into each other by almost imperceptible gradations. I purpose now to refer to some of these feigned or self-inflicted diseases.

[1] See an interesting treatise on the *Pathology and Treatment of Hysteria*, by Mr. Brudenell Carter.

Spitting of blood is a common symptom in hysterical patients.

When this symptom occurs, the mouth and throat should be carefully examined to ascertain the source of the bleeding. It will sometimes be found that the bleeding is from a *leech-bite* in the interior of the mouth, or that the gums have been cut or pricked. Some years since I had in the hospital a good example of artificially induced hæmoptysis.

A girl was sent up from the country by her mistress, who had taken a great interest in her, and who sent with her an elaborate daily report of the symptoms, which for minuteness of detail would have done credit to the most industrious clinical clerk. The chief symptoms were cough or hæmoptysis. The cough was of the characteristic loud barking, spasmodic kind. The blood was florid and fluid, as if mixed with a considerable amount of saliva; it was not mixed with mucus or puriform matter, and it was noticed that the blood was not brought up by an effort of coughing, but was simply spat out of the mouth. There was no physical sign of disease within the chest. The girl had a florid face, with an hysterical manner and physiognomy, and the catamenia were said to be irregular. From the first we suspected a trick in the matter of the hæmoptysis, and the nurse was directed to watch her. After some days I examined the interior of the mouth with direct sunlight, and I saw at least a dozen fine cuts or scratches in the mucous membrane over the hard palate. Some had been quite recently made, while others were nearly healed. She strenuously denied that she had cut or scratched her mouth; but from that day there was no return of hæmoptysis, and after she left the hospital I heard that she confessed that she had been in the habit of scratching her mouth with a needle. My friend the late Dr. Russell, of Birmingham, told me of a curious source of hæmoptysis which had been discovered by one of his clinical clerks. He ascertained that the patient's tongue was continually covered with blood, and that she was constantly sucking at a carious tooth. The pulp of the tooth was exposed and vascular, and out of this she contrived by suction to draw a continual supply of blood.

Hæmatemesis.—Vomiting of blood is another symptom

which occasionally occurs in hysterical women. This may be a genuine symptom vicarious of suppressed catamenia. It may also be a result of swallowing and then vomiting the blood of some animal.

I had one patient, not long since, in the hospital who made a poor attempt to deceive us by pretending to have vomited a fluid which we found to be milk coloured with compound tincture of lavender.

In December 1867, a girl, E. P., aged 21, was admitted into the hospital on account of vomiting of blood, which had occurred, she said, almost daily for about four years. She complained of pain in the stomach. Her cheeks were florid. She occasionally, while in Twining ward, brought up some blood, usually during the night. I suspected fraud, examined her mouth and throat carefully, and directed her to be watched; but we could not discover the trick. She went out on December 12th, and stayed with a relation at Fulham. On December 23rd she was re-admitted with a severe attack of diphtheria, from which she died on January 2nd, 1868.[1] Before she died she confessed to the Sister that her blood-vomiting was a deception; but she did not say how she had practised it, and the Sister, seeing how ill she was, was unwilling to question her as to the particulars.

Retention and *suppression* of urine are amongst the commonest of hysterical complaints. As to *retention*, the urine will usually be passed readily enough if the patient be left to herself.

In some cases, however, the bladder becomes distended and paralysed by a rapid secretion of pale watery urine, which has but little stimulant action upon the bladder; and in such a case it may be necessary to use the catheter.

Suppression of urine is occasionally feigned; and sometimes, by way of adding to the interest and variety of the case, the patient swallows her urine secretly, and then vomits it openly, to show that the stomach has taken upon itself to do the work of the kidney.

Some time since a friend came to me in a state of a great anxiety about one of his patients, a young lady, who was sup-

[1] Hospital Case Book, vol. xxix. p. 226.

posed to have had almost complete suppression of urine for eight days; and this alarming symptom had followed upon her taking a dose of oil of turpentine as a vermifuge. He had introduced the catheter two or three times, and had drawn off only a few drachms of urine. The urine so obtained was quite normal; it contained neither blood nor albumen. The turpentine, therefore, had not set up nephritis, and there was no pain or other evidence of irritation of the urinary organs.

She was lying in bed, eating and drinking as usual, and had no symptom of illness. This was a very clear case. I saw her, and directed that she should be closely watched; and, that being done, the bladder soon became distended with urine.

A case of this kind may quickly be brought to the test. Let the patient be watched incessantly by a trustworthy attendant, and let her swallow copious draughts of tea or any other simple drink. If then the bladder does not become distended in the course of twelve hours, you may conclude that the patient's *kidneys*, and not her *morals*, are at fault

Charcot, discussing the subject of hysterical ischuria, states that until recently he, in common with most physicians, had regarded *all* reported cases as the result of fraud; but he gives full particulars of one patient who was reduced to a very helpless condition by contraction of all four limbs, and who, in addition, was so closely watched by trustworthy attendants that deception seems to have been rendered impossible. It would appear that, for many weeks together, the renal secretion was reduced to a minimum; while on some occasions there was absolute anuria for several days, but never beyond eleven consecutive days. During the same period there was copious and frequent vomiting of a liquid containing urea. The blood drawn from a vein contained no excess of urea, and the patient presented no uræmic symptoms. Charcot suggests that during the continuance of the ischuria there may have been a decreased disintegration of tissue and a corresponding diminution of excrementitious matter. He, however, confesses his inability to explain phenomena the reality of which he is compelled to admit. It is well, therefore, to bear in mind the possible occurrence of such an extremely rare and exceptional case.

The tricks of hysterical patients are almost endless. *Earth-worms* will be brought as having been passed from the bowels, *pebbles* as having come from the bladder.

Some years since a woman in King's College Hospital excited great interest and curiosity by *an eruption of pemphigus* running into large blisters, which appeared on one or other arm at each catamenial period. More than once she was admitted under the care of Dr. Budd, and there was much speculation as to the probable explanation of this unusual phenomenon. The mystery was at length explained by the discovery of a blistering plaster on her arm.

My friend (Mr. Henry Lee) told me of a female patient of his who had a sore on her leg that bled at every monthly period. In order to prevent any artificial excitement of the bleeding, a piece of sheet lead was bandaged over the sore. At the next period it was found that bleeding had occurred beneath the lead, but it was also found that the metal had pinhole perforations through its substance.

A woman one day brought her daughter to me at the hospital in consequence of the sudden and alarming appearance of a strange blackness round the girl's eyes. The girl was most unwilling to face the light, but I saw at once that this example of what we may call *hysterical meladerma* was the result of smearing the skin with soot and grease. I took a wet towel and wiped away the blackness. The girl then rolled on the floor, and had a violent hysterical fit.

My friend (Dr. Waggett) told me lately (November 1867) that he had met with a precisely similar case in the daughter of a professional friend of his own.

These patients sometimes inflict upon themselves a terrible amount of suffering. A girl once came under my care in the hospital with great pain and irritability of the bladder, the urine containing blood and pus. Her sufferings were extreme, and had been for many months. I requested Sir William Fergusson to examine the bladder. He found a stone, and on further examination discovered the two points of a hair-pin projecting through the posterior wall of the bladder into the vagina. Sir William crushed the stone, and extracted it with the hair-pin through the urethra. The girl, of course, was

asked how the hair-pin got into the bladder. At first she denied all knowledge of it, but, after a time, remembered that she had once accidentally *sat down upon a hair-pin !*

In the forty-first volume of the *Medico-Chir. Transactions* Dr. Murchison has recorded the case of a young Scotchwoman who, for two years, kept up an artificial swelling of the left arm (a factitious elephantiasis) by secretly tying a garter round the limb at the insertion of the deltoid muscle. She proposed to have the arm amputated at the shoulder-joint, and was admitted into the Aberdeen Infirmary, where Dr. Keith detected the trick, and cured her by restraining both her arms and hands in a strait waistcoat.

Two years after this she fancied or pretended that she had heart disease, and a surgeon unwisely inserted a seton over the epigastrium. When seen again by Dr. Keith, five years after the insertion of the seton, there was a deep, round, fiery ulcer, of the exact shape and size of an old copper penny of the reign of George III., which she had very quickly nipped out with her nail along with the dressings. Dr. Keith warned her that she would make a hole into her stomach. In spite of the warning, she continued to keep up pressure by the coin and a belt, until she had established a large oval communication between the stomach and the skin. When last seen by Dr. Murchison, more than four years after the stomach had been thus opened, the gastro-cutaneous fistula still existed, and she had to keep the hole corked to prevent the escape of the contents of the stomach.

Many years ago I saw, with Sir William Fergusson and Sir James Paget, a young lady who had long suffered from a recurrence of inflammation and sloughing of the forefinger of the right hand. On several occasions the finger was reported to have nearly healed, when a fresh sloughing occurred, and on each occasion the recurrence of the mischief took place during the night. This history corresponded with no known form of natural disease, and when questioned the unhappy girl admitted that she had destroyed her finger by the repeated application of a caustic. And so effectually had she done this that ultimately the finger had to be amputated.

I have before said that some of the most characteristic

hysterical symptoms occasionally occur in *men*; and I mentioned the case of *globus* occurring at night in a man 60 years of age (p. 270). It is more common in younger men.

I have seen two good examples of hysteria in medical students.

I was called up one night to a student whom I found in a paroxysm of hysteria, with globus in his throat, and a dread of instant death. He was of a nervous temperament; he had been working hard, and was anxious about an approaching examination. It appeared that he had been dreaming, and when he awoke he found his heart beating violently; he was alarmed by this, and as he continued to direct his attention to the heart the palpitation increased in violence. With some difficulty he got up and rang the bell for help. I found that he had no symptom of organic disease. I ascertained the cause of his fright, and after some time, though not without considerable difficulty, I succeeded in persuading him that his fears were groundless. He remained unusually nervous for some days after this.

About the same time that this case occurred, one of the students resident in the College, while wrestling with one of his companions, fell and struck his head against the wall. This caused a considerable amount of pain, and for some hours he was in a state of hysterical delirium. He appeared only half-conscious of what was going on around him, and talked an amount of wild nonsense which was more amusing than alarming. The next day he was well.

When I was house-physician of the hospital, I admitted a little shoemaker whom I had known for some time as an excitable man of intermittingly intemperate habits. He had now a sharp attack of gout, and one evening he astonished the ward by setting up a loud and continuous shouting. I was called to him, and found him still roaring at the top of his voice. He went on in spite of my loud remonstrance. So I took him by the shoulders and gave him a vigorous shaking; this was immediately successful, the shouting stopped, and was not repeated. I think it probable that he had been sleeping and dreaming, and that he was only half-awake until aroused by the shaking.

These are some of the examples which I have seen of what

I think may reasonably be called *hysteria in males*. Shakspeare gives us an eminent example of hysteria in the male in the person of King Lear, who exclaims—

> Oh, how this mother swells up toward my heart!
> *Hysterica passio!* Down, thou climbing sorrow,
> Thy element's below!

The treatment naturally divides itself into the remedies to be employed during the paroxysm and the treatment to be pursued in the interval between the paroxysms. Then certain symptoms and complications require special remedies.

Before the paroxysm has become fully developed the symptoms are more or less under the control of the patient's will, and a vigorous application of cold water, or even the threat of it, will often stop it entirely. This remedy should always be promptly applied to any girls who threaten to become hysterical at the sight or sound of another in a fit.

Dr. Russell Reynolds says the attack may be arrested by a plan suggested by Dr. Hare—viz. preventing the patient from breathing for a certain time, by holding the nose and mouth. The effect is to compel the patient, when allowed to do so, to draw a long breath, which is usually followed by relaxation of the spasm and a cessation of the fit.

When the paroxysm is fully established, it probably passes altogether from under the control of the will, and will often take its course almost uninfluenced by treatment.

One main object is to place the patient where she cannot injure herself during her struggles; the best arrangement often is to put her on a bed or mattress upon the floor. Let her dress be loosened, and then leave her as much as possible to herself. Do not allow a number of sympathising women to crowd about her, but clear the room of all but one attendant, and let her watch the course of events. Any attempt to mechanically restrain the movements of her limbs only increases the patient's struggles, and should therefore be forbidden. The paroxysm will, in many cases, soon pass into a quiet slumber, from which the patient will awake with feelings of languor and indisposition, and frequently with a coated tongue. For this an aperient may be given; and an endeavour should then be made to ascertain the cause of the paroxysm.

Various remedies have been employed to cut short the paroxysm—draughts of asafœtida, or ether, or valerian, and enemata of asafœtida or turpentine. I believe that these means are usually quite ineffectual. The convulsions may always be suspended temporarily by chloroform; but they usually return as soon as the effect of the chloroform has passed off. A full dose of opium, or morphine, or chloral will often send the patient to sleep, and so stop the convulsion. If the patient cannot be induced to swallow, the sedative may be given in the form of enema, or morphine may be injected hypodermically.

I have before referred to Charcot's statement that by energetic compression of the ovary the most violent paroxysm of hystero-epilepsy may be cut short. The compression must be continued for two, three, or four minutes, when he declares, 'You are almost certain to find all the phenomena of the seizure disappear as if by magic.' I have had no experience of this method, but the authority of Charcot should ensure for it a careful and extended trial.

In the intervals between the paroxysms a general tonic plan is most suited for these patients. A plain, nourishing diet with a sufficient proportion of animal food is desirable, and with this a moderate allowance of wine or beer, unless, for any reason, alcoholic stimulants are unsuitable. Some of these patients have not only a great distaste for wholesome food, but also a morbid craving for such strange articles of diet as wax-candles, sealing-wax, chalk, cinders, and slate-pencils. It scarcely need be said that, not only should these be forbidden, but they should be carefully kept out of the patient's way.

Daily exercise in the open air should be insisted on, and, when the circulation is sufficiently vigorous to ensure reaction, a cold shower or sponge bath in the morning is of great use. In anæmic cases full doses of iron, with or without quinine and strychnine are beneficial; the bowels being at the same time regulated by aloetic aperients. These patients should avoid hot rooms, late hours, strong emotional excitement, and every kind of mental and bodily dissipation. On the other hand, any occupation which will take them out of themselves and render them useful to others should be encouraged.

The employment of opiates and alcoholic stimulants requires great care and judgment. The temporary relief which they afford is often very great, and on that account an incautious sanction of their use by the medical attendant may lead to their habitual abuse by the patient. The local neuralgias and myalgias may usually be relieved by such anodyne applications as contain belladonna, opium, or aconite; but, as a rule, the less the patient's attention is directed to the seat of her suffering by local applications the better. It is especially important to avoid the uncalled-for use of the catheter or the *speculum vaginæ*. The employment of these instruments often has a demoralising effect upon hysterical women, and therefore, except in cases of absolute necessity, their employment is unjustifiable. On the other hand, if there is reason to suspect displacement or disease of the uterus or ovaries, the necessary means must, of course, be adopted both for the detection and the treatment of the local malady.

Rigid contraction of the limbs or of the jaws (hysterical trismus) may sometimes be overcome by a continuous cold douche, which may require to be repeated if the contraction returns. The rigidity may be overcome by the inhalation of chloroform, and the limb, being then extended, may be kept in position by a well-adapted apparatus.

I have before mentioned that hysterical loss of voice is best treated by Dr. Mackenzie's laryngeal galvaniser. If the patient can thus be made to shout, the natural voice is at once restored.

Lastly, in the treatment of those pitiable cases of extreme emaciation and exhaustion to which I have before referred, the most marvellous results have been obtained by the combined employment of entire seclusion and separation from injurious home associations, careful feeding, massage, and electricity. For the details of this rational method, and for the histories of some of the very striking cures which have thereby been effected, I refer you to two small but most interesting and instructive books, entitled—the one, *Fat and Blood, and how to Make Them*, by Dr. Weir Mitchell; the other, *The Systematic Treatment of Nerve Prostration and Hysteria*, by Dr. W. S. Playfair.

CHAPTER XII.

A PECULIAR FORM OF NERVOUS APNŒA: ITS PHYSIOLOGY AND TREATMENT.[1]

Nervous Palpitation and Dyspnœa while Reading Aloud—Illustrative Case—Physiological Explanation—Means of Prevention—Suspended Breathing during Laryngoscopic Examination and while Sitting to a Photographer.

THERE is a peculiar and distressing form of nervousness about which I have not unfrequently been consulted, and which has still more frequently been brought to my notice in the course of conversation with friends and with patients who have consulted me about other symptoms. In most of the cases that have come under my observation, the nervous affection to which I refer has occurred while the patient has been engaged in reading aloud, with his family, either prayers or a chapter out of the Bible. The reader finds that his heart begins to flutter, while his breathing is oppressed; and his utterance becomes indistinct and broken. With this, there is sometimes more or less of a feeling of giddiness, and a tendency to faintness. Sometimes, by a determined effort of the will, the difficulty and the distress are overcome, and the reader continues and completes the work in hand; but not unfrequently the symptoms become more and more distressing, until the patient, voiceless and panting, is compelled to give in and discontinue the reading. Most of those who have spoken to me on the subject have been men, but I have known the same difficulty occur to women. Several of my patients have been clergymen; and more than one of them, to the distress and alarm of themselves and their congregations, have broken down in the manner described, while engaged in reading the service in church. All who have suffered in this way have been of a

[1] *British Medical Journal*, October 29, 1870.

sensitive and excitable temperament; some have been under the influence of nervous depression or a temporary derangement of health. In some instances, some emotional disturbance has been the immediate exciting cause of the nervous paroxysm; it requires no small amount of resolution and nerve to get through some of the lessons which the clergy have to read to their congregations. In some cases, the recollection of a former attack and a dread of its recurrence have increased the liability to the attacks, and provoked their frequent return. One of the most curious examples of this was afforded by a clerical friend who, one day, had to run to his church in order to avoid being late. When he went into the reading-desk, his breathing being still hurried by the recent exertion, he had some difficulty in getting through the first part of the service. His breathing then gradually became quiet, the difficulty in reading ceased, and for the time he thought nothing more of the occurrence. On the next occasion, when he began to read the same service, he had gone, without hurry, into the desk; but the recollection of his former suffering brought about the same embarrassing train of symptoms—palpitation, difficulty of breathing and of utterance—to such a degree, that for a few minutes he could scarcely continue the service. Again and again, at the commencement of several successive services, the memory of his former distress and a nervous apprehension of its recurrence brought back the same train of symptoms; and several weeks elapsed before he got free from the distracting weakness.[1]

The vexation, annoyance, and even alarm, occasioned by these nervous seizures are often very great; and the patient begins to fear that he has some serious organic disease of heart or brain.

Now, it is a matter of some practical importance to be able

[1] Some years after this occurrence I was sitting next to the same clerical friend at a dinner party, when he began to tell me how much he had suffered some time before from inhaling chloroform at a dentist's. He became so faint that he had to lie down for some hours. In the midst of his narrative I saw that he was becoming very pale, and asked him if he did not feel faint. He replied that he did, and immediately he had to leave the table and lie down on the sofa. On this occasion, again, a mental influence brought back the physical prostration which had previously resulted from the chloroform.

to give an intelligible explanation of the phenomena, and upon this to base the suggestion of a simple and efficacious remedy. The explanation which I am in the habit of giving, and which I believe to be the true one, is this. Some kinds of emotional excitement, while they quicken and disturb the heart's action, partially suspend the breathing. It is the suspended breathing, the partial apnœa, the gradual emptying of the chest by the expiratory effort of speaking, that is the main cause of the increasing discomfort and the difficulty of articulation. We know, as a physiological fact, that a partial suspension of the breathing involves, as its necessary correlative, an impeded circulation, primarily through the lungs, and secondarily through the whole system; hence the feeling of giddiness, faintness, and increasing sense of exhaustion. That this is the true explanation of the phenomena in question is proved by the fact that a few deep inspirations, deliberately taken, usually suffice quickly to remove the distressing sensations. A deep inspiration, filling the chest with air, at once renders vocalisation more easy and the circulation more free; and I repeat that a few deep inspirations speedily lessen and remove the distress which had resulted from a partial arrest of the respiratory movements. I am in the habit of saying to these patients that, when they are threatened by a nervous attack while reading, they may ward it off, and escape all serious annoyance, if, while continuing to read, they give so much attention to their breathing as to ensure a continual succession of deep inspirations. I tell them to bear in mind that, while the organ is continually emitting sound, the bellows must be regularly and vigorously worked. It scarcely need be said that, if any discoverable bodily derangement or debility appears to contribute to the nervous disorder, this should be treated by suitable remedies.

In connection with this subject, it is not without interest to remark that most patients, whether men or women, who are subjected for the first time to examination by the laryngoscope require to be told that, while the examination is being made, they must continue to breathe freely. I had practised laryngoscopy for many months before I discovered that the slight nervousness experienced by most patients on having their throats in-

spected causes them to hold their breath; and this suspension of breathing soon distresses the patient, and embarrasses the operator. The direction which I now invariably give to my patients is, in substance, this: 'While I introduce the mirror and look at your throat, do not hold your breath, but continue to breathe in and out freely.' And I do not attempt to introduce the mirror until I see that my patient understands and obeys my directions.

Some time since, while I was discussing this subject with Mr. Ernest Hart, he reminded me of a precisely analogous instance of nervous apnœa. It happens occasionally that, when a nervous patient is about to inhale chloroform, emotional excitement so completely suspends the respiratory movements that the patient has to be encouraged, and instructed to continue his breathing, before the inhalation can proceed.

On another occasion I was discussing the same subject with a professional photographer, who told me that he often finds that when a nervous subject is told to sit still, preparatory to the removal of the cap from the apparatus, the sitter suspends the breathing and at once assumes an unnatural and anxious expression.

CHAPTER XIII.

LECTURE ON THE PATHOLOGY OF COMA AND ANÆSTHESIA.[1]

Coma—Definition—Various Conditions under which it occurs—One Condition common to all is a suspended or lessened Oxidation of Brain-tissue—Conditions essential for Oxidation of Brain-tissue—Free Movement of duly Oxygenised Blood and Freedom from any Agent which prevents the Blood from Giving up Oxygen to the Tissues—Coma of Epilepsy, of Syncope, and of Uræmia—Impeded Return of Venous Blood—Pressure of Depressed Bone or a Tumour on the Brain—Extreme Cold in causing Anæsthesia—Ligature of a Main Artery—Coma from Apnœa—Inhalation of Nitrous Oxide or Pure Nitrogen—Chloroform, Ether, &c., impede Oxidation—Experiment with a Lamp—Diagnosis of the Various Forms of Coma.

Coma may be defined to be a condition in which the functions of the brain proper—the cerebrum—are more or less completely suspended. The subject of coma comes under our consideration in connection with various diseases. Coma is an important symptom and result of cerebral hæmorrhage; it is one of the results of a fit of epilepsy; it may be a consequence of uræmic poisoning or of poisoning by opium, alcohol, chloroform, or ether. A state of drowsiness passing into coma occurs sometimes in ill-nourished and exhausted infants, and coma may result from the pressure of a tumour or of a depressed piece of bone upon the brain.

The circumstances under which coma occurs, then, are very various; but if we take an extended survey of the subject, so as to include all cases in which unconsciousness results from disease or accident or well-designed experiment, we shall find a number of facts pointing to the conclusion that, however diverse may be the remote causes of coma, the proximate cause in every instance is a *suspended or diminished oxidation of the brain tissue.*

[1] *Medical Times and Gazette*, April 3, 1869.

That the oxidation of the brain tissue may be effected it is necessary:

1. That there be a free current of blood through the capillaries of the brain.

2. That the blood be duly aërated or oxygenised.

3. That the blood be unmixed with any material which prevents or impedes the giving up of oxygen from the blood to the tissues.

The blood being the vehicle by which oxygen is carried from the lungs to the brain, it is obvious that a free current of blood through the cerebral vessels is necessary, in order to maintain the oxidation of the nervous tissue. The blood, in moving through the capillaries, undergoes a rapid change of composition. In the pulmonary capillaries it exchanges carbonic acid for oxygen, and in so doing, its colour is changed from black to red. On the contrary, in the systemic capillaries its oxygen is replaced by carbonic acid, and again it assumes the dark venous hue. Each particle of blood during its brief passage through the capillaries, while giving up its oxygen, of course loses its power as an oxidising agent. Therefore, an arrest of the capillary circulation through an organ immediately suspends the oxidation of its tissues, and, if that organ be the brain, loss of consciousness is an inevitable result. For the purpose of our present inquiry it is immaterial whether, as I have here assumed, there is a direct oxidation of the nervous tissue by the blood, or whether, as some physiologists believe, the functions of the brain are maintained by the oxidation of certain materials *within the capillaries*. It is certain that stagnant blood in the capillaries can no more maintain the functions of the brain than stagnant air in the lungs can maintain the function of respiration or stagnant air in a small closed vessel the process of combustion. This consideration enables us to explain the loss of consciousness in *epilepsy* and in *syncope*. In epilepsy, as is now generally admitted, the loss of consciousness is immediately due to an arrest of the cerebral circulation caused by a sudden and extreme contraction of the minute arteries of the brain. With the arrest of the oxygen-bearing blood-stream there is an immediate suspension of the brain's functions. And the phenomena of

epilepsy are exactly imitated when death occurs from a rapid and copious hæmorrhage or from sudden arrest of the circulation through the lungs by embolism, thrombosis, or the admission of atmospheric air into the veins during an operation at the root of the neck or in the axilla, or when, in the lower animals, the arteries which supply the brain with blood are compressed or ligatured.

In syncope the heart's action is enfeebled and the circulation ceases more or less completely, and in proportion to the degree in which the cerebral circulation fails, the functions of the brain, and especially consciousness, are suspended.

Closely allied to the unconsciousness of syncope is the drowsiness, sometimes passing into coma, which occurs in anæmic and exhausted infants. The red corpuscles are probably the chief agents by which the oxygen is conveyed from the lungs to the tissues. So that when, with a deficiency of red blood, there is combined a languid circulation, consequent on weakness of the heart, drowsiness and even coma are intelligible results.

That *uræmic coma* is in some cases partly a result of a deficiency of red corpuscles is probable; but a more constant and powerful factor in the causation of uræmic coma is to be found in the interrupted blood-supply resulting from the contraction of the minute cerebral arteries upon the morbid blood, whose free passage they resist. And here we have anatomical evidence in support of this explanation; for we find, in cases of chronic Bright's disease, the muscular walls of the minute cerebral arteries hypertrophied, in consequence of their continued over-action; and we know that over-action of the small arteries implies undue resistance to the supply of arterial blood.

As the cerebral circulation, and, with that, the functions of the brain, may be suspended in consequence of an interrupted flow of *arterial* blood to the brain, so the circulation may be arrested by an impeded return of *venous* blood *from* the brain. Thus a tight ligature on the neck may so compress the jugular veins as to cause first a venous and then a capillary stasis in the brain, and with an arrest of the

capillary circulation there is suspended function—in other words, coma or unconsciousness.

I have met with several instances of giddiness and even momentary loss of consciousness occurring during a violent fit of coughing. This is explained by the check given to the return of venous blood from the head during the violent expiratory efforts of coughing, when the superficial veins of the neck and face are often seen to be enormously distended in consequence of the impediment within the chest. Some pathologists would say that the unconsciousness and the coma which result from obstruction of the veins are due to *congestion* of the cerebral vessels. It is true that in these cases there is venous and capillary congestion; but it is, I think, equally true that the impaired cerebral function results, not from the excess of blood in the vessels, but from the fact that the blood in the capillaries of the brain is nearly, if not quite, stagnant.

While obstruction of the cerebral arteries causes anæmia, and obstruction of the veins causes congestion or over-fulness of the vessels of the brain, both the one and the other tend to suspend the functions of the brain; the essential cause of the suspended function in either case being not the mere excess or deficiency of blood in the cerebral vessels, but the arrest of the blood-stream through the capillaries.

We have next to consider a class of cases in which *pressure on the brain* is the cause of coma—for example, the pressure of a depressed piece of fractured cranium, the pressure of a tumour or of a clot of blood, the pressure of inflammatory or other effusions within the cranial cavity. In what way does pressure upon the brain cause coma or loss of consciousness? Possibly in more ways than one, but mainly, as I think, by interrupting the circulation, not merely through that portion of the brain which is directly subjected to pressure, but also through the surrounding parts, to which, over a considerable space, the pressure may be communicated through the soft and yielding cerebral tissue.

It is obvious that the disturbing effect of pressure upon the circulation and the functions of the brain will be greater

in proportion, not only to the degree and extent of the pressure, but also to the *suddenness* of its occurrence.

We have another example of anæsthesia from an arrest of the circulation in the complete insensibility of the skin produced by extreme cold, and in the unconsciousness which Dr. Richardson has produced in birds by freezing their brains. The congelation stops the circulation, and with it, of course, the oxidation of the tissues. The application of cold, even before it actually freezes the tissues, lessens sensibility; partly, perhaps, by diminishing the blood supply, and partly by checking the chemical action of oxygen. So the drowsiness which results from exposure of the whole body to extreme cold is probably explained, as the late Dr. Snow suggested, by the diminished consumption of oxygen which is thus induced. For 'the flame of mammalian life, like the flame of inorganic combustion of hydro-carbon, can only be sustained at a high temperature; a certain reduction is as fatal to one, as it is to the other in the Davy lamp.'[1]

The use of the *hot blast* in furnaces affords another illustration of the important influence of temperature upon the process of combustion.

We have another example of local anæsthesia consequent upon an impeded circulation, in the diminished sensibility, as well as the lowered temperature of a limb for a certain period after ligature of its main artery.

The late Mr. Moore, of the Middlesex Hospital, told me that he once treated an aneurism in the ham by pressure upon the femoral artery, and the interrupted circulation thus induced caused for a time complete loss of sensation in the leg below the knee.

I have before told you that, for the normal discharge of the functions of the brain it is essential not only that there should be a free current of blood, but that the blood be duly aërated or oxygenised. A suspension of respiration is quickly followed by unconsciousness, convulsions, and coma. When, from any cause, respiration is suspended, the resulting phenomena are complicated. There is—1, a more or less complete

[1] Professor Rolleston's address in Physiology at Oxford, *British Medical Journal*, 1868, vol. ii.

deoxidation of the blood; 2, the arterial blood becomes dark coloured, like venous blood, and contains an excess of carbonic acid; 3, the circulation of unaërated blood through the minute systemic arteries is checked by the contraction of their muscular walls; 4, there is a rapidly increasing obstruction and soon a complete arrest of the pulmonary circulation, and, as a consequence, of the systemic circulation also. Each of these conditions—namely, the deoxidation of the blood, the excess of carbonic acid, the impeded flow of unaërated blood through the systemic arterioles, and the diminished flow through the lungs into the systemic arteries—may contribute to the suspension of the cerebral functions.

The phenomena are much more simple when anæsthesia results from the inhalation of *nitrous oxide* or uncombined *nitrogen* gas. Nitrous oxide is a rapidly acting anæsthetic, causing complete unconsciousness in less than a minute. At a high temperature it is a powerful oxidising agent, but at the temperature of the body it gives up no oxygen, and is exhaled again unchanged. When inhaled in place of atmospheric air it rapidly replaces the oxygen; and, this being done, the functions of the brain are completely suspended and there is a state of profound coma, which quickly passes off when atmospheric air is again allowed to enter the lungs.

The action of unmixed nitrogen—in other words, of atmospheric air deprived of its oxygen—appears to be essentially the same as that of nitrous oxide, when inhaled; but a longer inhalation of nitrogen is required to produce anæsthesia, probably because nitrogen diffuses into the blood and replaces the oxygen less rapidly than nitrous oxide.

There is no reason to conclude that the inhalation of either nitrous oxide or nitrogen causes an accumulation of carbonic acid in the blood. The anæsthesia which results from the inhalation of these gases is due simply to the deprivation of oxygen. The lividity of the surface which often occurs, is a result, mainly, of distension of the veins, and not solely of black blood in the arteries. One effect of inhaling the nitrous oxide is to make the breathing slow and shallow, and at length entirely to suspend the respiratory movements of the chest and abdomen; with this there appears to be a sudden arrest

of the circulation through the lungs, consequently great distension of the systemic veins and lividity of the surface.[1]

The arrest of the circulation through the lungs is the result of contraction of the minute pulmonary arteries, similar to that which occurs in cases of ordinary apnœa.

The third condition which I before mentioned as being essential for the integrity of the cerebral functions, is that 'the blood be unmixed with any material which prevents, or impedes, the giving up of oxygen from the blood to the tissues.'

The late Dr. Snow,[2] after a careful inquiry into the mode of action of anæsthetic vapours, arrived at the conclusion that 'chloroform, ether, and similar substances, when present in the blood in certain quantities, have the effect of limiting those combinations between the oxygen of the arterial blood and the tissues of the body which are essential to sensation, volition, and in short all the animal functions. These substances (he goes on to say) modify and in larger quantities arrest the animal functions in the same way and by the same power that they modify and arrest combustion, the slow oxidation of phosphorus, and other kinds of oxidation unconnected with the living body, when they are mixed in certain quantities with the atmospheric air.'

The influence of chloroform vapour in arresting combustion may be shown by one or two very simple experiments. I put a few drops of chloroform into a tumbler, then gradually lower a short lighted taper into the tumbler. As the taper descends into the glass the flame begins to smoke and is soon extinguished by the vapour.

I have here one of Pillischer's so-called 'Queen's reading-lamps.' If now I incline the mouth of the bottle containing chloroform towards the openings by which the air passes upwards to the Argand burner, you see that at once the flame becomes smoky. The heavy vapour passes in with the air and impedes the combustion of the oil. And if I pour a few drops of chloroform into the metal reservoir below the burner

[1] See *ante*, p. 33.

[2] *On Chloroform and other Anæsthetics, their Action and Administration.* Edited, with Memoir, by Benj. W. Richardson, M.D. 1858.

the lamp will continue to smoke until all the chloroform has evaporated. It cannot be that the chloroform vapour acts by excluding or greatly lessening the supply of air to the lamp, for the air-spaces are so large that if I cover eleven out of the twelve with my handkerchief, sufficient air enters through the one opening to maintain the flame bright and smokeless.

Dr. Snow goes on to argue that all narcotics probably act by impeding oxidation. He refers to the observation of Dr. Prout, who discovered that fermented and spirituous liquors diminish the amount of carbonic acid given off by the lungs. He also refers to Beneke's observation that not only does alcohol lessen the amount of carbonic acid exhaled by the lungs, but also the amount of all the urinary constituents, which, as we know, are products of oxidation. And Dr. Snow proved by a series of careful experiments upon himself and upon animals that the inhalation of the vapour of chloroform or of ether, lowers the temperature of the body at the same time that it lessens the amount of carbonic acid exhaled by the lungs. In short, Dr. Snow collected a large amount of evidence in support of the proposition that the action of narcotics is mainly due to their power of lessening the oxidation of the tissues.

Dr. Snow further proved that the diminished oxidation is not explained by the combination of the narcotic substance with the oxygen of the arterial blood; for, as he says, the vapours of chloroform and ether escape, for the most part, unchanged in the expired air. Then, in the case of chloroform, the amount of material capable of combining with oxygen is quite insufficient to deoxidise the blood. And lastly, 'to increase the amount of oxygen in the respired air does not prevent the action of narcotics.'

The results of Dr. George Harley's experiments tend to confirm this explanation of the action of narcotics. Dr. Harley has shown that a mixture of chloroform, alcohol, morphine, and other narcotic substances with the blood out of the body lessens the amount of oxygen absorbed and of carbonic acid given off, when the blood is shaken up with atmospheric air.

This, then, completes the evidence in support of the proposition that *the proximate cause of coma is a suspended or diminished oxidation of the nervous tissue.*

So that while in one class of cases the coma is a result of an arrested circulation of blood, in another defective oxidation of blood, and in a third the addition of some narcotic to the blood, the proximate cause is in every instance identical—namely, a defective oxidation of the nervous tissue.

And now let us pass on from the discussion of the theory of coma to the important practical question of *diagnosis.*[1]

You are called to a patient in a state of insensibility; you know nothing of his previous history, except that he has been found unconscious in the street, and you are required to ascertain the cause of his alarming condition. You must first consider what are the *possible* causes of the symptoms. He may have a clot of blood in his brain, the result of disease or a blow on the head; he may be in that comatose condition which not unfrequently follows an epileptic fit; he may be poisoned by opium or by an excess of alcohol; or he may be suffering from the effects of uræmia.

Now, you will be less likely to make an erroneous diagnosis if you continually bear in mind that in some of these cases an accurate diagnosis is not only extremely difficult, but absolutely impossible until the progress of the symptoms has been watched for a certain time. That the case is one of cerebral hæmorrhage would be rendered probable by such symptoms as extreme relaxation or convulsive twitchings of the limbs on one side, lateral deviation of the features, or inequality of the pupils; but with hæmorrhage on the *surface* of the brain none of these paralytic symptoms may be present. Marks of external injury, especially about the scalp, should be carefully looked for in every case. A black eye or a cut or bruised scalp may have resulted from a drunken man falling down, but the fall may have fractured his skull or ruptured a blood-vessel in the brain.

That the coma is a sequel of an epileptic fit might be suspected if it were found that the tongue is bitten and bleeding, and if there were hæmorrhagic spots beneath the conjunctivæ

[1] *Medical Times and Gazette*, June 12, 1875.

or the skin; though these signs are frequently absent in epileptic cases. Epileptic coma is usually of short duration, and the speedy return of consciousness removes any doubt that may have existed as to the nature of these cases.

In cases of poisoning by opium, one of the most striking and constant symptoms is extreme contraction of the pupils. The skin, too, is usually bathed in a profuse perspiration. With respect to the contraction of the pupils, it has been observed that in cases of apoplexy with hæmorrhage into the pons Varolii the pupils have been as much contracted as in opium-poisoning.

When the patient's breath is tainted by the odour of some alcoholic liquor, we of course suspect that he is drunk. It must not, however, be forgotten that as a drunken man is especially liable to be seized with apoplexy, and to suffer from accidental mechanical injury in the streets of a crowded city, so we may have to deal with the complication of alcoholic intoxication and cerebral hæmorrhage on a fractured skull. It is a common practice to give brandy or some form of alcoholic stimulant to anyone who has become faint or giddy, and so it may happen that an unconscious patient's breath is tainted with the odour of drink administered after the onset of an apoplectic seizure.

In cases of uræmic coma the urine is usually albuminous, and presents other physical and chemical signs of renal disease. Uræmic coma, in a large proportion of cases, is preceded by convulsions. The tongue is commonly brown and dry in these cases, and the breath has a most peculiar and characteristic fœtor. Yet, even with all this evidence of renal disease, the case may not be one simply of uræmic coma; there may, in addition, be the complication of cerebral hæmorrhage, which, as we know, is a frequent result of chronic Bright's disease.

The difficulty of diagnosis between renal disease and drunkenness is sometimes increased by the fact that the urine may be rendered temporarily albuminous by alcoholic intoxication. A remarkable case of transient alcoholic albuminuria occurred when my friend and colleague the late Dr. Baxter was house-physician to the hospital. A man

between twenty and thirty years of age, was brought in one night by the police. He was unconscious, and breathing stertorously. He appeared to be drunk, and a large quantity of vinous liquid was pumped out of his stomach. The unconsciousness continued, and it was then suspected that he might be suffering from uræmic poisoning. This suspicion was confirmed by the fact that his urine, drawn off by a catheter, was 'loaded with albumen.' He was then put into bed, cupped over the loins, and a purgative was given. When Dr. Baxter visited the ward the following morning, he found the man up and dressed, and clamouring for his discharge. He said that he had been very drunk overnight, but now he had nothing the matter with him; and he passed some urine, which was found to be, in every respect, quite normal. The temporary albuminuria was the result of renal congestion, caused by the excretion of an excess of alcohol or the products of alcoholic excess through the kidneys.

In all doubtful cases of this kind it is better to err, if you err at all, on the side of caution and safety. Obviously it is better to allow a drunken man to recover his senses in the ward of a hospital than to send an apoplectic patient, or one with a fractured skull, to die in the cell of a police-station. When a mistake is made on the opposite side, and a supposed drunkard dies apoplectic, it is a very natural, though it may be a very erroneous inference, that someone is to blame and deserving of punishment.

CHAPTER XIV.

ON THE ETIOLOGY AND PATHOLOGY OF THE SO-CALLED 'DENTIST'S LEG.'[1]

Various Persistent and Painful Sensations after Standing long in one Position—Caused by Pressure of Contracted Muscles on Blood-vessels and Nerves—The Means of Prevention and Cure.

At the meeting of the Odontological Society in June, 1884, Mr. Oakley Coles read an interesting paper on the 'Maintenance of Health amongst the Practitioners of Dental Surgery.'[2] The reading of the paper was followed by an instructive discussion, in the course of which mention was made of the pain in the back and legs resulting from over-fatigue in muscles which are engaged in maintaining the body for a long period in one constrained position. One speaker, Mr. Dennant, of Brighton, said he had himself 'suffered from what their medical friends were learning to call the "dentist's leg."' About two years since the pain became unbearable after standing three or four hours, resembling very much the application of scalding water to the outer part of the thigh in the region of the external cutaneous nerve. It seemed to be due to nervous exhaustion from the undue strain thrown upon the part. Medical friends and common-sense suggested rest as the remedy, and this he secured by means of the 'Lyons stool.' He had used this for about two years with great benefit, and could now get through a day's work with comparative comfort.

Some time since I had my attention directed to this subject in consequence of having been consulted by one of my

[1] *Lancet*, August 15, 1885.

[2] *Transactions of the Odontological Society of Great Britain*, vol. xvi., No. 8, new series.

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friends, a dentist, about 35 years of age, who described to me a sensation of numbness in one thigh which he feared might be a precursor of paralysis. I soon, however, relieved him of his anxiety by giving him what I have no doubt is the true explanation of the perverted sensation. Our every-day experience teaches us that overstrain and fatigue may be direct causes of pain in the muscles concerned; but, besides this, the long-continued rigid contraction of the muscles which are engaged in maintaining such a fixed position as the operating dentist often has to assume, must greatly impede the circulation, not only through the muscles, but also through the integuments. As the alternate contraction and relaxation of the muscles—in walking, for instance—assists and quickens the circulation, so the state of fixed and rigid contraction must obviously impede and retard the circulation, by exerting a continuous pressure upon the blood-vessels, and more especially upon the soft and easily compressed veins. The impeded circulation affects not only the muscles, but also the skin and subcutaneous tissues, and the nerves which supply the different tissues; and one result of a defective circulation through the nerves is to cause various perverted sensations—such as numbness, a sensation of 'pins and needles,' or a painful feeling of heat and scalding. The immediate cause, then, of the painful sensations experienced and so graphically described by Mr. Dennant appears to be not, as he suggests, 'nervous exhaustion,' but perverted nerve function, directly due to a mechanical impediment to the circulation through the rigidly contracted muscles and their associated nerves. It is probable, too, that direct compression of the nerves by the firmly contracted muscles may have some influence in the causation of the perverted sensations referred to the cutaneous terminations of the nerves.

I have often been consulted by men and women beyond middle age who have been alarmed by a feeling of numbness or 'pins and needles' in the extremities. In these cases the defective circulation, which is the direct cause of the perverted sensation, is often the result of an enfeebled condition of the heart, with or without excessive general obesity, and often with more or less general emphysema of the lungs, a condition

of things which tends to impede primarily the pulmonary, and secondarily the systemic circulation. These perverted sensations in the limbs, in so far as they result from an interrupted or defective blood supply to the nerves, are strictly analogous to the more serious condition, coma, which we discussed in the last chapter.

To return to the 'dentist's leg.' The obvious means of prevention and of cure consist in rest for the overstrained limb, or such a frequent change of position as is equivalent to a certain amount of rest. Standing in one position is notoriously more fatiguing than walking, and for the obvious reason that while in standing one set of muscles is in a constant state of active contraction, the circulation through them being thereby retarded and enfeebled, walking involves alternate contraction and relaxation of the muscles, with an invigorated and quickened circulation.

More than one speaker during the discussion referred to the benefit to be derived from some form of active muscular exercise after the day's work. It is probable, too, that systematic friction and massage of the affected limb would be beneficial.

CHAPTER XV.

THE ASSOCIATION OF SENILE DEGENERATION OF THE BLOOD-VESSELS OF THE BRAIN WITH CEREBRAL SOFTENING AND MENTAL DECAY.

Structural Changes the Result of Senile Degeneration of the Cerebral Blood-vessels—Similar Changes sometimes occur in Early Life as a Result of Special Morbific Agencies—Malnutrition of Brain consequent on Vascular Degeneration—Degenerative Changes often occur in the Systemic Arteries throughout the Body—Symptoms vary according to the Parts of the Brain affected—Amongst the most Distressing are those which affect the Intellect—Diagnosis—Treatment.

The object of this chapter is to emphasise the very frequent connection of senile degeneration of the blood-vessels, especially of the *arteries* of the brain, with cerebral softening and loss of mental power. The structural changes which occur in the small blood-vessels of the brain, as a result of senile degeneration, have been well described and illustrated by Sir James Paget,[1] and they are now well known to all pathologists. The changes are essentially the same as those which affect the larger arteries, the true nature of which, as a fatty and calcareous degeneration, was first described by Mr. George Gulliver.[2] The most constant appearance is that of minute shining black-edged particles, partly calcareous and partly oily, irregularly scattered beneath the outer surface of the minute blood-vessels of the brain. As the degeneration advances these particles increase in number and size until the whole extent of the affected vessel is thick-set with them, so that the natural structures, if not quite wasted, can hardly be discerned. With the wasting and disappearance of the normal

[1] *Surgical Pathology*, 3rd ed. p. 106.
[2] *Med.-Chir. Trans.*, vol. xxvi. p. 86.

structures the blood-vessels undergo various changes of shape. Often the outer layer is rendered uneven and tuberous by clusters of oil particles beneath it, and sometimes there occur small aneurismal dilatations of the diseased vessel. The vessels most liable to these changes are the arteries of about $\frac{1}{800}$th of an inch in diameter; but they exist generally at the same time in the veins of the same or of a less diameter. As a general rule the disease decreases nearly in the same proportion as the size of the vessel, and the smallest capillaries are least, if at all, affected. But there are many exceptions to this rule; and it is not rare to find vessels of from $\frac{1}{2000}$th to $\frac{1}{3000}$th of an inch in diameter having parts of their walls nearly covered with the abnormal deposits.

Although the changes here described are commonly the result of old age, analogous degenerations may occur in the prime of life, as results of alcoholic excesses or of some inherited or acquired cachexia, such as the syphilitic or the cancerous or the gouty. In the advanced stages of chronic Bright's disease, too, the elastic arteries are liable to degenerative changes, partly due to the abnormal condition of the blood which they transmit, and partly to the excessive strain to which they are subjected between the hypertrophied left ventricle behind and the resisting hypertrophied muscular arterioles in front.

As a result of these degenerative changes in the blood-vessels, the nutrition of the brain suffers in various ways.

1. The elasticity of the larger arteries being impaired by degeneration of their walls, involves a lessened blood current through their canals and a diminished blood supply to the nervous tissues.

2. Degeneration of the muscular coat of the arterioles implies the loss or diminution of the power of regulating the blood supply in accordance with the physiological requirements of the tissues.

3. The structural changes in the walls of the capillaries must tend to impair their osmotic powers, and to interfere with that interchange of materials between the blood and the tissues which is essential for the maintenance of healthy nutrition.

4. When the coats of the blood-vessels, whether arteries, veins, or capillaries, have undergone structural changes, the blood has a tendency to coagulate within the diseased vessels; and so the brain tissue, over an extent depending on the size and the number of the obstructed vessels, being deprived of its blood supply, rapidly undergoes degeneration and softening.

As we are now referring mainly to the *senile* changes in the blood-vessels, we do not discuss the subject of obstruction of the cerebral blood-vessels by embolism, since that may occur as a result of disease of the cardiac valves, or of aneurysm and other causes in young subjects.[1]

5. The vessels which have undergone degenerative changes are less able to resist the blood pressure to which they are subjected, and consequently they are liable to become dilated into aneurismal pouches, and, as a result of their rupture, hæmorrhage into the brain tissue is of frequent occurrence.

The loss of the controlling power over the blood supply which results from the degeneration of the muscular coat of the arterioles increases the risk of capillary hæmorrhages.

A common result of the defective blood supply to the brain is that its tissue is ill-nourished and becomes more or less softened, and the specific gravity of the brain substance is lowered. The softened tissue in the white substance of the hemispheres may be quite colourless, but the softened portion, both in the white and the grey matter, has often a red or a yellow tinge, the colour being due partly to the engorgement of vessels in the immediate neighbourhood of other vessels which have been obstructed, and partly to the extravasation of blood-colouring matter into the softened tissue.

On microscopic examination, the nerve tubes are seen to be broken up, and fatty granules take the place of the myeline. Many free oil globules are also found between the tissue elements, and aggregated into masses which constitute the 'granular corpuscles' of Gluge. Oil globules also collect *outside* the walls of the capillaries, and are distinct from the oily particles in the substance of the degenerate vessels. In the patches of extravasated blood are seen

[1] This subject is referred to in the chapter on *Thrombosis and Embolism*.

amorphous yellow granules or flakes and ruby-coloured crystals of hæmatodine.[1]

The changes in the blood-vessels which result in atrophy and softening of the brain tissue are rarely, if ever, limited to the cerebral vessels.

In the great majority of cases, similar degenerative changes occur in many, if not in the whole, of the systemic arteries, although those in different organs and regions of the body may be implicated in different degrees. The coronary arteries of the heart are often affected, and the resulting malnutrition and weakening of the muscular walls of the heart increase the embarrassment of the cerebral circulation.

It will often be found that, in patients who present many of the symptoms which result from degeneration of the cerebral blood-vessels, the subcutaneous arteries are everywhere hard and tortuous, the result of atheromatous and calcareous degeneration with impaired elasticity; and the observation of this fact is an element in the diagnosis of the brain disorder. On the other hand, it will sometimes be found that, while the superficial arteries which we are able to examine are hard and tortuous, in consequence of senile degenerative changes, the intellect and the other cerebral functions remain for the most part unimpaired. We conclude, therefore, that the cerebral vessels in these cases are comparatively free from the structural degenerations which affect other parts of the arterial system.

The *symptoms* which result from degeneration of the cerebral blood-vessels are those which are usually described under the head of chronic softening of the brain, and it may be that no serious permanent symptoms occur until the brain tissue has undergone some atrophic change. The symptoms present great variety in different cases, according to the parts of the brain which are chiefly implicated. Every function of the brain may be more or less seriously impaired. The intellect is usually weakened, memory fails, speech is often affected, the speaker stops in the middle of a sentence, being unable to

[1] Dr. Bastian has given a very complete account of the morbid anatomy of softening of the brain in Reynolds's *System of Medicine*, vol. ii. p. 460.

recall or to utter the next word. There may be more or less of general paralysis, with special weakness of certain sets of muscles. The legs are sometimes dragged with a scraping movement of the sole of the foot at each step. Hemiplegia is a common form of paralysis, a result of implication and perhaps plugging of one middle cerebral artery; and with right hemiplegia there is often more or less complete aphasia. Sudden impairment of vision in one eye may sometimes be seen by the ophthalmoscope to have been caused by a thrombus in a branch of the retinal artery. The symptoms, whether paralytic or otherwise, which result from a thrombus pass away sometimes, after a few minutes or hours, almost as suddenly as they came; the probable explanation being that a soft clot which had temporarily blocked an artery has been broken up and carried on into the terminal vessels, where it causes but little impediment to the circulation. Thus a complete hemiplegia may in a few hours give place to only a slight weakening of the affected side. But the same artery is liable to be again plugged, and perhaps permanently, with the result of a sudden and persistent paralysis. Pain in the head, more or less diffused, is sometimes complained of. Giddiness and confusion of thought are common symptoms. Various sensations, the result of central changes, may be referred to the cutaneous surface, or to certain sets of muscles, assuming the form of hyperæsthesia, or of numbness with formication. Muscular rigidity sometimes occurs. The whole character and temper often become painfully changed. The man of powerful intellect, calm and self-possessed, becomes irritable, imbecile, peevish, and sometimes delirious and violent. The control of the bladder and rectum may be more or less impaired. In some cases convulsion occurs, and this may be quickly succeeded by fatal coma, or the convulsion may recur again and again at uncertain intervals, constituting what is sometimes called *senile epilepsy*. In other cases, a sudden termination is the result of the giving way of an artery and an outburst of cerebral hæmorrhage.

The Laureate, in one of his later poems ('The Ancient Sage'), has given a touching and graphic description, evidently the result of close and accurate observation, of the

wreck and ruin of intellect which may be the outcome of old age.

It is a sceptic who speaks :

> The statesman's brain that sway'd the past
> Is feebler than his knees ;
> The passive sailor wrecks at last
> In ever-silent seas ;
> The warrior hath forgot his arms,
> The learned all his lore ;
> The changing market frets or charms
> The merchant's hope no more.

The above is a fragment of a description, the whole of which is most interesting and pathetic and strictly true to nature.

The diagnosis of these cases of degeneration of the cerebral blood-vessels with tendency to atrophic softening is seldom attended with difficulty. The characteristic features are the progressive loss of brain power in one more or less aged and with evidence of general arterial decay and rigidity. It is, however, to be noted, as I have before intimated, that different portions of the vascular system may be affected in very different degrees. Thus we may sometimes find decided evidence of cerebral softening in an elderly subject, whose radial and other superficial arteries appear to have lost little of their original softness and elasticity ; while, on the other hand, an old man with hard and tortuous radial arteries may for years present few or no signs of failing brain power. In some cases, while the arteries of the limbs are little affected, the coronary arteries of the heart have undergone degenerative change in common with those of the brain ; and in consequence the ill-nourished heart has a peculiar rapid tumbling action, while the pulse is feeble, irregular, and often intermitting. This combination of a feeble heart with rigid cerebral vessels must obviously increase the hindrances in the way of a due supply of blood to the brain, and favour the tendency to degenerative changes in the tissue of that organ.

The dull pain which is sometimes complained of by these patients is much less constant and severe than that caused by the growth and pressure of a cerebral tumour.

The pain of chronic meningitis, too, is usually more severe

than that of cerebral softening, and the progress of the disease towards a fatal termination is more rapid.

Treatment.—The treatment of symptoms which result from senile degeneration of the tissues can obviously be only palliative. We have no power to put back, or even to arrest, the hand upon the dial. The symptoms of impaired cerebral circulation may in the earlier stages be relieved by measures calculated to improve the general nutrition. Nutritious food should be given at short intervals, and even during the night, with wine or some other form of alcoholic stimulant. Exposure to cold and damp is to be carefully avoided. Fatigue, whether of mind or body, may be very injurious. Vertigo and confusion of thought may often be relieved and warded off by maintaining the recumbent posture with the head only slightly raised. All lowering medicines are, of course, to be avoided. Constipation, which might involve a dangerous straining at stool, should be obviated by the mildest laxatives.

As a tonic, a combination of quinine, iron, and strychnine may be given. Insomnia, with tendency to delirium, may often be counteracted by liquid or solid food with wine. If these means fail, a cautious use of chloral or the bromides is often successful. Daily driving in an open carriage when the weather and season permit should be encouraged. All mental and emotional excitement should be carefully avoided. Many troubles and complications may arise requiring special care and management, but in spite of all treatment the tendency is towards an inevitably fatal termination.

CHAPTER XVI.

CASES OF POISONING BY HOMŒOPATHIC CONCENTRATED SOLUTION OF CAMPHOR.

Convulsions and Hemiplegia caused by Twenty-five Drops of Homœopathic Solution of Camphor—Violent Headache, Vomiting, &c., after Eight Doses of Three Drops taken every Five Minutes—Profound Coma after a Teaspoonful Dose—Epileptiform Convulsions after a Teaspoonful Dose—A Swiss Guide rendered giddy and unable to proceed by about Twenty Drops—Faintness and Giddiness caused by Seven Drops of the Solution—Epileptiform Convulsion caused by about Fifteen Drops—Unconsciousness caused by an uncertain amount applied to an Aching Tooth—An Eton Boy rendered Unconscious by about Fifteen Drops—Epileptiform Convulsions caused by Seven Doses of Three Drops in the course of about Six Hours—Camphor in Solution much more active than in the Solid State.

I FIRST brought this important subject to the notice of the profession in a paper which was published in the seventh volume of the *Transactions of the Clinical Society* (1874), and of which the following is the substance:—

Miss S——, æt. 20, consulted me at the beginning of September 1873 on account of certain nervous symptoms which were believed to have resulted from an overdose of camphor taken some months before.

The following history was obtained from the patient, her mother, and my friend Mr. Charles H. Drake, of Brixton.

Miss S—— had always enjoyed good health, and had never shown any symptoms of nervous disorder. On February 19th, 1873, she had a slight cold and sore throat, for which she took at bedtime a dose of 'Epps's homœopathic concentrated solution of camphor.'

The patient says that in the presence of a servant who watched her, she dropped twenty-five drops into a wineglass of water and drank it off. The medicine at once caused a burn-

ing sensation in the mouth and throat, which made her call for more water, which she drank. She then took some gruel and lay down in bed. She believes that she immediately went to sleep. In a short time her sister, who occupied another bed in the same room, hearing her make a strange noise, spoke to her, but receiving no answer she got up and found the patient insensible, foaming at the mouth, black in the face, and violently convulsed. The sister had often seen epileptic fits in a lady of her acquaintance, and she ran to her mother and said that her sister was in a fit. In a few minutes the convulsion ceased, and was followed by vomiting of a pink, probably blood-tinged, fluid, having a strong odour of camphor. Meanwhile Mr. Drake had been sent for, and when he arrived about an hour had elapsed. She was then in a deep sleep, from which she could with difficulty be aroused. The throat looked inflamed, the tongue was covered by a thick creamy fur, and the pupils were dilated. A mustard emetic was given and more camphorous liquid was ejected. Some strong coffee was then given. For several hours the breath continued to smell of camphor, and she was very drowsy during the night and the following day. For several days she complained of pain and tenderness over the stomach, increased by taking food. Numbness of the tongue continued for a fortnight, and the left arm and leg were partially paralysed. She dragged the left leg along the ground in walking. Gradually these symptoms passed away; but when she consulted me seven months afterwards, she complained that since taking the poisonous dose she had never passed a night without being disturbed by distressing dreams. She had continued to feel weak and nervous, and occasionally had a feeling of weakness and numbness in the left arm and leg.

The symptoms which occurred in this case were similar to those which in several recorded cases have resulted from an overdose of camphor. Pereira says that 'in its power of causing stupor camphor agrees with opium, but it differs from the latter in its more frequently causing delirium and convulsions.' In the fourth edition of Pereira's *Materia Medica* (vol. ii. part i. p. 455) five cases are cited in which convulsions resulted from a large dose of solid camphor. In one case the

dose was two scruples, in another two scruples, and in the case of three children aged respectively 5 years, 3 years, and 18 months, about half a drachm caused convulsions, and in the youngest child a single attack of convulsions was followed by fatal coma.

It seems probable that while the convulsive attack in my patient was a direct result of the camphor entering the circulation, and perhaps causing a defective blood supply to the brain by exciting spasm of the cerebral arterioles (see the chapter on Epilepsy), the paralytic symptoms and the subsequent nervous derangement may have been due to an injury inflicted on the brain by the violent epileptiform paroxysm.[1]

I took the residue of the solution of camphor to Messrs. Bell & Co., in Oxford Street, and Mr. Gale did me the favour to analyse it. He reports it to be a saturated solution of camphor in rectified spirit, the proportion being an ounce of solid camphor to an ounce and a quarter of rectified spirit. So that this so-called homœopathic camphor is stronger than the spiritus camphoræ of the British Pharmacopœia in the proportion of seven and one-fifth to one. The spiritus camphoræ P.B. contains one ounce of camphor in nine fluid ounces of rectified spirit.

There is some doubt as to the amount of this concentrated solution that my patient took. She believes that she took only twenty-five drops, which would contain about ten grains of camphor (twenty-five measured minims would contain twenty grains). It is obvious that when the spirituous solution is mixed with water the camphor is precipitated in a state of very minute subdivision, and so in a state more favourable for rapid absorption than when camphor is taken in the form of coarse powder. The printed directions on the bottle are: 'Dose, two to five drops on a lump of sugar, which may be repeated every half-hour or oftener if necessary.' The patient, probably thinking that a homœopathic medicine could not be poisonous, went much beyond the prescribed dose; but that a literal compliance with the printed directions

[1] I have lately (January 1887) heard from Dr. Frodsham, Miss S——'s present medical attendant, that she has had no return of nervous symptoms, and that she is now in good health.

may be attended with serious consequences is shown by another case, the particulars of which were given to me by the patient himself.

The Rev. W. R——, æt. 64, a highly intelligent, energetic clergyman, in the spring of 1870 had a slight cold, for the relief of which he was advised to take three drops of homœopathic solution of camphor every five minutes for an hour. In order to ensure strict compliance with the directions he sat down to write letters, while his daughter, watch in hand, sat by him and gave the dose at regular intervals. After taking the eighth dose he suddenly felt intense pain in the head, and, saying to his daughter, 'I can take no more of the medicine,' he went into another room, where his wife seeing him exclaimed, 'Why, you are as pale as death!' He vomited several times, then went to bed, where, for forty-eight hours, he suffered from intense headache. When he got up he felt very severe pain in the spine, which made it difficult for him to maintain the erect posture. This pain continued for about a month, and it was two months from the time of taking the camphor before he was able to resume his duties in the church. He has since remained in his usual good health.

This gentleman informs me that since the occurrence of his own case, which excited much interest amongst his friends and acquaintances, he has heard of several instances in which very unpleasant and alarming symptoms have resulted from the employment of the same concentrated solution of camphor.

I am indebted to Mr. Delamark Freeman for the particulars of the following case, which I give in his own words:—

'A young lady, aged 19, about eighteen months ago took, for an attack of diarrhœa, a teaspoonful of homœopathic solution of camphor in water. Soon after taking it she was seized with burning pain in the mouth, fauces, and pit of the stomach, and she complained of feeling giddy, great dimness of vision, *tinnitus aurium*, numbness and tingling of the arms and legs, with loss of muscular power. When I saw her, which was soon afterwards, I found her in a state of insensibility, in which she remained for more than three hours. There was coldness of the surface with a quick and feeble pulse, conjunctivæ insensible to the touch, respiration feeble; tickling of the feet

caused no reflex action, but she had slight convulsions. Her breath smelt strongly of camphor. After an emetic of mustard she gradually became sensible, and had the appearance of a person awaking from a very profound sleep. Afterwards she had burning heat of the skin, full and quick pulse, twitching of the eyelids, much agitation, headache, giddiness, and inability to walk from loss of muscular power. Her mouth was in some places stained white by the flocculent deposit of camphor, and in other parts the mucous membrane was slightly red, swollen, and painful. It was some days before the symptoms entirely passed away. I examined the solution, and found it to be a saturated spirituous solution of camphor, by far stronger than the ordinary spirit of camphor of the Pharmacopœia.'

Modern homœopaths admit that there are amongst them 'men of high dilutions, men of low dilutions, and men of no dilutions at all;' and it would appear that the disciples of Hahnemann have passed from the irrational and ludicrous extreme of infinitesimal dilution to the dangerous extreme of the greatest possible concentration of active and poisonous drugs.

It is difficult to conceive what useful purpose can be served by the employment of so concentrated a solution of camphor as that in question, the preparation being so hot and acrid that it cannot be taken, even in small doses, without being largely diluted. Then there is an obvious risk that this concentrated solution may be mistaken for the much weaker solution of the British Pharmacopœia; a mistake which, in spite of the printed directions on the bottles, was probably made by both the young ladies who suffered so seriously for their error.

I have thought it right to bring forward these cases in the hope that their publication may serve as a warning of the danger which attends the employment of homœopathic concentrated poisons.

Appendix to Dr. G. Johnson's *Paper on Poisoning by Homœopathic Camphor.*

On July 2nd, 1874, I (G. J.) saw, in consultation with Mr. G. H. Wade, of Chislehurst, Mr. C. P——, æt. 18, articled clerk. On June 27th Mr. P—— had a slight diarrhœa, for which he took at 3 P.M. about a teaspoonful of Rubini's saturated solution

of camphor, in water. Immediately after swallowing the dose he went out to the cricket field, a distance of about a quarter of a mile. Arrived there, he began to feel confused and giddy. For this he took a small quantity of brandy and water. In about half an hour after taking the camphor he suddenly fell down and was violently convulsed. The bystanders said he appeared to be in an epileptic fit. Within about ten minutes from the time of the seizure Mr. Wade happened to be driving past, and was called to him. The patient was then in a state of semi-consciousness, and was making ineffectual attempts to get upon his feet. Mr. Wade immediately had him put into his carriage and drove him home. He then gave him an emetic of sulphate of zinc and common salt, which acted quickly, and brought away a large quantity of precipitated camphor. The patient then went to bed, and remained there until the following morning. During the five days since the attack he had felt weak and had an occasional sensation of giddiness ; but, in consequence probably of the speedy ejection of the camphor by the emetic, the after-symptoms were fewer than in the previous cases. With the exception of the slight diarrhœa, this youth was in good health and had had no illness since the summer of 1868, when for a few hours he had headache and delirium after being exposed bareheaded to the heat of the sun.

The chief anxiety of the patient's friends was to know whether the attack of convulsion was a result of the overdose of camphor. After my experience of similar cases I had no hesitation in saying that it was, and that no return of the convulsion was to be apprehended.[1]

Postscript.

From the 'British Medical Journal,' December 6, 1873.

To the Editor.

'Sir,—I regret that I did not observe the notice of Dr. George Johnson's communication to the Clinical Society on

[1] On December 29, 1886, Mr. Wade, in answer to my inquiry, wrote me the following note:—'Mr. C. P—— has had no sign of convulsion or any other nervous symptoms since you saw him. Of course he was somewhat unwell for a little time after, but nothing requiring a note.'

the above subject; had I seen it I should have forwarded to him the following case, in addition to the three which he read. In September 1871 I was staying at the Bel Alp, with Johann Jaun as guide. Jaun is an overland guide, and is well-known as an active and vigorous man. His age is perhaps 28. One morning we started at 3.30 A.M. for the ascent of the Schienhorn, and a London gentleman (whose name I forget), who, with his guide, intended to cross the Beich Grat, accompanied us on our way.

'When we had reached the *névé* of the Beich Glacier, Jaun, who had fallen back, came up to me and said he was suffering so severely from diarrhœa that he feared he should be unequal to the ascent. As the morning was very fine I was much disappointed, and mentioned the state of the case to my companion. He promptly brought forth a bottle of "homœopathic camphor," with which he had been armed by his own homœopathic attendant against diarrhœa. I caught at any chance of a cure, and gave my guide ten drops of it, which was double the dose ordered to my companion. As another loose motion occurred, I gave him a second dose of ten drops at most. I say "ten at most," because in my slightly contemptuous estimate I dropped it carelessly upon a bit of sugar. My intention was to give him a liberal five drops. He may, however, have taken twenty drops in all. Soon afterwards he began to feel very ill, and in particular to suffer from a distressing giddiness (*Schwindel*). He had also some headache and nausea, and became so nervous that he feared to walk any farther upon the snow. We were certainly upon a slope, but it was not very steep; and before taking the medicine he would have walked upon it without a thought. As it was, we had to deposit him in a cave for safety, and I gave him some ice-water and bathed his head. When quiet he seemed better, and my companion and myself walked on to the summit of the pass with the other guide, and remained there until Jaun crawled up about an hour and a half later, still feeling very giddy and nervous. He picked out the safest place on the rocks, and there fell off into a sort of lethargy, from which I gently aroused him from time to time. He was quite unable to take food, but he drank some wine and water

at intervals. As the morning was hot I was able to remain about two hours longer on the summit, when Jaun felt able to return homewards; fortunately also a gentleman came up from Ried with two guides, so that we reached the Bel Alp without further difficulty. He grew better as we returned, and next day was quite well. There was no diarrhœa after the second dose. When I read Dr. Johnson's cases I see that we were lucky to come off so well, and that I was to blame in leaving Jaun behind without assistance. Without the other guide, however, we could not have proceeded, and my companion had a long walk before him.

'I am, &c.,

'S. CLIFFORD ALLBUTT.

'Leeds, Dec. 1873.'

The publication of the preceding paper, with the appendix and postscript, served to direct the attention of the profession to the subject, and led to the publication of other cases of camphor poisoning. In the *British Medical Journal*, Feb. 6th, 1875, appeared the following communication from myself:—

Another Case of Poisoning by Homœopathic Solution of Camphor.

I am indebted for the particulars of the following case to a former pupil, who gives me permission to publish them, but begs me to withhold his name, for the reason that it is a rule of the public service in which he is engaged that no medical officer shall publish his cases without special permission, which is not always granted when asked for. I give the case as reported in a letter addressed to me by the medical officer who saw the patient.

'I was called in to see a lady about 35 years of age, and found her with a very pale face and weak pulse. She said she had been suffering from a bad cold and feverishness; that she had got up late, and had her dinner at about two o'clock. About an hour after dinner, her sister, who is an experienced nurse, dropped seven drops of homœopathic solution of camphor on a lump of sugar, and gave it to her.

Immediately after taking it, she was attacked with a very faint feeling, which compelled her to lie down flat on the hearthrug, and she nearly lost consciousness. This lasted about five minutes. When I arrived, her face was very pale, and her pulse was weak. I ordered her to bed, and gave her some aromatic spirit of ammonia and lavender. She was very drowsy after she got to bed, but had no more vertigo or faintness; and in a day or two she got rid of her cold. I am led to believe that this was a case of camphor poisoning similar to those which you have published, for the following reasons: the attack occurred immediately after taking a dose of camphor; the patient says that she had never before had a similar feeling of faintness; and the vertigo or faint was followed by drowsiness.'

There can, I think, be no doubt that the symptoms were a direct result of the camphor. The dose was smaller than in any other case which I have met with, in which poisoning has resulted from the incautious use of this dangerous compound. Seven drops would be an uncertain quantity; but seven measured minims would contain rather more than five and a half grains of camphor.

The *British Medical Journal*, Feb. 20, 1875, contained the following two communications:—

'*Case of Poisoning by Homœopathic Solution of Camphor.*

'The case of camphor poisoning related by Dr. George Johnson in the *Journal* of the 6th instant has induced me to look up my notes of a case that came under my observation about fourteen months ago.

'Last winter, a young lady about 20 years of age, who generally enjoyed good health, while dressing, about half-past seven, to go out to an evening party, became very giddy and unwell. She went into the next room to her sister; said she felt very ill; threw her arms around her, and was immediately seized with strong convulsions. I was sent for; but, not being at home, she was seen by Mr. Armstrong. The convulsions had passed off. In about a quarter of an hour there was free vomiting, and the patient remained unconscious for

some time longer. I was requested by a second messenger to see her as soon as I came home. I did so a little before eleven. She complained at that time much of headache and giddiness, and was evidently confused. Her pulse was quick; skin moist. Her father told me downstairs that she had had an epileptic fit. I expressed my unwillingness to believe it was a case of epilepsy, until I failed to discover that it originated from some other cause. At the request of her parents, I visited her again in two hours—about half-past 1 A.M. Having had some sleep, she seemed more comfortable, and was decidedly clearer mentally. After much conversation and inquiry, which did not elicit anything of importance bearing on the illness, when I had almost despaired of gaining any further information, her mother told me that the only thing her daughter had taken that evening after dinner was a few drops of camphor while she was dressing, for a slight cold in the head; and "that of course," she added, "could not make her ill." I thereupon expressed a wish to see what remained. A half-ounce bottle was produced, more than half-full, labelled "homœopathic solution of camphor," with the name of a respectable firm. There was no intimation of the dangerous potency of the drug. It was bought, I was informed, because it was considered good for a cold; and many ladies took it for that reason. The lady said, moreover, that as it was a *homœopathic* preparation, she never doubted its being a safe one. I dropped fifteen drops (which was admitted by the patient to be about the quantity taken) into a tumbler with a wineglassful of water, when a large deposit of camphor took place. The previous perusal of Dr. George Johnson's and Dr. Clifford Allbutt's striking and instructive cases of poisoning by this strong solution enabled me at once to assure the friends of the patient that it was a case of camphor poisoning, and that there would be no recurrence of the attack, if the dangerous medicine were at once discarded from domestic use.

'It is worthy of remark, that, while chemists carefully label laudanum in small quantities "poison," they do not hesitate, not only to sell a drug considerably more deadly in its character, to anybody who asks for it, without any intimation whatever as to dangerous properties, but rather mislead the

public by using an adjective which, in former years at least, was associated with everything innocuous and absurd.

'A. Legat, M.D., South Shields.'

'*Poisoning by Homœopathic Tincture of Camphor.*

'Reading, in the *British Medical Journal* of February 6, Dr. George Johnson's statement of cases showing the poisonous effects of the homœopathic solution of camphor, I was immediately reminded of one which occurred on the evening of Christmas Day. I was sent for to see a young man who had suddenly fallen down insensible. On reaching the house, he had recovered, and was seated on a chair, looking pale, and with a weak pulse. A small half-ounce bottle was shown me, labelled "mother tincture of camphor." He said he had been accustomed to apply two drops at a time into a decayed tooth for toothache; but, not finding relief, had repeated and increased the quantity several times on the present occasion, when suddenly, surrounded by a party of friends, he fell down insensible. Not being then aware of the strength of these homœopathic solutions, I attributed the effects to sudden fear lest he should have overdone the thing. After lying for some minutes unconscious, his friends told me he gradually came round. He had emptied the contents of the bottle. How much it contained I cannot say. The young man was in good health, had walked two miles to visit his friends, and was going to walk back.

'Frederick C. G. Ellerton, Lindley,
near Huddersfield.'

In the same journal, Feb. 27, 1875, I published the following:—

'*Case of Poisoning by Homœopathic Camphor.*

'I am indebted for the particulars of the following case of poisoning by homœopathic solution of camphor to my friend Mr. Gooch, of Eton. The following is Mr. Gooch's narrative:

'"About three years since, I was called to see an Eton boy, aged 14, who was supposed to have fainted. I found him lying on his bed, almost pulseless, and quite insensible, with

his extremities cold, and his face and lips pallid. He had shortly before had his dinner, and I thought that, perhaps, he might be suffering from an attack of indigestion. Whilst I was administering stimulants, and applying hot bottles, &c., to his feet, a servant accidentally found a small bottle on his table, which was labelled 'Rubini's Homœopathic Camphor'; and, thinking that he might have taken some of its contents, I gave him, with difficulty, an emetic. This had the immediate effect of causing him to vomit his dinner, mixed with an immense quantity of camphor, which scented the whole room most powerfully. In about half an hour he became sensible, and told me that he had a cold, for which he had purchased the camphor; but instead of dropping two or three drops upon a lump of sugar, as directed, he poured it out on the sugar, and after taking it, immediately became insensible. In the evening, all the effects of the drug had gone off; he passed a good night, and had no unpleasant symptoms afterwards.

'"He was a strong, healthy-looking boy. The bottle found in his room was very small, holding about half a drachm, and it was about half full when I saw it; so that he may have taken about fifteen drops of the solution, if an ordinary lump of sugar would hold so much."

'This is the seventh case of poisoning by the homœopathic concentrated solution of camphor that has come to my knowledge during the last two years. I suspect that, if all the cases of this kind that have come under the notice of the profession within the United Kingdom during the same period were published, the list would be a very long one, and it might serve as a warning of the danger which attends the incautious use of this highly concentrated poison.'

The following case was published by me in the *British Medical Journal*, May 29, 1877:—

'*Another Case of Poisoning by the so-called "Homœopathic Solution of Camphor."*

'I am indebted to my friend Mr. Philip Grubb, of Warminster, for the following notes of a case of poisoning by homœopathic camphor:—

'"On Wednesday, April 11, the following case occurred in my practice. A young gentleman, aged 18 years, reading for Oxford, of fair average health, in whose family no trace of hereditary tendency to epilepsy exists, for the cure of a cold took on sugar, between 6.30 A.M. and noon, seven doses of homœopathic camphor. Each dose, he says, was three drops, though I fully expect that he took more than three drops each time. Within five minutes after taking the last dose, without the slightest warning, he had a severe epileptic fit, in which his tongue was badly bitten. I did not see him during the actual convulsion, but from the description there can be no doubt as to its nature. It lasted more than fifteen minutes. He has been under treatment ever since the fit, feeling, as he says, 'queer,' complaining of a peculiar cold sensation on the tongue, extending for about half an inch from the tip. After the immediate effect of the attack went off, I put him on the bromide of potassium, which, however, did not seem to agree with him. I am now (May 7) giving him nux vomica, liquor potassæ, and infusion of cusparia; and he is all but well, though not yet quite what he was before this occurred. I enclose the two labels which were on the bottle containing the camphor.

'"'Saturated Spirit of Camphor, as used by Dr. Rubini. Ten times the strength of the ordinary Spirit of Camphor.'

'"'Concentrated Solution of Camphor. Dose: two or three drops on sugar every fifteen minutes; less frequently when relieved. Prepared by J. J. O. Evans, Chemist, and Agent for Homœopathic Medicines, Medical Hall, Teignmouth.'

'"It is right to mention that, about a month before the fit, my patient had a fall while hunting; but he declares that this left not the slightest trace of injury."'

In the *Times* of March 5, 1875, under the signature of 'A Physician,' I wrote to warn the public of the serious danger which is incurred by the incautious use of the so-called homœopathic preparation of camphor. The publication of this letter gave rise to some correspondence, the result of which is summarised in the following editorial article, which appeared in the *British Medical Journal*, March 13, 1875:—

'"*Poisoning by Homœopathic Solution of Camphor.*

'"The subject of poisoning by the homœopathic concentrated solution of camphor, to which the attention of the profession was first called by Dr. George Johnson's paper in the Clinical Society's *Transactions*, has during the last few days excited a more wide-spread interest in consequence of the *Times* having published on the 5th instant a letter by 'A Physician,' the object of which was to warn the public of the danger which is incurred by the incautious use of this concentrated poison. In Dr. Johnson's paper, five cases of poisoning by this strong solution of camphor are recorded; and four have been recently published in our columns. The slighter symptoms have been giddiness, faintness, headache, and drowsiness; but in five out of the nine cases a dose ranging from fifteen drops to a teaspoonful caused epileptic convulsions and more or less profound and alarming stupor; and it is probable that death would have resulted in more than one instance if a large proportion of the poison had not been speedily ejected by vomiting. The symptoms described are those which are well known to result from an overdose of camphor. Nevertheless, Dr. Bree, in a letter which appeared in the *Times* on the 6th instant, expressed a doubt whether camphor was really the cause of the symptoms, and gave as a reason for his doubt, that he had taken camphor in three-grain doses with no other than a beneficial effect. He had prescribed it for others in much larger doses; and 'in the British Pharmacopœia the dose is given as from one to ten grains, or what is equal to from two to twenty drops of the homœopathic solution.'[1] Dr. Bree's letter was promptly answered by 'A Physician' and by Prince Louis-Lucien Bonaparte, both of whom pointed out that Dr. Bree's error consisted in comparing camphor in the solid and dry state with camphor dissolved in spirit. It is a well-known fact that the action of a poison is, *cæteris paribus*, in direct proportion to its solubility. Camphor, being very insoluble in the fluids of the alimentary canal, when given in the form of a dry solid is in great part eliminated without

[1] Dr. Bree much under-estimated the strength of the solution, five minims of which contain four grains of camphor.

being absorbed, and has little medicinal or poisonous effect; but, when taken in the form of a spirituous solution, it is much more readily absorbed, and in a corresponding degree more active. The compilers of the Pharmacopœia apparently consider that camphor dissolved in spirit is at least three times as active as solid camphor; for, the dose of the latter being from one to ten grains, that of the spirit of camphor, which contains one grain of camphor in ten minims, is given as from ten to thirty minims. We have, then, no difficulty in understanding that this saturated solution of camphor, which contains more than a grain and a half of camphor in every two drops, should, in any dose above five drops, cause unpleasant symptoms; and that, in doses of fifteen drops and upwards, it should act as a strong poison. If the public could be made to understand that modern homœopaths have gone from the harmless extreme of infinitesimal dilution to the dangerous extreme of the greatest possible concentration of active and poisonous drugs, they would be more cautious in playing with these dangerous weapons. This homœopathic solution of camphor is as actively poisonous, drop for drop, as the prussic acid of the Pharmacopœia; and we maintain that any chemist who sells it without labelling it as 'poison' should be as liable to censure and to penal consequences as if he sold prussic acid, or the much less powerful laudanum, without a 'poison' label."'

On the same day and to the same purport the *Medical Times and Gazette* had a leading article on the subject, and the following week the *Pharmaceutical Journal*, in an editorial article, suggested that, 'after the evidence that has been given, there can be little doubt as to the advisability of using a "caution" or even a "poison" label in selling this dangerous preparation. This precaution, however, appears to be rarely if ever acted upon. I have never met with a "caution" label upon any bottle containing this poison. The printed directions are always vague, and not seldom, if acted upon, would lead to most disastrous consequences. I have a small collection of bottles with these misleading labels; one which was sent to me by my friend Mr. Parsons, of Bridgewater, who obtained it from a patient, has the following printed label: "The

Saturated Solution of Camphor (as advised by Dr. Rubini). Dose: four drops on a small lump of sugar every five minutes until the symptoms begin to yield. In very severe cases the dose should be increased from five to twenty drops every five minutes." '

Notwithstanding the public warnings which have been uttered against the incautious use of this dangerous preparation, I not seldom meet with patients who are in the habit of using it; it is probable, therefore, that cases of poisoning will, from to time, occur.

It is most important that every member of the profession should be aware of the alarming symptoms which may result from an overdose of this drug, and that he should be prepared to meet them by the prompt and thorough evacuation of the stomach.

One, and that not the least weighty, of the reasons for directing public attention to the grave symptoms caused by this homœopathic solution, is the consideration that the medical attendant may thereby be assisted to distinguish an epileptiform convulsion the result of a poison from an attack of true epilepsy, and may thus be enabled to reassure the patient and the patient's family and friends.

CHAPTER XVII.

CASES OF LEAD-POISONING FROM VARIOUS SOURCES.[1]

The sources of lead-poisoning are very numerous and sometimes difficult to trace. Of the three sources of this form of metallic poisoning illustrated by the following cases, the first—the use of whitelead as a cosmetic—has often been publicly referred to; but as the danger of the practice appears not to be so generally known as to prevent its continuance, I have thought it well to republish the first case with the others, which had a less well-known origin.

Case of Lead-Poisoning in a Ballet-Dancer, the Result of Using Flake-White as a Cosmetic.

Elizabeth R——, aged 22, married, was admitted into Twining ward on June 25, 1875, with well-marked symptoms of poisoning by lead. She states that four months ago her hands and arms began to be weak and unsteady in their movements. She found it difficult to write, and, in consequence of weakness of her legs, she could not walk so well as usual. She lost flesh. After a time she had pains in the abdomen, and constipation, and she had a coppery taste in the mouth when she first awoke in the morning. The weakness of the hands and arms continued to increase, and she noticed that the muscles of the thumbs and forearms were wasting. A few days before her admission the weakness of the hands and arms suddenly increased, so that she had very little power to grasp an object, and she was unable to extend the fingers.

On admission she was found very anæmic. The muscles of the forearms and of the thumbs were much wasted, the wasting being greater in the right arm and hand than in the left. There was complete wrist-drop on the right side, incomplete on

[1] *Medical Times and Gazette*, August 28, 1875.

the left. She was unable to extend the fingers of either hand. She could flex the forearms on the upper, and raise both arms above her head. There was some weakness and flabbiness of the muscles of the legs and a blue line at the edge of the gums.

Here was an unquestionable case of chronic poisoning by lead. Then the question arose, What was the source of the poison? She had worked as a milliner, her chief occupation being that of trimming hats. It did not seem probable that in doing this work she could have been exposed to the poison of lead. Then it came out that she, being the wife of a scene-shifter at Drury Lane Theatre, also acts as a ballet-dancer, going on the stage four or five nights a week; and as a preparation for this she has been in the habit of powdering her face with flake-white. This flake-white is mainly composed of carbonate of lead. My son analysed a portion of the powder which the patient had been accustomed to use, and he found it to consist of carbonate of lead, with a minute quantity of chalk. The symptoms present in this unfortunate woman are thus completely explained. When powdered whitelead is applied to the skin of the face it is partly absorbed through the skin, while much is probably inhaled as dust through the mouth and nostrils, and thus it enters the circulation and pervades the whole system.

Our patient tells us that a friend of hers, who is also a ballet-dancer, is suffering from the same symptoms as herself. There is nothing new in this: symptoms of chronic poisoning by lead have often been traced to the use of powdered white-lead as a cosmetic, and also to the habitual employment of hair-dyes containing lead. It would be well that all persons employing cosmetic powders should be informed that flake-white contains a poisonous compound of lead.

Case of Lead-Poisoning in a Portmanteau-Maker from Working with 'American Overland Cloth.'

Some time since I had under my care in the hospital a portmanteau-maker who had repeatedly suffered from symptoms of chronic lead-poisoning. Our search for the poison

was for some time unsuccessful, but, persevering with our inquiry, at length it came out that he was in the habit of working much with a material called 'American overland cloth.' He procured some of this for our inspection. It consists of canvas, covered rather thickly with a white coating, the surface of which is glazed and coloured black. This white coating was found by the late Professor Miller to be a mixture of chalk and carbonate of lead. When the cloth was cut, the white coating formed a cloud of dust. The man worked with this material in his own living- and sleeping-room; sometimes, as he admitted, eating his meals without washing the dust from his hands. In addition, he was in the habit of using the fragments of cloth as fuel for melting his glue, as a result of which some of the lead probably became volatilised, and entered the system through the lungs. Having thus traced the poison to its source, we were able to instruct the man how, in future, to avoid it.

Cases of Lead-Poisoning from the Use of Mincing-Machines.

In the *Lancet* of March 13, 1886, there appeared a letter from Mr. Chadwick Brown, who stated that during his twenty years' practice as a dentist he had often met with the blue line on the margin of the gums, characteristic of chronic lead-poisoning, amongst a class of intelligent well-to-do patients, who could not have been exposed to the poison of lead in the course of their ordinary avocations. He found that what was common to all these cases was that, having lost their teeth, they were in the habit of using a mincing-machine.

Mr. Brown then goes on to say:—'I next proceeded to examine a number of the machines in question at the houses of friends, in the shops, and at one of the exhibitions at the Agricultural Hall, and found that in all the mill was formed by a number of small wheel-like segments strung on a rectangular axis, and each bearing three rudely-cut, oblique teeth, working between a series of fixed plates or blades. The latter were of steel; but the former, I found to my surprise, were of pure lead, so soft as to be easily cut with a

knife or scratched with the thumb-nail, and leaving a black line when drawn along a sheet of paper; should they be pressed against one of the fixed blades, a thin film or shaving of lead would be worked off, and since meat when raw has always a slightly acid reaction from the presence of lactic acid, a certain amount of the lead must be liable to solution in the juice. Fats, too, though not containing any free acid, act in the same way on lead. At any rate, the use of lead in contact with moist and acidulous articles of food in a machine involving a considerable amount of friction cannot but be a source of danger to health, though it is only in the case of persons who use such machines daily that unmistakeable symptoms are produced; and I think that some substitute of a harmless metal or a hard wood ought to be insisted on.'

It appears to me that Mr. Chadwick Brown has done good service by directing attention to this, in addition to the other numerous sources of chronic lead-poisoning.

CHAPTER XVIII.

TWO CASES OF GRAVE NERVOUS DISORDER EXCITED BY A FOREIGN BODY BENEATH THE CICATRIX OF A WOUND.[1]

CASE I.

Traumatic Tetanus—Recovery after the Removal of a Foreign Body from a Wound in the Thigh.

H. N——, aged 13, a newspaper boy, was admitted into Craven ward on June 24, 1870. Three weeks before, whilst getting over a hedge, a pointed piece of wood pierced his trousers and wounded his thigh. He stopped the bleeding by tying a handkerchief round the thigh: the wound soon healed, and he is confident that no portion of the pointed wood remained beneath the skin. About nine days before his admission the lower jaw began to feel stiff; this gradually increased until it interfered with mastication. Then the muscles of the trunk and limbs began to be affected with painful spasms, and in this state he came into the hospital.

On his admission he was well nourished, with a healthy colour. There was a peculiar expression of face, resulting from spasm of the occipito-frontalis, corrugator-supercilii, and other facial muscles. He could separate the incisor teeth only to the extent of about half an inch. There was some rigidity of the abdominal and erector spinæ muscles, and movement of the body occasionally increased this rigidity to a painful degree of spasm, the pain being especially severe in the back; the spasm also implicated the muscles of the legs. The temperature, pulse, and respiration were normal.

At the upper third of the thigh there was a cicatrix about

[1] *Proceedings of the Royal Medical and Chirurgical Society*, 1871, vol. vi. p. 313.

half an inch long; the scar and the tissues beneath felt unusually hard, and pressure caused considerable pain. While examining the patient, I remembered that Dr. Taylor[1] had recorded two fatal cases of traumatic tetanus, in one of which there was found *after death* a piece of rusty iron beneath the cicatrix, and in the other a splinter of wood. Suspecting the presence of a foreign body beneath the tender cicatrix in this case, I requested the house-surgeon, Mr. Whitmore, after chloroform had been given, to incise the cicatrix. In doing so he discovered and removed a small dark mass. This proved to be a piece of woollen stuff from the boy's trousers, which had been driven in and lodged beneath the skin. The piece of wool was about the size of a small pea. A poultice was applied to the wound, and during the next twenty-four hours no medicine was given. There was continuous rigidity of the muscles, with occasional paroxysms of spasm and pain. He was now ordered to take fifteen grains of chloral hydrate, at first every four hours, afterwards at longer intervals. Between June 26 and July 9 he had twenty-four doses of chloral, amounting in all to 360 grains. The symptoms gradually subsided, the spasms became less frequent and less severe, the last slight attack of spasm occurring on July 13, the wound healed, and he was discharged cured on July 27.

The following case, it will be seen, was of a somewhat similar character:—

CASE II.

Trismus, with Facial Neuralgia and Palsy and a Recurrence of Epilepsy—Recovery after the Removal of a Foreign Body from a Wound on the Cheek.[2]

Henry M——, aged 44, a wheelwright, living at Wimbledon, was admitted into Craven ward on July 17, 1872. He stated that on July 4 he was cut on the left cheek by a blow from a metal axle which fell against his face. The wound bled freely; he washed it and then went to a druggist and had it strapped up. The cut soon healed, but the cicatrix remained painful. On July 11 the pain was very severe, and on the 12th ten times more so. On the evening of the 12th he had an epi-

[1] *On Poisons*, 3rd ed. pp. 96–7.

[2] *Transactions of the Clinical Society of London*, vol. xi. p. 38.

leptic fit. Here it may be mentioned that from the age of eight or nine he was for several years subject to epileptic fits. The first fit, he believes, was caused by his drinking too much beer in a hayfield on a hot day. The fits occurred on an average about once in eighteen months, but, until the occasion just now mentioned, he had been free from the fits for twelve years. On the morning after the fit (the 13th) he had some difficulty in opening his mouth and in closing the left eye. This difficulty continued and increased until his admission on July 17.

He had then the healthy look of a man of temperate habits, which he declared himself to be. There was palsy of the facial muscles on the left side; the features being drawn to the right, especially under the influence of emotion. If he attempted to close the left eye alone, it remained wide open, and, when he attempted to close both eyes at once, the left eyeball remained partially uncovered. He could not separate the incisor teeth more than one-eighth of an inch. The left masseter muscle felt hard and rigid, its hardness being but little increased by a voluntary effort to forcibly close the mouth. There was a scar about three-quarters of an inch long an inch below the left eye, which was very hard, tender, and painful when touched; and there was a feeling of numbness in the skin on the left side of the face. The urine was normal. When I saw him, the day after his admission, the symptoms remained the same.

Having a vivid recollection of the case above reported, I asked the patient if he thought it probable that dirt had got into the wound; to which he replied that he thought it very likely, for the metal axle which wounded him had its end covered with dirty grease. I then requested Mr. Birch, the house-physician, to cut through the cicatrix, and, this being done, a sharply angular piece of flint, nearly as large as a grain of wheat, was discovered and removed from the wound; it weighed half a grain.

Water dressing was applied to the wound and no medicine was given. During the next two days the symptoms remained the same; there was much pain in the cheek, and he slept badly. The temperature was normal. He was now ordered

to take twenty grains of chloral night and morning. From this time the symptoms gradually passed away. On July 23 he could separate the incisors to the extent of one-third of an inch; on the 26th to that of half an inch; and on the 29th, when he left the hospital to look after his business, the mouth opened to the extent of two-thirds of an inch, the facial palsy had diminished, but he was still unable to completely close the left eye. On August 2, when he came to show himself, he could close the eye, and again on the 9th he could separate the front teeth to the extent of an inch and an eighth, and he considered himself quite well, though there was still a slight trace of facial palsy. From that date he was not seen until September 26, when he came and reported that for some weeks he had been perfectly well, and no appearance of spasm or palsy remained.

In this case it appears that a jagged piece of flint in the cicatrix excited pain and numbness on that side of the face, paralysis of the muscles supplied by the portio dura, spasm of those supplied by the inferior maxillary division of the fifth nerve on the same side, and a recurrence of epilepsy after an interval of twelve years.

There can, I think, be little doubt that, small as was the offending body in each of these cases, the disease, in spite of any drug treatment, would have gone on to a fatal termination if the exciting cause had not been discovered and removed. The cases afford an instructive illustration of the principle to which I referred in my Introductory Address (p. 7)—namely, that 'one of the most important duties of the practitioner is the correct interpretation of symptoms, the object being to get behind the symptoms so as to ascertain their cause.' In both cases the chloral appeared to have a beneficial soothing influence after the removal of the local irritant.

CHAPTER XIX.

ON FOREIGN BODIES IN THE THROAT AND AIR-PASSAGES.[1]

False Teeth impacted at the Back of the Tongue—A Penny Coin in the Gullet of a Child—False Teeth impacted at the Lower End of the Œsophagus—An Impression of a Foreign Body in the Throat after its Removal—Sudden Death from Closure of the Larynx by a Piece of Orange—Suffocation by a Piece of Tendon of Meat under the Epiglottis mistaken for Apoplexy—A Button in the Larynx—A Sixpence across the Glottis—A Half-sovereign across the Glottis—A Buckle in the Larynx; Fatal—A Plum-stone in the Larynx—Symptoms of a Foreign Body in the Larynx—A List of Substances that have passed through the Glottis—Physical Signs of a Foreign Body in a Bronchus—Results of a Foreign Body in a Bronchus—Diagnosis—Foreign Body in the Trachea—Treatment of Foreign Bodies in various parts of the Air-passages—Case of Mr. Brunel—Precautions to be observed when attempting to dislodge a Foreign Substance by Inverting the Body of the Patient.

The subject of foreign bodies in the throat and air-passages is one of considerable interest and practical importance. It is one of those subjects which may be said to occupy a neutral territory between the domains of medicine and surgery. The physician is not unfrequently required to assist in the diagnosis of a doubtful case, and also to advise as to the expediency of an operation, while the surgeon is often called upon to operate for the removal of the foreign body or to open the windpipe for the relief of urgent dyspnœa.

A foreign body may obstruct respiration, occasion much distress, and even endanger life, by becoming lodged in the throat, without entering the air-passages. A very remarkable case of this kind has been recorded by Sir James Paget.[1] The following is an abstract of this instructive case. In July 1861, a gentleman, aged 60, had one evening a fit of faint-

[1] *Medical Times and Gazette*, Jan. 18, 1862.

ness or epilepsy. He wore sets of artificial teeth in both the upper and lower jaw. One set of teeth disappeared after the fit, and could nowhere be found. He sent for a medical man, who found him suffering from difficulty of swallowing and some dyspnœa; his tonsils, too, appeared enlarged and red. In a few days these symptoms abated, and he returned home. He then had increasing difficulty of swallowing, and a copious secretion of mucus, occasional vomiting, a short hacking cough, and once or twice he hawked up a little blood. There was not much pain, but a sense of constriction about the cricoid cartilage. The voice was rough and rather hissing, and his breathing was sometimes attended with a wheezing sound. Many times it was suspected that a foreign body was in the throat, but none could be seen; and when the lost teeth were spoken of, the patient declared that they were much too large a mass to have lodged in his throat, and that they must have been thrown away. The symptoms got worse, and in November 1861 he came to London to consult Sir James Paget. His feeble appearance and emaciated look excited a suspicion of cancer of the œsophagus. On examination of the mouth and fauces, Sir James Paget could see nothing unnatural, until, by extremely depressing the tongue, he saw something white near the epiglottis. Then, passing his finger to the side of the epiglottis, he felt teeth, and hooked out a whole set, consisting of nine teeth with a gold plate and fittings. The mass had been impacted between the base of the tongue and the back of the epiglottis, the teeth being directed upwards, and the notch in the palate-plate next to the root of the tongue. After the removal of the foreign body the patient soon regained his usual state of health; but he has since had a violent fit of epilepsy, during which his medical attendant remarked that if his teeth had not been removed he must have broken them to pieces. This occurrence elucidated the former mystery. Without doubt the previous displacement of the teeth had occurred during a fit of epilepsy. I learn from Sir J. Paget that the patient has since died of brain disease and epilepsy.

With reference to this case one remark suggests itself. If the throat had been examined by the laryngoscope, the teeth

must have been seen immediately after they were lost, and the patient might have been spared much suffering.

In the following case, which occurred in my own practice, the laryngoscope was used with advantage:—F. C. W——, a fine healthy boy, aged a year and eight months, was brought to me on May 2, 1867, by his parents, who told me that, two days before, he had swallowed a copper penny. When the child was taken to his mother by a servant immediately after the accident, he was black in the face; his eyeballs apparently starting out; and he seemed to be in imminent danger of suffocation. These alarming symptoms soon subsided; and when the medical attendant arrived, he supposed that the coin had passed into the stomach. The child's father, feeling alarmed and anxious, took him to one of the hospitals in the east of London, where the house-surgeon, after learning the history, said there was nothing to be done; but he desired that the patient might be taken to him again on the following day. When the child was brought to me, about fifty-two hours after the accident, I ascertained that, since the coin had disappeared, he had been quite unable to swallow solids, while the swallowing of liquids was attended with difficulty, and often excited coughing. Some water that I gave him to drink made him cough violently. There appeared to be much irritation about the throat, and there was a frequent discharge of salivary mucus from the mouth; this discharge was occasionally tinged with blood. Respiration was attended with a moist rattling noise in the throat. There were frequent fits of coughing, which almost entirely prevented sleep; the child looked weary and anxious, and the distressing symptoms had gone on steadily increasing. With such a history it could scarcely be doubted that the coin was impacted in the throat. Up to that time the only treatment had been the administration of a dose of castor oil. Using a piece of soft wood to keep the mouth open, I endeavoured to reach the coin with my finger; but I failed to do so. Then, while keeping the mouth open by the wooden gag between the teeth, I introduced a small laryngeal mirror. At first, I found that the surface of the mirror became instantly smeared over and dimmed by the profuse mucous secretion from the throat, so that I could see nothing

I next swept the mucus out of the throat by a brush on a bent wire; when, quickly introducing the mirror, I saw the coin sticking in the upper part of the œsophagus, the surfaces front and back, and the upper margin just below the opening of the larynx. I then took a pair of long, slender, curved forceps, opening front and back, and, guiding them by the throat-mirror which I held in my left hand, I seized the edge of the penny and brought it out. Immediately after the removal of the coin, the child retched and coughed violently for a few seconds. I then gave him some milk and water, and it was pleasant to see the eagerness and ease with which he drank it. From that time all symptoms of irritation rapidly subsided; but it was not until the third day after the removal of the penny that he could be induced to swallow solids. When I saw him again, a week after his first visit to me, he had lost all his discomfort, and he looked a model of health and happiness. It is probable that the alarming symptoms of suffocation which occurred immediately after the coin got into the throat resulted from the partial closure of the larynx, while the foreign body was sliding over the epiglottis on its way to the gullet. That the continued impaction of the coin in the gullet would have been speedily fatal by the extension of inflammation and swelling to the larynx scarcely admits of a doubt.

When a foreign body in the throat can be seen or felt, and reached from above, an endeavour should be made to extract it, and not, at first, to push it further down. In the summer of 1867 there was in the hospital, under Sir William Fergusson's care, a woman, who said that two months before her admission, four false teeth had become displaced and lodged in her throat during sleep. She was awakened by a cough, with pain in the throat and dyspnœa, and she was unable to swallow her saliva. The doctor, when he arrived, at once endeavoured to push the teeth down the gullet by a bougie, and he repeated the attempt day after day. On her admission the mass was found to be impacted at the lower end of the gullet; Sir William Fergusson failed to extract it by means of the longest forceps that he could obtain, and the woman went out unrelieved. It is probable that when the foreign body was in

the upper part of the gullet it might have been removed easily and speedily.

It is worthy of note that when a foreign body with an uneven surface has been caught in the throat and has scratched the mucous membrane, it often happens that an impression of the irritating substance being still impacted in the throat remains for a long time after it has been dislodged. In November 1866, a surgeon called and told me that, three weeks before, a piece of bone had got into his throat and excited spasm. He immediately pushed a bougie down his throat, then he excited vomiting two or three times by thrusting his fingers into his throat. An uneasy sensation had remained in the throat from that time, and he believed that the bone was still lodged there. On examination with the mirror I saw no foreign body, but the mucous membrane of his larynx was congested. This may have been partly caused by his somewhat rough treatment of himself, and partly it was a result of catarrh, from which he was then suffering. He was a nervous, anxious man, and I had some difficulty in persuading him that the bone was not then in his throat.

A *complete* closure of the larynx by a foreign body may cause very sudden death. On the table there is a specimen showing a piece of an orange impacted beneath the epiglottis, and completely plugging the larynx. This caused instant death.

A *partial* closure of the larynx may cause gradual apnœa with some of the symptoms of apoplexy, and the diagnosis may be doubtful. The following case occurred to a former pupil of this College, from whom I obtained the history :— An elderly man, an inmate of a prison, suddenly fell down while at dinner, and was supposed to be in a fit. When the surgeon arrived, he found the man blue in the face and breathing stertorously. Suspecting that there might be some foreign body in the throat, a probang was passed, and it was thought that something had been pushed down the gullet. The symptoms continued, and the man died in the course of the afternoon. After death a piece of tendon of meat was found under the epiglottis. There was an inquest, and the surgeon was blamed for not having performed tracheotomy,

which of course he would have done if he had known the real state of the case. Here again the laryngoscope might have shown the true cause of the symptoms.

A foreign body may enter the larynx, and remain there, causing much distress for a variable period. Some years ago, before the laryngoscope had come into use, the late Mr. Partridge had in the hospital a boy, about 5 years of age, who got a button in his larynx. There was aphonia, with stridulous and difficult breathing. Tracheotomy was performed, and an attempt made to detect and remove the button by passing a probe upwards through the wound. The button was never found, but the distress gradually subsided. The tracheal opening was allowed to heal, and he went out still suffering from hoarseness. Here, again, the laryngoscope would have been of use for diagnosis, and possibly for extraction. In the summer of 1867 I examined with the mirror the larynx of this patient, then a young man. The mucous membrane was thickened, and the true cords were red and uneven. This condition rendered him still rather hoarse.

Dr. Sanderson and Mr. Hulke have published [1] an interesting case which occurred to them at the Middlesex Hospital. On November 2 a muscular man, aged 27, in good health, was talking while he had a sixpenny-piece in his mouth. During the act of laughing the coin disappeared, and he immediately fell to the ground suffocated. A surgeon who was called in thought him dying, and took him to St. Mary's Hospital, where he arrived in three-quarters of an hour from the time of the accident. He said the throat was examined, but nothing found. Soon the breathing became easier. He remained there until one o'clock the next day, when he was discharged at his own request. His breathing was quite natural, but he could not speak above a whisper. He attended for a fortnight as an out-patient, having a sore-throat and some pain, and difficulty in swallowing liquids. His voice remained as before—whispering. He lost flesh and strength; but in other respects he was in fair health, and had no difficulty of breathing either on exertion or otherwise. On January 3 his breathing became suddenly embarrassed, and at

[1] *Medico-Chirurgical Trans.*, vol. xlviii.

twelve o'clock he was taken to Dr. Sanderson, who, suspecting that the coin was still in the throat, told the man to go to the hospital for laryngoscopic examination. On returning home, and going upstairs, he was suddenly seized with a feeling of suffocation as urgent as immediately after the accident. He rushed downstairs in agony, and in so doing tripped; thereupon the dyspnœa suddenly ceased, and he was again able to breathe as freely as ever. Next day, at 2.30, he went to the hospital. The breathing was only slightly embarrassed. The sixpence was at once seen lying flat across the glottis, the edge in the ventricle on either side. Behind the edge of the coin there was a breathing space between the arytenoid cartilages. The mucous membrane was red and puffy. It was considered impossible to seize the coin with forceps from above, on account of its horizontal position. Bent loops of wire were prepared for the purpose of jerking it out. Attempts were made to dislodge the coin by holding the man's head downwards and slapping the back of his neck, while he was directed to cough, but could not, on account of his inability to fill the chest. The respiratory murmur was everywhere inaudible. Then it was resolved to perform tracheotomy. This was done under chloroform, and an attempt was made to seize the coin by forceps introduced from below through the opening. The coin could be felt but not seized. The forceps passed behind it through the glottis, and could be felt by the finger introduced through the mouth. The coin was at length pushed upwards within reach of the finger, by which it was drawn forwards over the epiglottis on to the root of the tongue. At this moment, by a sudden gulp, the coin was swallowed, and the breathing was free. The next morning it was passed by stool. The wound healed, and recovery was complete.

In December 1870, my colleague, Mr. Henry Smith, had under his care a hospital patient in whose larynx a half-sovereign was impacted. The man, while intoxicated, having placed a half-sovereign in his mouth, the coin disappeared, and the patient was suddenly seized with dyspnœa, which for a time was urgent. Four days afterwards he came under Mr. Smith's care. Meanwhile he had continued to suffer from pain in the throat, loss of voice, difficult and stridulous

breathing, dysphagia and cough, with copious mucous expectoration. Asked by Mr. Smith to make a laryngoscopic examination, I had no difficulty in demonstrating the presence of the coin in the exact position of the sixpence in Dr. Sanderson's and Mr. Hulke's case, to which I just now referred. The metallic surface formed a striking contrast with the congested and swollen mucous membrane by which the margins of the coin were overlapped. So tightly was it impacted that it would evidently be impossible to dislodge it by placing the man head downwards. With Mr. Smith's concurrence I attempted to seize it with the laryngeal forceps, but, although I could touch the surface of the metal, I found it impossible to grasp its margin. I then suggested to Mr. Smith that if he made an opening below the glottis he might push a bent probe against the under surface of the coin, and so thrust it into the mouth. Accordingly Mr. Smith made an opening in the crico-thyroid membrane, introduced a strong silver probe, and, using considerable force before the coin could be dislodged, he pushed it into the man's mouth, whence it was immediately ejected. The operation was very neatly and cleverly done in the space of about three minutes, and the man made a rapid and complete recovery.[1]

In a case which occurred some years since at Birmingham, a buckle having remained eleven months in the larynx of a child, ultimately caused death. The case is recorded as follows:—'Mr. Oliver Pemberton read notes of a case of foreign body in the larynx, and exhibited the *post-mortem* appearances of the same. The child, seven years and a half old, came under Mr. Pemberton's care in August last. She was said to have swallowed in the September of the previous year a portion of a japanned buckle. She was seen by a surgeon within half an hour of the accident, when she presented an anxious appearance, and suffered from difficulty of breathing and loss of voice. There was no difficulty in deglutition. The symptoms began to abate, and when Mr. Pemberton saw

[1] See Mr. Smith's history of the case, with a wood-cut, *British Medical Journal*, Jan. 7, 1871, p. 7. On the same page of the journal Mr. Pridgin Teale records a case in which a piece of bone, having been impacted in the larynx, was removed through an opening in the trachea.

the child she was healthy, and suffered no inconvenience beyond the weakness of voice; there was no stridor; and no abnormal physical signs could be observed upon auscultation of the lungs or of the larynx. On account of the absence of urgent symptoms laryngotomy was not deemed advisable, and the child returned home. Three days after her return she was seized with violent vomiting from an overloaded stomach. This probably stirred the foreign body, set up inflammation of the laryngeal mucous membrane and caused death. The body of the buckle, an inch long, was found lying vertically in the larynx posteriorly, while the teeth lay in the angle of the thyroid cartilage.'

Dr. Stokes [1] gives the curious case of a boy who made a whistle of a plum-stone by perforating it on each side and removing the kernel. While he held this between his lips it passed, during a deep inspiration, into the larynx, and became fixed there. So little inconvenience did this occasion at first, that, on finding he could still whistle through the stone, he went about for some hours, pleased with this new and convenient mode of blowing his whistle. He then got paroxysms of suffocative cough, with pain in the throat and epigastrium, a bloated countenance, and a quick pulse. The chest was clear on percussion, and the respiratory murmur natural. The whistling in the larynx sufficed for the diagnosis, and tracheotomy was performed. During the performance of the operation the boy declared that he had coughed up the stone and swallowed it. The symptoms were relieved, and the whistling had ceased, but as the wound closed the distress and the whistling noise returned, proving that the stone had remained in the larynx, and that the noise had ceased only in consequence of air having been admitted below. Soon after, it was found to change its position and to pass down the right bronchus, again to be driven upwards into the larynx. By a second operation it was extracted, and the boy recovered without a bad symptom.

It has usually been observed that when the foreign body has become impacted in the larynx the symptoms from the first have been violent and distressing. The ordinary

[1] *Diseases of the Chest*, p. 291.

symptoms have been incessant spasmodic cough, croupy breathing, hoarseness, or complete aphonia, pain in the region of the larynx, and fits of suffocative dyspnœa. The termination of the case may be by sudden death, in consequence of closure of the glottis; or the foreign body may be expelled, or it may fall into the trachea. Then there may be an interval of comparative ease, succeeded either by a return of laryngeal symptoms, if the body is movable and light enough to be coughed upwards to the larynx, or, if it remains impacted in the bronchus, symptoms of inflammation of the lung may supervene.

An inspection of the widely open state of the glottis during deep inspiration shows that bodies of considerable size may easily pass between the vocal cords. A curious variety of substances have passed 'the wrong way' and got into the bronchi. Thus the root and fangs of a tooth, an artificial tooth, a piece of bone in several instances, a cherry-stone and a plum-stone in several instances, a piece of flint, a button in two cases, an ear of grass in two cases, an ear of rye, a Portugal ducat, a half-sovereign (Mr. Brunel's case), a sixpence, a shilling in two cases mentioned by Sir Thomas Watson, a piece of slate-pencil, a nut, a piece of nutshell, a piece of walnut-shell, a piece of nutmeg, a piece of cheese, a glass bead, a leaden shot one-eighth of an inch in diameter, an iron nail, a metallic screw, a kidney-bean, &c.[1]

A foreign body, having passed down the trachea, usually enters the *right* bronchus. The reason is that the right bronchus is larger than the left, and the septum between the opening of the bronchi is inclined to the left.

The *physical signs* of a foreign body in the bronchus are—natural resonance on percussion, with total absence, or feebleness of the respiratory murmur over one lung, according to

[1] The lower animals are sometimes killed by foreign bodies in the air-passages. Some time since I was staying in the country with a friend when one of his harriers died suddenly while eating his dinner of raw horse-flesh. The keeper who was looking on, saw the dog snap at one of the other hounds. Immediately after this the animal fell on the floor, was convulsed, and quickly died. I was present at the inspection. A piece of flesh about an inch long and as thick as one's thumb was found to have been arrested at the bifurcation of the trachea, so that the orifice of each bronchus was almost entirely blocked.

the degree of obstruction of the bronchus; diminished mobility of the obstructed side; and exaggerated, puerile breathing over the unobstructed lung. The bronchus will be obstructed more completely by a body of a globular form than by one which is flat or of irregular outline. In the case of Mr. Brunel, a half-sovereign having fallen into the right bronchus, careful auscultation by Dr. Chambers and others discovered no difference in the respiratory sound over the two lungs. If the foreign body be movable so as to be coughed upwards towards the larynx, the respiratory sound returns for a time over the previously obstructed lung. In some cases there is the alternation of the physical signs of obstruction of the bronchus, and distressing spasm of the glottis when the body is driven up to the larynx. This is especially apt to occur with foreign bodies of low specific gravity, such as vegetable substances; it is less likely to happen with heavy metallic substances, except when the patient's body is inverted. High specific gravity of a foreign body, however, does not of necessity prevent its being expectorated. Thus, in one case a leaden shot was expectorated; in another, a Portugal ducat; in another, an iron nail; in another, an artificial tooth;[1] in another, an elongated glass bead (case in the hospital, to be presently related); and in another, a sixpence.[2] It is probable that in some of these cases the foreign body has been coated with mucus, which would of course lessen its specific gravity. In a case recorded by Dr. Spitta, to which I shall hereafter refer, a metal screw encased in mucus was easily expectorated by a child.

The foreign body, having entered one or other bronchus, may occasion scarcely any inconvenience, more especially if its shape and size are such as not to entirely close the tube. This was the case with a boy under Sir William Fergusson's care in the hospital a few years ago, who had a long glass bead in his left bronchus, as shown by comparative feebleness of respiratory murmur on that side. As there were no symptoms of suffering in the lung or elsewhere, nothing was done. He went home to Harrow, and after several months he one day gave a cough, and said, 'Oh, here is my bead.'

[1] Stokes, p. 283.

[2] Mr. Cock's case, quoted in Chelius's *Surgery*, vol. ii. p. 398.

In the majority of cases, however, there are symptoms of distress. The mere obstruction of a bronchus may cause urgent dyspnœa, by suddenly and greatly lessening the vital capacity of the lung. Then usually there are symptoms of irritation and inflammation of the lung, pain, dyspnœa, cough with blood-tinged expectoration, high temperature, and quick pulse. On physical examination there may be dulness on percussion, and crepitation. Most of these symptoms were present in the case of a boy who, a few years ago, was in the hospital, under the care of Mr. Henry Smith, and, when the boy died, the lower lobe of the right lung was found engorged, and in parts consolidated and suppurating, with a plum-stone in the main bronchus. One interesting phenomenon I observed in that case. The boy one day had a sudden increase of dyspnœa, which continued for some hours, and then rapidly subsided. During the height of his distress, I found signs of obstruction over the *left* lung, which had before been free, and more air than previously was heard entering the *right* lung. The stone must have shifted its position. It had probably been coughed into the trachea, and then fallen back into the *left* bronchus, so that the increase of distress was due to the obstruction of the sound lung while the other was in a state of inflammation. In a few hours, the urgent dyspnœa subsided, and the signs of obstruction of the left bronchus had ceased.

The cerebral circulation often suffers in consequence of the violent cough, the defective aëration of the blood, and the resulting impediment to the circulation. Convulsions have occurred, and even apoplexy in old persons. In other cases the brain may be so injured that death takes place with cerebral symptoms, even after the removal of the foreign body.

One result of obstruction of a bronchus may be acute emphysema of portions of unobstructed lung from forcible over-distension.

The ultimate result of the inflammation set up by a foreign body is very variable. It may destroy life within a few days, or the inflammation may subside, and the body may remain for months, and even years, in the bronchus without exciting violent symptoms. The ear of grass on the table

was expectorated, encased in mucus, four months after it had entered the trachea of a child who recovered. Sir Thomas Watson quotes the case of a lady in which an ear of barley was expectorated seven years after its entrance. During that period she had suffered repeated attacks of hæmoptysis. Her recovery was perfect. In a case referred to by Dr. Stokes a piece of bone was expectorated, with copious hæmoptysis, after fifteen years. The patient recovered (p. 280). In one case, quoted by Dr. Gross, a piece of bone entered the windpipe of a child aged three, and was expelled sixty years afterwards. The symptoms were protracted cough, pain in the right side of the chest, purulent expectoration, and hæmoptysis. In another case, a piece of bone was expectorated after having remained seventeen years in the air-passages. Some relief occurred; but hectic and emaciation followed, and death in a year and a half afterwards.[1] Other cases are recorded, in which the symptoms of pulmonary disease have continued long after the expulsion of the foreign body, and in some with a fatal result.

On the table there is a preparation showing an ear of rye in an abscess which is common to the lung and the liver through the diaphragm. Sir Thomas Watson gives the following history from Mr. Mayo:—'The young son of an English nobleman was riding in a carriage, and had an ear of rye in his mouth. The carriage made a sudden jolt, and the ear of corn disappeared. Little was thought about this at the time, but soon afterwards symptoms of pulmonary irritation set in, attended with hectic fever, and with the most fetid expectoration. The boy gradually sank.' Dr. Spitta has recorded the case of a child, aged two years and a half, through whose larynx a metallic screw passed into the trachea and right bronchus, where it set up alarming inflammatory mischief. A month and two days after the accident the screw was coughed up, completely encased in mucus, and the child rapidly recovered.[2]

Diagnosis.—The chief diagnostic signs and symptoms in cases of a foreign body in a bronchus are—1. Signs of obstruction of one bronchus, usually the right, either

[1] Stokes, p. 283. [2] *Lancet*, September 16, 1876.

partial or complete, and either persistent or intermitting. 2. Physical signs of obstruction of the bronchus, alternating with symptoms of laryngeal irritation and spasm. There may be a sudden rush of air into the lung when the foreign body is coughed up towards the larynx, and an equally sudden obstruction of the bronchus when the body falls back again. 3. Signs of irritation and inflammation of the lung may be present, but when the bronchus is completely obstructed there may be no moist sounds or respiratory sounds of any kind audible over the obstructed portion of lung. 4. The symptoms already mentioned occur suddenly in a person either previously healthy or labouring under symptoms of a different class.

Usually a foreign body passing through the larynx goes into one or other bronchus, but Dr. Stokes quotes one case in which the trachea was obstructed (p. 288). A gentleman aged 20, previously in good health, while conversing in the act of eating a piece of cheese after a hearty dinner, suddenly fell from his chair in a state of insensibility. On the supposition that a foreign body had become fixed in the œsophagus, a probang was speedily passed, and after about ten minutes he partially recovered. Soon after, the attack recurred with great violence; the face was much congested, and the breathing spasmodic and stertorous. He was then bled twice without relief. A loud rattling in the throat came on, the patient tossed his arms about, and all the muscles of inspiration were in violent action. The surface of the body became pale and cold. Hours had now elapsed, and it began to be suspected that the case was one of tracheal obstruction. The chest was normally resonant; but the vesicular murmur was everywhere extremely feeble, notwithstanding the violent efforts at inspiration, and there was an increasingly loud rattle in the trachea. Tracheotomy was performed, a crucial incision being made through the tube, and the angles cut off, when suddenly a mass of pultaceous matter was ejected through the opening, with complete and instantaneous relief to the symptoms. Respiration became easy, the expansion of the lung full and audible, the patient breathed through the glottis, and recovered without a bad

symptom. In about four weeks, however, he had some symptoms of cerebral irritation, and a fit resembling epilepsy. During the next two months these attacks recurred several times, becoming gradually less severe. They then ceased entirely, and he has had no return. They were probably a result of the disturbed cerebral circulation during the hours of apnœa.

Sir B. Brodie refers to two cases in which a foreign body was found in the trachea after death.[1] In one case the foreign body was the berry of the bladder-senna, about the size of a large pea. The patient was a child aged 6. In the other a portion of the claw of a lobster was found in the trachea of a child aged 12.

It is obvious that a body sufficiently large to obstruct the trachea must cause more urgent distress than results from the obstruction of a bronchus. In such a case the respiratory phenomena will be the same on the two sides, and the diagnosis will depend on the absolute feebleness of the respiratory sounds over the entire chest, and not on the comparison of the respiration over the two sides.

In some cases the foreign body, when movable in the trachea, has been heard to strike against the larynx when it was driven upwards by a forcible expiration. A plum-stone in one case and a bean in another was thus audible.[2] In one case an orange pip, and in another a bean, was *felt* to strike against the larynx at each expiration when the finger was placed on the trachea.[3]

It should be borne in mind that the impaction of a foreign body in the *œsophagus* may compress the posterior membranous wall of the trachea, and cause the same symptoms as if it were actually in the trachea. In a doubtful case, therefore, a bougie should be passed down the œsophagus. Dr. Stokes mentions one case in which a piece of money lodged in the œsophagus caused croupy breathing and other laryngeal symptoms (p. 265), and another case in which such urgent

[1] *Med.-Chir. Trans.*, xxvi. 293–4.

[2] Ryland *On Diseases of the Larynx*, pp. 283–4.

[3] Report of two cases by Mr. Couper, of the London Hospital, *British Medical Journal*, February 12, 1870, p. 153.

symptoms were produced by a plum-stone in the œsophagus that his first impulse was to perform tracheotomy with his penknife. An œsophageal bougie was introduced, and the substance having been pushed into the stomach, the symptoms ceased, and a day or two after the plum-stone with which the child was known to have been playing previous to the accident was voided by stool.

We have now to consider the general rules to be observed in the *treatment* of cases in which a foreign body is lodged in the air-passages.

A foreign body in the larynx is a continual source of danger; therefore it should be removed as speedily as possible, even though the present symptoms may not appear alarming or very distressing. By the aid of the laryngoscope a foreign body anywhere above the glottis may not only be seen, but in most cases removed by properly constructed curved forceps. If, in consequence of its shape or position, it cannot be removed from above, an opening below—either laryngotomy or tracheotomy—may enable the surgeon, as in Mr. Smith's case before mentioned, to push the coin or other foreign body up into the mouth by means of a bent probe.

It may be possible, by means of the laryngeal mirror, to see a foreign body in the upper part of the trachea, and thus to confirm the diagnosis. If the obstruction is in the trachea, and if the symptoms are urgent, no time should be lost in opening the trachea, when the body will probably be spontaneously expelled, or it may be extracted by forceps.

A foreign body having passed into the bronchus, what is to be done?

It should be borne in mind that foreign bodies of various kinds have been expectorated without surgical aid. For instance, the bead in the case of Sir William Fergusson's patient. Sir Thomas Watson mentions the case of a boy who, after several weeks, coughed up a small nail; and in Dr. Spitta's case, before mentioned, a metallic screw was similarly ejected. Tracheotomy, therefore, is not to be resorted to immediately and as a matter of course in all cases. There are, however, three classes of symptoms which call for im-

mediate surgical interference. These are—1st. *Persistent and urgent dyspnœa*, resulting from the lessened vital capacity of the chest occasioned by the obstruction of a main bronchus. 2nd. *Paroxysmal dyspnœa*, resulting from spasm of the glottis occasioned by the foreign body being from time to time coughed upwards against the larynx. 3rd. *Symptoms of irritation and inflammation of the lung*, which, if allowed to continue, may speedily lead to fatal disorganisation.

Tracheotomy in the case of a foreign body in a bronchus may be a means of relief in various ways. 1. It may allow of the expulsion of the offending substance by an effort of coughing. 2. By means of long and slender forceps the foreign body may be extracted from the bronchus.

Mr. Liston once succeeded in extracting a piece of bone from the right bronchus of a woman aged 38, after it had been fixed there more than six months. The removal of the bone gave immediate relief, and the patient left the hospital well in nine days.[1] In another case a button was removed from the right bronchus of a boy aged eight.[2]

The facility of grasping a foreign body will depend much on its form. A spherical body, or one nearly spherical, would probably be in close contact, on all sides, with the bronchus, and could not be seized by forceps without risk of injuring the bronchial tube. A flat body, like a coin, offers the greatest facility for extraction, and is also one little likely to be expelled by coughing, on account of its density, and the small surface presented by its edges to the expiratory current of air.

In the case of Mr. Brunel, Sir B. Brodie and Mr. Aston Key failed to seize or even touch the half-sovereign, which had entered the right bronchus. The contact of the forceps with the internal surface of the trachea induced violent coughing, with convulsive action of the diaphragm and abdominal muscles, so that it was not deemed prudent to persevere, and the coin was ultimately got rid of by a process illustrative of a third mode in which tracheotomy affords relief in this class of cases.[3] The following is an abstract of the remarkable and instructive case of Mr. Brunel, to which I have before

[1] Ryland, *op. cit.* p. 300. Also Liston's *Practical Surgery*, p. 415.
[2] Chelius's *Surgery*, vol. ii. p. 402. [3] *Med.-Chir. Trans.*, xxvi. p. 286.

referred:—Mr. Brunel, on April 3, 1843, being engaged immediately after dinner in amusing some children, placed a half-sovereign in his mouth. It slipped behind the tongue, and a violent fit of coughing, in which he had the appearance of being nearly choked, was the consequence. This was immediately followed by vomiting. In the course of the evening he coughed at intervals, and a sense of soreness and stiffness of the throat remained for twenty-four hours. During the next two days he experienced little or no inconvenience. Then he was exposed to cold; had cough, expectorated mucus tinged with blood, and experienced pain in the situation of the right bronchus. These symptoms continued. Dr. Chambers and Sir B. Brodie were consulted, and agreed that the half-sovereign was in the right bronchus. This opinion was confirmed by an experiment which Mr. Brunel made on himself. He had placed himself in a prone position, with his sternum resting on a chair, and his head and neck inclined downwards, and, having done so, he immediately had a distinct perception of a loose body slipping forward along the trachea. A violent convulsive cough ensued. On resuming the erect posture, he again had the sensation of a loose body moving in the trachea, but in the opposite direction—that is, towards the chest. This experiment was afterwards repeated by the physician and surgeons. On April 25 he was strapped in the prone position on a platform made movable on a hinge in the centre. By this means the head was lowered to an angle of about eighty degrees with the horizon. At first no cough ensued, then the back of the chest was struck by the hand and he began to cough violently. This process was twice repeated, and on the last occasion the cough was so distressing, and the sense of choking so alarming, that it was thought imprudent to repeat the experiment unless some precaution were used to render it safe. On April 27 tracheotomy was performed, the object being twofold: first, to extract the coin with the forceps if possible; second, that the artificial opening might prevent the risk of suffocation by spasm of the glottis when the body was again inverted. I have already stated that the coin could not be grasped or even touched by the forceps. On May 13—the wound in the trachea having been kept open by the occasional introduc-

tion of a probe—the patient was again placed on the platform and inverted. The back was struck with the hand; two or three efforts to cough followed, and presently he felt the coin quit the bronchus, striking almost immediately afterwards against the incisor teeth of the upper jaw, and then dropping out of the mouth. There was no spasm of the glottis, nor any of the distress which caused so much alarm on the former occasion. The patient quickly recovered.

Sir T. Watson refers to two cases in which, a shilling having passed through the larynx, the coin fell out when the body was inverted. In both cases the patients were men, and in neither case was tracheotomy performed.

There is one point of practical importance to be borne in mind when an attempt is made to dislodge a foreign body from the trachea or bronchi by placing the patient's head downwards. It is this—that at the moment of the inversion the patient should be directed to take *a deep inspiration.* In so doing he opens wide the glottis, and thus facilitates the exit of the foreign body. The inspiratory current of air would have no appreciable effect in retarding the movement of the foreign substance downwards by its own gravity, and it is essential for its escape that the glottis be widely open. The patient should be cautioned to make no noise in his throat at the moment of inversion. Any vocal sound of necessity implies a closed glottis, which would of course prevent the exit of the foreign body.

ADDENDUM.

After the publication of the preceding lecture Mr. George Padley, of Swansea, wrote to the *Lancet* (November 1878) and expressed his opinion that the plan adopted in Mr. Brunel's case, of fixing the patient on an inclined plane in the *prone* position, involves two errors. 'First, the aperture of the glottis being triangular (or nearly so) with the apex forward, the coin in the prone position of the body would gravitate towards this, the narrowest part of the opening, and would therefore have less easy exit; secondly, strapping the patient to a platform would, if the coin or other substance failed to be

at once expelled, effectually prevent him, by his own instinctive effort, from assuming the upright position, and, by thus causing the coin to fall back into the bronchus, putting an end to the spasm of the glottis and the alarming symptoms consequent upon it.'

Mr. Padley then gives a practical illustration of the plan which he recommends.

A man came to him with a sixpence in his bronchus. 'A strong bench having been fixed, with the legs of one end on a couch, and those of the other end on the floor, the patient was made to sit upon the upper part of it with his knees placed over the end. He was then directed to lie back upon the inclined plane thus formed, and, having done so, he instantly raised himself up with the sixpence in his mouth.

'The supine position probably favoured the exit of the coin, and the readiness with which by his own effort, the knees acting as a fulcrum, he regained his upright position would have saved him from the danger of spasm in the event of its non-expulsion. If by this proceeding the opening of the larynx or trachea by operation could be avoided it would of course be a great advantage.'

In my opinion, Mr. Padley's simple and rational method should be adopted in the treatment of this class of cases.

CHAPTER XX.

A LECTURE ON THE RELATION BETWEEN CROUP AND DIPHTHERIA.[1]

Origin of the term Diphtheria—Historical Account of the Disease from Aretæus to Bretonneau—Distinction between Membranous Croup and Catarrhal Laryngitis—Diphtheria often co-exists with other Diseases—Three distinct Diseases to which the term Croup has been applied—Cases of Diphtheria complicating other Diseases, Surgical Wounds, and Accidental Injuries.

GENTLEMEN,—Some of you probably will remember that, at the beginning of the year 1870, the relation between croup and diphtheria formed the subject of discussion in the *British Medical Journal*—a discussion which had its origin in a paper which I there published *On the Morbid Anatomy of Croup and Diphtheria*.[2] In that paper I expressed my belief that, while most English writers on croup had confounded together two essentially distinct diseases—namely, simple catarrhal laryngitis and the specific diphtheritic membranous laryngitis—the true doctrine is that of the French pathologists, who teach that membranous croup is always diphtheritic, and that laryngitis from mere exposure to cold never results in the formation of false membrane.

At the commencement of the year 1875, the *Lancet* published simultaneously a lecture by Sir William Jenner *On Croup and the Diseases that resemble it*, and a paper of mine *On Certain Points relating to the Etiology, Pathology, and Treatment of Diphtheria* (*see* Chapter XXI.). In that paper, I reiterated my former statement that membranous croup is no other than laryngeal diphtheria; and Sir William Jenner stated that his opinion had undergone some modification. He said: 'I am

[1] *British Medical Journal*, Sept. 18, 1875. [2] *Ibid.*, Jan. 1, 1870.

inclined now to the belief that there is no such disease as idiopathic simple membranous inflammation of the larynx. I say I am inclined to that belief. I am not sure that it is true; but, as I formerly thought that the weight of evidence was in favour of their non-identity, I now incline, from my further experience, to think that the two diseases are really identical; that the so-called croup' (that is, membranous croup) 'is really diphtheria.' The simultaneous statement of this pathological doctrine by Sir William Jenner and myself appears to have excited considerable interest, and has given rise to a prolonged discussion in the columns of the *Lancet*. Latterly, too, several communications on the same interesting subject have appeared in the *British Medical Journal*. I propose now to set forth briefly the main facts relating to this subject, which appear to me to be established almost beyond controversy.

In the first place, then, it is an unquestionable fact that, although the name *diphtheria* is a modern invention of Bretonneau, the disease which we now recognise by that appropriate designation has existed for centuries. Bretonneau, in his first memoir on *Diphtheria*, translated by the New Sydenham Society, gives an interesting historical account of the disease, and shows that Aretæus describes it, and speaks of it as being so common in Egypt and Syria, that it had received the name of the Egyptian or Syriac ulcer. Two or three centuries later, the same disease is described by Aëtius; but, to come nearer to our own time, ever since the end of the sixteenth century, the disease now recognised as diphtheria has shown itself frequently in Spain, Italy, France, England, Sweden, and in America. Dr. John Starr, in the *Philosophical Transactions* (1749–50), describes under the name of *morbus strangulatorius* a disease which he says 'has within a few years reigned in several parts of Cornwall with great severity.' In his description we recognise the unmistakable features of diphtheria. He speaks of a 'white body seen on the palate and tonsils,' and gives a woodcut illustration of a membranous cast of the larynx, trachea, and primary bronchi, which was expectorated by one of his patients. He also describes the formation of a white membrane on blistered cutaneous surfaces.

Huxham, in his *Dissertation on the Malignant Ulcerous Sore-throat*,[1] evidently confounds together the two diseases, scarlet fever and diphtheria. We recognise diphtheria in his description of ash-coloured spots on the tonsils, uvula, palate, and pharynx, in the noisy breathing resulting from the extension of the disease in the air-passages, and in the expectoration of portions of membrane from the windpipe. He speaks of the discharge from the nostrils as being so acrid that it excoriated the lips and hands of the patients, and the fingers and arms of the nurses. He believes that the 'rheum' from the throat and nostrils, being swallowed, caused griping pains, diarrhœa, and excoriations of the anus and buttocks. While passing into the air-passages, 'the windpipe itself was sometimes much corroded by it, and pieces of its internal membrane were spit up.'

In the first volume of the *American Philosophical Transactions*, originally published in 1771, Dr. Samuel Bard gives an admirable description of diphtheria, under the name of *angina suffocativa, or sore-throat distemper*, as it appeared at New York. He describes the membrane on the tonsils as being frequently, but not invariably, present. He speaks of the membrane in the air-passages as being 'preternaturally thickened mucus,' without much inflammation; and he contrasts this with a case in which, death having resulted from 'a very violent inflammation of the internal membrane of the trachea, there was no such mucous lining to be discovered upon it.' He describes the formation of membranes on the abraded skin; and he recognises the infectiousness of the disease, but rather, as he says, 'from the breath of the infected persons,' than from 'any generally prevailing disposition of the air.' And this, he says, explains the fact that a whole family may suffer from the disease while the next-door neighbours escape. He speaks of one family in which seven cases occurred, three of which were fatal.

In the year 1765, Dr. Home published his treatise on the *Nature, Cause, and Cure of Croup*. The word 'croup' had long been in popular use in Scotland to designate a disease, or a group of diseases, attended with the symptom of noisy

[1] Third edition, 1759.

breathing. Dr. Home was the first to describe a membrane lining the air-passages as the essential anatomical character of croup. A careful study of his cases clearly shows that under the name of croup he included two diseases essentially different—namely, simple laryngitis from cold, and the specific disease diphtheria. Most of the cases that recovered belonged to the former class, while the fatal cases were diphtheritic. His two first fatal cases of croup with false membrane occurred in a brother and sister aged respectively 7 and 5 years, who died within a few days of each other. The fact of two fatal cases occurring thus in quick succession would alone excite a suspicion that the disease was diphtheria. In the second case, there was 'purulent' expectoration; 'the amygdalæ were a little swelled and covered with mucus;' and after death, besides the false membrane in the trachea, the back of the tongue was covered with mucus, and 'all about the glottis was covered with tough viscid mucus.' It can scarcely be doubted that these cases of so-called croup were really diphtheritic.

Cullen, in his *First Lines of the Practice of Physic*, 4th ed., 1784, in describing *cynanche trachealis*, refers to Home as 'the first who has given any distinct account of it;' and, after describing the purulent and sometimes membranous expectoration, says that, 'when the internal fauces are viewed, they are sometimes without any appearance of inflammation, but frequently a redness and even swelling appear; and sometimes in the fauces there is an appearance of matter like to that rejected by coughing.' Under the head of *cynanche maligna*, Cullen evidently includes scarlatina anginosa and diphtheria. The latter cases are indicated by the appearance of white or ash-coloured spots on the fauces, and by the fact that 'the larynx and trachea are often affected in the same manner as in the cynanche trachealis;' as a consequence of which, 'the cynanche maligna often proves fatal by such a sudden suffocation as happens in the proper cynanche trachealis.' But he adds: 'There is reason to suspect that dissectors have not always properly distinguished between the two diseases.'

Mr. Henry Rumsey, in *An Account of the Croup as it appeared in the Town and Neighbourhood of Chesham, in*

Buckinghamshire, in the Years 1793 *and* 1794,[1] says that 'frequently large films of a white substance were formed on the tonsils,' evidently what we now call a diphtheritic exudation; and he describes 'a film or membranous substance lining the cavity of the trachea.' Mr. Rumsey, after referring to the opinion of Drs. Home and Cullen, that croup is an inflammatory disease, makes the following remarkable comment. 'It appears to me that the croup is an inflammation of its own kind. If it consisted in common inflammation, we might expect to find the same appearances (that is, the same kind of concretion on the surface of the trachea) every day, as its mucous membrane is so frequently the subject of inflammation attended with an increased secretion. The matter, however, of which this substance is formed possesses different properties from those of the mucus which is thrown out upon the membrane of the nose or of the trachea in common catarrhal affections.' In these remarks of Mr. Rumsey we have the earliest intimation of the specific character of that inflammation of the larynx and trachea which results in the formation of false membrane, and of the essential difference between that and what he calls common inflammation.

Dr. Cheyne's treatise on the *Pathology of the Membrane of the Larynx and Bronchi* was published in 1809. In describing croup, he says: 'I have seen children so affected that I at first imagined they were suffering under the second stage of croup; but, upon examination, I discovered sloughs on the tonsils and uvula. The cough, voice, and breathing were those of the second stage of croup.' Dr. Cheyne doubts whether these were cases of 'true croup;' but it can scarcely be doubted that they were cases of diphtheritic croup. Dr. Cheyne mentions two cases in which croup occurred as a complication of scarlet fever. In a child, there was croupy cough and breathing, and 'the fauces were sloughy.' In this case no examination was made after death; but, in the case of a soldier who died, 'the trachea was found lined by a membrane as in croup.' Unquestionably, these were cases of diphtheria associated with scarlet fever.

[1] *Trans. of Society for Improving Med. and Surg. Knowledge*, vol. ii.

I have shown you that our English writers on croup, from Home and Cullen downwards, unquestionably met with cases of membranous exudation on the fauces and air-passages which could have been no other than the disease which we now recognise as diphtheria; but until the publication of Bretonneau's memoirs on *Diphtheria*, if we except the brief statement which I just now quoted from Rumsey, no one had pointed out the essential distinction between the specific diphtheritic inflammation of the throat and air-passages, with its resulting membranous exudation, and simple catarrhal inflammation of the same structures.

Most writers on croup before the time of Bretonneau, and many writers since, have confounded two essentially distinct diseases—namely, a specific diphtheritic laryngo-tracheitis and simple catarrhal inflammation of the air-passages. Bretonneau, Trousseau, Guersant, and all the leading French pathologists, agree in defining true croup to be that form of the disease which is attended with a membranous exudation. They also agree that this is a specific product of the diphtheritic poison; while 'false croup,' or, as they call it, 'stridulous laryngitis,' is a catarrhal inflammation excited by cold, and not resulting in the formation of a coherent membrane. I believe this pathology to be correct; but it is unfortunate that the term croup should have been diverted from its original significance of a croaking or crowing noise, to express an anatomical condition. It is unquestionable that this attempt to restrict the application of the term croup has been a source of confusion, especially to English practitioners, who, having been accustomed to think of croup as an inflammatory disease the result of exposure to cold, often fail to distinguish between this simple inflammatory croup and the 'true croup' of the French.

It is evident that most cases of membranous croup are diphtheritic, and the only question that remains for consideration is this: Is there a form of membranous croup which is not diphtheritic? It is possible that there may be more than one form or modification of morbid poison which may excite a membranous exudation in the air-passages. Of this, however, we have no proof; but the evidence is very weighty

against the opinion entertained by some that exposure to cold alone may excite a membranous inflammation. Our experience of catarrhal inflammation of the mucous membrane of the air-passages is very great. We see it constantly under the forms of laryngo-tracheitis and bronchitis; and the product of this inflammation, at every period of life from infancy to old age, is the same—a muco-purulent secretion, and not a coherent false membrane.

On the other hand, we meet with numerous cases of diphtheritic inflammation affecting the nares, fauces, and air-passages, with the formation of a more or less coherent membranous exudation; this product being, as Mr. Rumsey said more than eighty years ago, entirely different from that of ordinary catarrhal inflammation.

It is, then, unquestionable that, in the vast majority of cases, a membranous exudation in the air-passages is the specific product of diphtheria. It is equally certain that, as a rule, catarrhal inflammation the result of cold, although intense enough to destroy life, as it does in very numerous instances, is attended with no such membranous exudation. The arguments which are sometimes put forth in support of the proposition that there is a form of membranous croup which is not diphtheritic, are quite inconclusive. It is admitted by the best authorities that the morbid anatomy of membranous croup is identical with that of diphtheria. The infiltration of the mucous membrane, by some supposed to be characteristic of diphtheria, may exist in the fauces and larynx, while in the trachea and bronchi of the same subject there is an unadherent, so-called croupous exudation on the surface. It is certain that the absence of exudation on the fauces will not prove the croup to be non-diphtheritic; for the diphtheritic process may begin in the larynx, or, what is more common, the exudation which first occurred on the fauces may become detached, while the disease is extending downwards into the air-passages. In some cases the *posterior* surface of the soft palate is covered by exudation, while none is visible on the *anterior* surface. I saw two sisters, aged respectively 9 and 6 years, who were taken ill simultaneously. The younger child had a diphtheritic patch on one

tonsil, which went no further, and she was soon well; the other had a croupy cough and breathing, but no visible exudation on the fauces, and she quickly died from obstructed breathing. There was no *post-mortem* examination; but there could be no question that the case was one of laryngeal diphtheria, without exudation on the fauces. Mr. Lattey, of Southam, mentions a case[1] in which a pellicle appeared on the tonsils on the second day, and separated on the third, when, a day or two afterwards, croupy symptoms occurred; so that, if this case had been first seen at that period, it might have been taken for a case of croup without exudation on the fauces.

It is sometimes said that, whereas membranous croup is a sthenic disease, diphtheria is asthenic; but it is notorious that, in many cases of undoubted diphtheria, there is a remarkable absence of constitutional symptoms until the larynx is invaded. Then, although diphtheria belongs to the class of epidemic and contagious diseases, it often occurs sporadically, and shows no disposition to spread from the sick to the healthy.

It has been urged, as an argument against the identity of membranous croup with laryngeal diphtheria, that the older writers on croup make no mention of the *paralytic sequelæ* which occur in some diphtheritic cases. In reply to this argument, I would remark first, that, since laryngeal diphtheria is usually fatal, it rarely happens that the sufferers from this form of disease live long enough to develop the paralytic sequelæ; and, secondly, it is remarkable that in none of Bretonneau's elaborate memoirs on diphtheria is there any reference to these paralytic symptoms, which had escaped the notice of that acute observer. We see only what we have been educated to observe. To deny the identity of laryngeal diphtheria with membranous croup because no reference in past times was made to paralytic sequelæ, would be as unreasonable as to doubt their identity because the frequent coexistence of albuminuria was never mentioned until Dr. Wade pointed out the fact, and so taught us to look for it.[2]

[1] *British Medical Journal*, May 29, 1875.

[2] After the publication of this paper in the *British Medical Journal*, Dr.

One remarkable fact in the history of diphtheria is, that it often occurs as a complication of other diseases. Thus it may coexist with scarlet fever, or with measles; and it is probable that a common catarrh may act as a concurring cause of the specific diphtheritic process. As an abraded skin may become inoculated by the diphtheritic poison, so may the throat when abraded by a simple catarrhal inflammation. Now, with reference to cases of this kind, I wish to impress upon you a point of great practical importance. Whenever you meet with a case of membranous croup which has been supposed to be a result of mere exposure to cold, make careful inquiry for any possible source of infection from a similar case; and, failing this, make a rigid search for insanitary conditions in the house where the disease originated. You must not be content with simply asking whether the drains and the water supply are such as they should be; but you must search for insanitary conditions, with the certain conviction that they exist; and, unless your experience is different from mine, you will find that the cases of supposed membranous croup from exposure to cold resolve themselves into cases of diphtheria, the result of poisoning by foul air or contaminated water or milk; for diphtheria in all its forms and varieties is as certainly as typhoid fever and cholera a disease of filth origin. If ever it should be found that membranous laryngitis occurs from simple exposure to cold, without evidence of infection or of septic poisoning, I shall abandon the belief, which at present I hold firmly, that membranous croup and laryngeal diphtheria are one and the same disease.

In future, if we would avoid confusion in the use of the word croup, we must associate it with a distinctive prefix.

Wade wrote to the same journal (Oct. 2, 1875, p. 426), giving extracts from a book published in the last century entitled *An Historical Dissertation on a Particular Species of Gangrenous Sore-Throat which reigned the last year among the Young Children at Paris*, which was printed in Paris in the year 1749, translated from the French of Dr. Chomel by N. Torriano, M.D. London, 1753. It is evident that the disease of which the author treats was diphtheria, and that he noted the symptoms, which we now recognise as paralytic. The symptoms particularly mentioned are difficulty of articulation and 'speaking through the nose by reason of the fallen uvula;' one child, in addition to speaking through the nose, became temporarily squint-eyed. The book referred to is in the library of the Royal Medical and Chirurgical Society.

There are three distinct diseases to which the term croup has been applied generically.

1. Spasmodic croup; syn., 'laryngismus stridulus,' 'crowing inspiration,' 'child-crowing,' 'thymic asthma.' This is a pure neurosis, and is often associated with infantile convulsions. It is the 'false croup' of some English writers.

2. Inflammatory croup; syn., 'catarrhal laryngitis,' the 'stridulous laryngitis' or 'false croup' of French writers. This disease is a catarrhal inflammation of the larynx and trachea, excited by exposure to cold, often complicated with laryngeal spasm, usually recurring again and again in the same subject in successive cold seasons, rarely fatal, and never associated with the formation of a coherent false membrane in the air-passages.

3. Diphtheritic croup; syn., 'laryngeal diphtheria,' 'membranous laryngitis,' 'membranous croup,' the 'true croup' of French pathologists; a specific contagious disease, which may be either sporadic or epidemic, and which may exist alone or as a complication of other diseases, as, for example, measles and scarlet fever. This form of disease is one of the most fatal complications of diphtheria.

Postscript.

The fact mentioned in the preceding lecture, that diphtheria is apt to occur as a complication of other diseases and mechanical injuries, has often led and probably still leads to erroneous conclusions with regard to the etiology and pathology of some of its manifestations, and more especially when it assumes the form of membranous croup. Cases of membranous croup from exposure to cold are from time to time recorded, but a complete history of such cases would probably show that exposure to cold was not the sole morbific agent. A brief reference to two cases which have occurred in my own practice will serve to illustrate the principle for which I contend.

A delicate youth, who had often suffered from catarrhal tonsilitis, had lately an attack of his old malady. The inflammation, pain, and swelling were less severe than they had been on several former occasions, but there was now the new

feature of a distinct, though soft, membranous exudation on the surface of each tonsil. The disease did not extend to the air-passages, and it soon yielded to treatment. The question arose, what was the pathology of the exudation on the tonsil? Was it a result solely of catarrhal inflammation excited by exposure to cold? The house was on high ground, and no expense had been spared by its wealthy and most intelligent owner to render it, not only beautiful and comfortable, but wholesome. There was no reason to suppose that the patient had come in contact with any sufferer from diphtheria. I therefore expressed to the father my conviction that sewer poison was the cause of the peculiar condition of throat. And I heard subsequently that a very offensive smell had repeatedly emanated from one closet at the foot of a staircase, and, later still, it had been found that, in consequence of some defect in the ventilation of the drains, sewer gas escaped into every closet when the water was rushing down. Here then, I take it, was the explanation of the membranous exudation, which in this case had 'followed a definite exposure to cold.'

The second case was that of a healthy infant, whose fore-skin had to be partially removed in consequence of congenital phimosis. The wound, made by an eminent surgeon, did not heal, and in a few days it was covered by a diphtheritic membrane. The child was the son of a wealthy man, whose spacious house stands on the eastern border of Hyde Park. The two gentlemen in attendance upon the child, feeling sure that the unhealthy condition of the wound was the result of some insanitary surroundings, removed the patient to another house, where I met them in consultation, and where the wound soon assumed a healthy appearance and healed. With some difficulty I persuaded the father to have his house thoroughly inspected, when grave sanitary defects were discovered and corrected. Now, if this child, instead of undergoing a surgical operation, had been exposed to cold, and had thus got a catarrhal inflammation of the larynx, he would very probably have had a membranous exudation on the inflamed mucous surface, and the case might have been reported as one in which 'the formation of false membrane in the air-passages had succeeded upon a definite exposure to cold.'

To complete the evidence in this case, I afterwards heard that shortly after the operation on the child, two of the servants in the house were suffering from sore-throat, and in one of them the disease assumed a distinctly diphtheritic character.

It is only with reference to cases occurring in private practice, and especially amongst the middle and upper classes, that the inquiries which are requisite in order to throw light upon the etiology of these diseases can be thoroughly carried out. Cases occurring in hospital practice are, for the most part, quite unsuitable for this purpose, and it is a mistake to suppose that any trustworthy inference can be drawn from a large number of imperfectly observed and recorded cases.

In the *Guy's Hospital Reports* for 1877, there is a 'Collection of Cases of Diphtheria and Croup,' communicated by the late Dr. Hilton Fagge. In that paper, in the section headed 'Cases of membranous pharyngitis or laryngitis due to local injury of the throat,' I find, amongst other cases, one (Case 91) of a child who, having 'swallowed a piece of hot potato, which lodged in his throat for some time,' was found after death not only to have flakes of lymph on his tongue and tonsils, but the interior of the larynx and 'the whole trachea were more or less lined with lymph.' Now, surely it is inconceivable that this exudation in the air-passages was a result solely of a piece of hot potato in the gullet. In the history of this case it is said 'there is no diphtheria in the neighbourhood,' but nothing is stated—probably nothing was known—by the reporter with regard to the sanitary or insanitary condition of the patient's dwelling. In another case (93) of scald of the throat 'a membrane was found on the pharynx and larynx, and down as far as the minute branches of the bronchial tubes;' and in a third case (94) of cut-throat there was 'plastic lymph in the pharynx and larynx, traceable into the smallest bronchial tubes.' Surely these cases prove too much to warrant their being designated 'cases of membranous pharyngitis or laryngitis, due to local injury of the throat.' They prove that a membranous exudation spreading into the air-passages far beyond the seat of injury must have been due, not to the mere local injury, but to a diffusible, specific, infecting poison, the source of which might probably have been discovered by a

careful investigation of the patient's surroundings, before and after the receipt of the injury.

The result of my own investigations has been the conclusion that, in the absence of direct contagion, the occurrence of membranous pharyngitis or laryngitis affords conclusive evidence of infection by sewage poison, conveyed through either air or water. It should, however, be borne in mind that milk may be the vehicle of the specific poison from an infected dairy.

CHAPTER XXI.

ON CERTAIN POINTS RELATING TO THE ETIOLOGY, PATHOLOGY, AND TREATMENT OF DIPHTHERIA.[1]

Evidence as to Diphtheria being a Contagious Disease—Cases Proving its Origin from Unsanitary Conditions—Pathology of the Disease—A Local Inoculation followed by a General Infection of the System—Cutaneous Diphtheria—Auto-Infection of the Air-Passages from the Exudation on the Fauces—Proofs of Secondary Blood Infection—Membranous Croup is always Diphtheritic—Means of Prevention and Cure—Value of Local Disinfectants—Indications for Tracheotomy—Nutritive Enemata—Mode of Examining the Throat.

I PROPOSE in the present communication to discuss some important practical questions relating to the etiology, the pathology, and the treatment of diphtheria.

There are some practitioners who, believing that diphtheria is a specific contagious disease, maintain that defective drainage and filth have little or no influence in its causation, while others, denying its contagiousness, assert that its origin and spread may always be explained by insanitary conditions. I believe that both classes of negationists are in error. I have no doubt that the disease, though not highly contagious, is communicable from the sick to the healthy, and I have as little doubt that it is often caused by filthy emanations from sewers and cesspools, and this, too, when it is in the highest degree improbable that any specific poison can have been introduced from without into the decomposing stuff that has excited the disease. In proof of the contagiousness of diphtheria, the following, amongst a multitude of similar cases, may be set forth.

M. Valleix, a colleague of Trousseau, while examining the

[1] Read before the West Kent Medico-Chirurgical Society, Dec. 4, 1874, and published in the *Lancet*, Jan. 2 and 16, 1875.

throat of a patient, received into his mouth a small quantity of saliva spurted out by the patient in coughing. Next day on one of his tonsils there was a pellicular deposit, and some hours later both tonsils and the uvula were covered by false membrane; the disease made rapid progress, and in forty-eight hours he died. Another of Trousseau's provincial colleagues was performing tracheotomy in a case of diphtheritic croup, when he applied his mouth to the wound to suck blood from the trachea. He thus inoculated himself, and died in forty-eight hours.[1]

The more recent case of the late Dr. Rabbeth, a former distinguished student of King's College, who sacrificed his own life while endeavouring to save that of a child, excited widespread interest, admiration, and sympathy. While performing tracheotomy in a case of laryngeal diphtheria, he applied his mouth to the canula, to free it from obstruction. The result was that, in a few days, he died of diphtheria, which invaded not only the larynx and trachea, but even the terminal bronchi.

Sir William Jenner states that Dr. E—— lived in Euston Road. He was attending a child ill from diphtheria, when he sickened with the same disease. As he recovered, his female attendant was attacked with it. No one in his house suffered except Dr. E—— and the young woman who was in constant attendance upon him. The child from whom Dr. E—— appeared to have caught the disease resided some distance from Euston Road.[2]

Mr. Simon's *Second Report to the Privy Council* contains the following piece of evidence:—'No case (says Mr. Eastes) of diphtheria had ever been seen in Folkestone during my time, until Isabella W——, aged $4\frac{3}{4}$ years, arrived from Boulogne on the evening of July 2, 1856, being then in an advanced stage of the disease. She died on the following day. On July 6, Catherine W——, her sister, aged 10, was attacked. But she had never been in France; she had always resided on the East Cliff, Folkestone, in the same house to which her dying sister was brought four days previously. One

[1] Trousseau's *Clinical Medicine*, New Sydenham Society, vol. ii. p. 497.
[2] *Diphtheria: its Symptoms and Treatment*, p. 52. 1861.

other case occurred in the same house three days after, and they all terminated fatally.'

In the same report Dr. Rumsey, of Cheltenham, states that 'a schoolboy convalescing from diphtheria contracted by him at Swansea, where it was epidemic, and arriving at home in an open healthy suburb of Cheltenham, where at the time there was no diphtheria, was received and embraced by one of his two sisters. On the fourteenth day afterwards she was attacked by diphtheria and suffered severely. Her sister, who did not meet her brother, helped to nurse her, and was attacked fourteen days after the first sister by the same specific disease.'

Trousseau gives a case of direct inoculation of the mother's nipple by a diphtheritic sucking infant. The nipple became covered by false membrane, but the disease was arrested by appropriate treatment.

I have to thank Dr. Dewes, of Coventry, for the following brief statement of very instructive facts:—The younger of two brothers, a boy named Arthur, was taken ill at home with scarlet fever on November 8. As his mother was expecting her confinement, he was sent to a cottage on the outskirts of the town, where he had a severe attack, with a copious eruption and diphtheritic exudation on his fauces, but not in his larynx. Meanwhile the elder brother, Alfred, was taken ill at school on November 16. Both boys had attended the same day-school; and at the same time some of the other boys were ill at home with scarlet fever. As the school-mistress was a relation of the boys, it was arranged that the school should be dispersed and the boy nursed there. He had scarlatina anginosa, but no diphtheritic exudation, and he became convalescent about November 25. On December 5 the man at whose cottage Arthur was staying sickened with scarlet fever, and he, like the boy, had also a diphtheritic throat. It now became necessary to remove the boy from the cottage, which was done on December 6 or 7. As the two brothers had been convalescent for a fortnight, they were both allowed to occupy the same room at the schoolhouse, and, although separate beds were provided for them, they soon got into the same bed. They were not allowed to leave the room,

and all went on well until December 18, when Alfred was observed to have a croupy cough, with bronchial râles throughout the chest. On the following day he was much worse, the croup and the bronchitis hourly increased in severity, there was a diphtheritic exudation on the tonsils and uvula, and he died on the third day from the onset of the croupy symptoms. In neither case was the urine albuminous at any period of the illness. The most probable explanation of these facts appears to be that at the cottage where the first boy, Arthur, was nursed, there was some sanitary defect which caused the scarlet fever to be complicated with diphtheria. The result of bringing the two boys together was, that Alfred caught a fatal diphtheria from his convalescent brother.

I received the following history from the father of the children referred to. About four years ago a girl, aged 15, was sent home to a large house in an open park in Hertfordshire, after having been ill for ten days at a school at Brighton. It was noticed that for some days after her arrival she had a discharge from the nostrils. As no warning was given that she had been suffering from an infectious disease, she was allowed to associate with her seven brothers and sisters. After she had been about a week at home, the schoolmistress wrote to say that she felt anxious to hear of her pupil, as there had been some cases of diphtheria at the school. In about another week a sister aged 10, and a brother aged 4, were seized, within a day or two of each other, with symptoms of diphtheria, and in a few days they both died. The children were isolated as soon as they sickened, and no other case occurred. In this case there can, I suppose, be no doubt that the young lady when she arrived at home was suffering from nasal diphtheria, and she thus became the vehicle of a fatal infection to her brother and sister.

The following case came under my own personal observation. In May, 1867, a gentleman living in one of the largest houses in Westbourne Terrace was under my care for diphtheria. During his illness, a gentleman from the country, who was staying in the house, spent much time in the patient's room, and helped to nurse him. When the patient was convalescent his friend sickened with what proved to be a

severe attack of diphtheria, from which he recovered at his country house.

I believe that all the cases which I have here cited are examples of the diphtheritic infection being conveyed either through the air or, more directly, by the actual contact of the morbid secretions with the tissues of the recipient.

To oppose to positive evidence of this kind such negative statements as that, in numberless instances, medical attendants and nurses have come into close contact with diphtheritic patients without taking the disease, appears to me a vain and frivolous objection. Diphtheria is not a highly contagious disease. In the scale of infectiousness it stands far below scarlet fever, for instance; and there is reason to believe that the susceptibility to the disease differs almost infinitely in different persons; but a medical attendant who entirely ignores the contagiousness of the malady is likely to neglect reasonable and necessary precautions to protect himself and others from the risk of infection.

When a case of diphtheria occurs in a house without evidence of importation from without; still more when several cases occur together or in quick succession, there will be good reason to suspect that sewers, cesspools, or contaminated water may be the source of the disease. My belief is that, in a very large proportion of cases, there is as close a relation between diphtheria and insanitary conditions as exists between typhoid fever and similar insanitary conditions; and I scarcely need say that, if this be so, the general recognition of the fact is of the greatest importance, with reference to the adoption of preventive measures. There is reason to believe that much more harm would result from ignorance of the filth origin of diphtheria than from practically ignoring its infectiousness.

Many instances have come to my knowledge in which fœtid fæcal emanations have appeared to be the direct cause of diphtheria. I will refer to a few of these.

The first cases of diphtheria that I remember to have seen occurred in the persons of two children of a village tradesman in Kent, in October, 1855. They were both taken ill simultaneously: one, a girl, aged 9½ years, got laryngeal symptoms and died; her sister, aged 6 years, got a diph-

theritic patch on one tonsil, and was quickly well. Another sister, aged 7, had died on May 1 previously, after a week's illness, in which sickness, sore-throat, and a very offensive discharge from the nose were the chief symptoms. No other cases had occurred in the village up to this time. The house stood by itself in a garden; there was no drainage. From what I have since seen of diphtheria, I have no doubt that the cause of the disease in these three children was the effluvium from a copious and most offensive fæculent accumulation, beneath a common open privy in the garden, to which the whole household resorted.

In January, 1872, I saw with my friend, Mr. Bateman, of Richmond, a lady, Mrs. B——, who was suffering from a diphtheritic throat. Mr. Bateman has kindly sent me the following note of the circumstances:—'The lady you saw with me at Ham was one of a family consisting of herself, her husband, four children, and three servants. The house is drained into a cesspool about twenty yards distant. The accumulation of many months was emptied one day while the wind was blowing towards the house from the cesspool, and a very offensive smell reached the house. Three days afterwards all the four children became feverish and complained of sore-throat; the tonsils were seen to be inflamed and covered with yellowish-white patches. In a few days two of the servants were attacked, one rather severely; and, lastly, Mrs. B——, the lady you saw with me. Her tonsils were inflamed and covered by false membrane. The only members of the family who escaped were Mr. B——, the husband, who was away from home all day, and one servant.' All the patients recovered.

When Mr. Bateman wrote me the history which I have just read, he went on to say: 'I have been called lately to a similar set of cases. The family consisted of the father and mother, seven children, and three servants. On going up the garden to the house my nose was assailed by a horrible stink, and, seeing some men at work close to the house, I stopped to see what they were doing. I found that they had ripped open a drain running in front of the house within ten yards of it, and they had opened a cesspool into which the drain flowed. I found the mother, five of the children, and two of the

servants suffering from sore-throat identical with the Ham cases.' In the same letter he says: 'I am at the present time (January, 1874) attending at another house where a young lady and the parlour-maid are suffering from severe sore-throat with the usual patches. A cesspool had been opened a few days before quite close to the house. I feel quite certain that all these cases were caused by the sewage filth, and in particular by that portion of it which, floating in the atmosphere, was inhaled by the persons affected.'

I am indebted to my friend Dr. Casey, of Windsor, for the instructive particulars of a succession of cases of diphtheria in a family at the village of S——, from November, 1872, to May, 1873. 'The family consisted of father, mother, and nine children, aged from 12 to 1 . The house is detached, and out of the village. There were no cases in the immediate neighbourhood, though the disease occurs there with, I think, more than average frequency. During November, 1872, two of the children (Nos. 6 and 7) passed through attacks of diphtheria of moderate severity. A week or so after their seizure (November 24), their mother, who was nursing them, was attacked by the disease in a malignant form, and died on December 1. After some days the five elder children were sent away, and some steps were taken to repair drains and disinfect. Six weeks after (January 15, 1873), the baby and a servant fell ill, and the former died (January 23). Then the three children remaining in the house (two of them convalescents) were sent away. The house was now most energetically disinfected, and drains examined. After the lapse of ten weeks all the children returned (April 9). A week after their return their uncle visited them, slept in the house two nights (April 15, 16), and returned home on the evening of the 17th. During his journey home to Worcester he felt unwell, was worse, restless and sleepless during the night, next day decidedly ill, and on the morning after that (19th) a patch of diphtheria was recognised. The attack which followed was a mild one. Six days after the uncle's visit (April 22) the youngest child showed the disease. This child had remained in the infected house until the second out-

break (January), and had since that been away with the two convalescents. The children were again removed; Nos. 1 and 4 to one house, and the rest (Nos. 2, 3, and 5) to another. But of the latter, two more (Nos. 2 and 3) were attacked in a few days (April 25) in succession; and more than a fortnight after removal from home, yet another child (No. 5) fell ill (May 11). This was the last and tenth case, two only of the children (Nos. 1 and 4) having escaped. And now, at last, the foul remains of a disused cesspool were found under the floor of the drawing-room, which room, I may add, had from the first been suspected of bad smell. After that had been remedied all the family shortly returned, and have continued well. The last case must, seemingly, be ascribed to *personal* contagion, for it occurred after the child had been for more than a fortnight away from the original source of infection, and in a presumably healthy farmhouse. On the other hand, the uncle's attack was almost certainly owing to *endemic* infection, for the lapse of time (ten weeks) and the vigorous measures adopted to purify the house (which was, when he slept in it, still bare of carpets and papers) renders the supposition that contagious matter from the previous cases lingered in the house and furniture improbable.'

It appears to me to be a matter of almost absolute certainty that the foul cesspool was the primary source of all these cases. It is possible, and not unlikely, that the mother may have caught the disease from the two children who were the first sufferers; and it is still more probable, as Dr. Casey suggests, that the last case, which occurred a fortnight after the removal from the pestilential house, was the result of contagion from the other children at the farmhouse. It is well to note that not only was the house detached, but the cesspool had long been disused. It appears, therefore, scarcely possible that a specific diphtheritic poison could have got into the cesspool from without: the poison must have been compounded within the precincts of that foul receptacle. Some time since I told the story which I have just now read to a medical officer of health, and he expressed his belief that the cesspool had nothing whatever to do with the diphtheria! His incredulity affords a good illustration of the very different

impression which is produced upon different minds by the same evidence.

My friend Mr. Salter, of Tolleshunt D'Arcy, in Essex, who has had a large experience of diphtheria, writes to me that he has 'had unquestionably a great many cases of diphtheria, whose origin can be distinctly traced to sewage poison, either gaseous or liquid;' and he gives me some particulars of an outbreak in one family, four children and a nurse having been attacked in quick succession, which he attributes to the percolation of sewage into the well which supplied the family with water.

In several instances, of late, an outbreak of diphtheria has been traced to contaminated milk.

I look upon the occurrence of an indigenous case of diphtheria in a house as an indication of the necessity for a most rigid inquiry into the condition of the drainage and the water-supply. At the beginning of the present year, a gentleman living in one of the best houses in Queen's Gate asked me to see his butler, whom I found suffering from a severe attack of diphtheria. The basement of the house looked the perfection of cleanliness, but I advised that a sanitary engineer should be called in to inspect the premises. The result was the discovery of an untrapped sink-pipe near the butler's sleeping-room.

Another case, seen with Mr. Patten at Ealing, was traced to the untrapped waste-pipe of a bath, which thus acted as a ventilator from the sewer into the bath-room and the adjoining bedroom.

It is notorious that in the houses of some of the most exalted and wealthy, and in open country districts, the sanitary defects which originate such diseases as diphtheria and typhoid fever are almost as common as in the meanest cottages and in the most crowded cities; but it is obvious that overcrowding in the small rooms and cottages inhabited by the lower classes must greatly increase the danger arising from other insanitary conditions.

In opposition to the doctrine which I am now advocating, it is sometimes stated with perfect truth that diphtheria never visits some houses which are dirty within and without,

and which are surrounded by every form of abominable filth. The reply to this is, that no one believes that any and every kind of foul emanation from decomposing organic matter will suffice to cause diphtheria. As every black powder containing charcoal is not the explosive compound which we call gunpowder, so every fetid gas escaping from a drain or a cesspool is not laden with the perilous stuff which will excite diphtheria. We believe that a combination of conditions, local and atmospheric, is required to generate or develop the morbid poison, and the absence of any one of these may prevent its formation. I learn from Mr. Salter that on several occasions, after his district has been free from the disease for many months, he has 'suddenly been called to three or four cases in one day, at distances of ten or twelve miles apart.' Such an occurrence can be explained only by the theory of some widely diffused atmospheric influence concurring probably with local insanitary conditions.[1] Mr. Salter tells me that, although he has 'had cases occurring in every month of the year, July, August, and September seem to be the months chiefly preferred by the disease.' These are the months during which, in consequence of the high temperature, the decomposition of organic matters would generally be most active. The analogy of other diseases renders it probable that, besides temperature, there are other climatic conditions which favour the occurrence of diphtheria at one season rather than another.

I now pass on to the consideration of certain points relating to the *pathology of diphtheria*.

The most characteristic feature of the disease is the albumino-fibrinous exudation which usually shows itself first on the palate, fauces, uvula, and tonsils; often passes down into the larynx, the trachea, and the bronchi; rarely down the œsophagus to the stomach. In some cases the mucous membrane of the nostrils is affected first, or simultaneously with that of the throat. Many practitioners appear to suppose that the diphtheritic exudation is a result of a previous

[1] In this respect diphtheria resembles other zymotic diseases, such, for instance, as cholera and small-pox. See Dr. Goodeve's remarks on the two factors requisite for the spread of an epidemic (*ante*, p. 94).

blood-poisoning, and that it is so far analogous to the eruption of small-pox and scarlet fever. I have long been in the habit of giving a different explanation of the phenomenon. I believe that the poison—whether inhaled with the air or swallowed with water—coming into contact first with the mucous membrane of the fauces or the nostrils, exerts there a local poisonous influence, and that the exudation is a direct result of this purely local action; that, in short, the diphtheritic poison applied to the mucous membrane calls out the membranous exudation, as the application of cantharides to the skin raises a blister, or as the poison of small-pox applied to a puncture on the skin raises a pustule at the point of inoculation.

The history of cases of *cutaneous* diphtheria affords support to this doctrine. It is a well-known fact that, in what we may call a diphtheritic atmosphere, a part of the skin from which the epidermis has been removed by a blister or other abrading agent may become the seat of the diphtheritic exudation. Trousseau,[1] referring to these cases, expresses his belief that 'an abraded surface has served as a door of admission for the disease, which for some time remains a local affection.' He compares the phenomena with those which occur when a syphilitic local sore is followed by constitutional symptoms. And he wisely insists upon the importance of promptly applying caustics and antiseptics to destroy the poison at the spot, and so to prevent the extension of the disease.

The local exudation, whether on the skin or on the mucous membrane of the nostrils or the mouth, tends to become a source of infection to the system at large, through the process of absorption by the lymphatics and the blood-vessels. In most cases a diphtheritic exudation on the throat is associated with enlargement of the lymphatic glands at the angle of the jaw. This is a result of the irritant action of the poison in its passage through the lymphatic absorbents to the general circulation; and it bears the same relation to the primary exudation on the mucous membrane as the enlargement of the inguinal glands bears to a chancre on the penis. In like

[1] Vol. ii. p. 523.

manner, the insertion of the virus of small-pox beneath the skin of the arm raises a pimple which subsequently becomes vesicular and then a pustule; meanwhile there is swelling of the lymphatics in the armpit, followed by fever and other results of a general blood infection. Trousseau [1] refers to the case of a man whose two children were suffering from a diphtheritic throat, one of whom died. While matters were thus going on, the father, who had an excoriation on his foot, began to feel acute pain there; this was soon followed by an ulcer, the surface of which became covered by a grey membrane. Meanwhile the glands of the groin and the inside of the leg were a good deal swollen. The disease was arrested by the local application of calomel to the diphtheritic skin. It will be seen, from the history of this and similar cases, how close is the resemblance between the phenomena of cutaneous and mucous diphtheria.

I wish now to direct attention to a feature in the pathology of diphtheria which appears to me to be of extreme interest, and which has a most important bearing upon the treatment of the disease. I allude to the *auto-infection* which must obviously result from the very frequent passage of air into the lungs over the morbid exudation in the throat, and the less frequent passage of food and the secretions of the mouth down the gullet to the stomach. We all know that one of the greatest dangers in cases of diphtheria is the tendency of the exudation to pass down the air-passages into the larynx, the trachea, and even into the minutest ramifications of the bronchi. This tendency in the exudative process to pass downwards is, perhaps, in part explained by the gravitation of the morbid secretions. Trousseau remarks [2] upon the tendency which cutaneous diphtheria has to pass downwards from the shoulder to the arm, from the neck to the back, and from the belly to the loins, and he says: 'It is very probable that the propagation of the diphtheritic inflammation is accomplished by the irritation induced by the long contact of the serous discharge which bathes the part as it runs downwards, or is retained by the dressings in particular situations.' This seems a very probable explanation of the downward ten-

[1] P. 519. [2] P. 523.

dency of the disease when it affects the skin, and, as I have already suggested, it may in part explain the tendency of the diphtheritic exudation to invade the air-passages; but when once attention is directed to the subject, it seems obvious that the inspiratory current of air passing over the morbid exudation in the fauces, must carry with it infecting materials into the air-passages, and thus explain the fatal tendency of the disease to invade the larynx, the trachea, and the lung.

If, as we have reason to believe, the breath and the oral secretions of a diphtheritic patient are so poisonous as sometimes to convey the disease to the attendants who are brought within their noxious influence, it can scarcely be doubted that the extension of the disease within the body of each patient is favoured by the process of auto-infection to which I have referred.

Trousseau makes an observation which is interesting in connection with this part of our subject.[1] He says that when the disease has invaded the larynx, the early operation of tracheotomy appears to prevent the extension of the false membranes to the bronchial tubes, whereas, if the operation has been delayed for forty-eight hours, the exudation is often found to have extended even to the minutest ramifications of the bronchi. Trousseau gives no explanation of this clinical fact, but the explanation which at once suggests itself is that, after the operation of tracheotomy, the air entering the trachea by the artificial opening below the infected larynx, ceases to carry into the bronchi the morbid products from the larynx and fauces, and so the extension of the disease is prevented.

It is an obvious inference from this view of the pathology of the diphtheritic process, that the persevering application of disinfectants to the fauces and air-passages constitutes one of the most important points in the treatment of the disease. We shall presently return to this part of the subject.

That the exudation on the mucous membrane of the throat is a result of the local action of the inhaled or imbibed poison, and not of a previous blood infection, is rendered probable by the occurrence of cases in which a copious formation of false membrane causes rapid suffocation with little or no preceding

[1] P. 482.

fever or other symptoms of constitutional disturbance ; the first symptoms to excite alarm in these insidious cases being hoarseness, croupy cough, and stridulous breathing. Most of us probably have met with such cases, and Trousseau refers to several.[1]

In most cases of diphtheria there is abundant evidence of blood infection during the progress of the malady. The high temperature, the albuminuria, the general constitutional disturbance, and the nervous symptoms and sequelæ are results, probably, of blood-poisoning. There are two distinct modes in which the blood becomes infected in these cases. First, by the direct passage of the inhaled poison through the pulmonary capillaries into the blood. Second, by the absorption of the morbid products from the exudation on the mucous membrane through the lymphatics and blood-vessels. As there is a class of cases, just now referred to, in which, with an abundant local exudation in the throat, the constitutional symptoms are of the mildest character, so there is an opposite class of cases in which constitutional symptoms, the result, probably, of blood-poisoning by inhaled drain miasmata, are overwhelming and rapidly fatal, while there is little or no appearance of false membrane on the surface of the throat.

Acute albuminuria is a very frequent complication of diphtheria—a fact which was first discovered by Dr. Wade, of Birmingham ; yet it is notorious that dropsy is an extremely rare result of this renal complication. On the other hand, the albuminuria of scarlet fever is usually associated with more or less of anasarca. The probable explanation of this difference is, that in cases of diphtheria the skin is not implicated and its functions are unimpaired, whereas in cases of scarlet fever the cutaneous secretion is checked by the inflammation which constitutes the rash. There can be little doubt that suppressed or diminished action of the skin is a powerful concurring cause of what is commonly called renal dropsy.

I wish to express emphatically my entire concurrence in the conclusion long since arrived at by Bretonneau, Trousseau, and all the leading French pathologists, that all cases of so-called croup which are associated with the formation of false

[1] P. 489.

membranes in the air-passages are essentially diphtheritic; and, on the other hand, that what we in this country call inflammatory croup, or catarrhal laryngitis, never results in the formation of false membrane. It is surprising that practitioners of large experience can have any doubt upon this subject; yet we find, even in some of the most recent English text-books, that perplexing attempts are made to distinguish between what the authors call 'true membranous croup' and diphtheritic croup. The attempt is hopeless and most confusing to the student, for it is certain that membranous croup and laryngeal diphtheria, as we now see them, are one and the same malady.

As with scarlet fever, so with diphtheria, the symptoms differ much in different cases. In some the local throat symptoms are severe, while the constitutional disturbance is slight; and in others the reverse is the case. In some the urine is highly albuminous, in others quite free from albumen. The exudation varies much in consistency and in extent. There is also great variation in the degree to which the mucous membrane and the submucous tissues become implicated in the morbid process. But there is reason to believe that in all these outward diversities, we have only varied manifestations of one and the same disease. Every variety may be met with, not only during the same epidemic, but sometimes even in different members of the same household.

If, as some writers suggest, there be a form of membranous croup which is neither the result of simple inflammation nor of the diphtheritic poison, I can only say that I have never met with the disease, nor with any satisfactory evidence that such a disease exists, or has been seen by others.

I now come to the consideration of that which is the end and object of all our etiological and pathological inquiries—I mean *the prevention and the cure of disease*. Certain general principles of treatment appear to follow directly upon what has been said upon the pathology of diphtheria. The infecting properties of the local exudation in the throat at once suggest the use of disinfectant applications to the false membranes; and I find a general concurrence of opinion amongst those who have seen much of the disease that such applications are

attended with very beneficial results. The object to be attained by these remedies is so to change the morbid exudative material that it shall lose its infecting properties, whether these be exerted by being absorbed through the lymphatics or by passing on into the air-passages. The substances which have been most used for this purpose are solutions of chlorine, of permanganate of potash, and of sulphurous acid. I believe that these are all useful applications, but, on the whole, I give the preference to the chlorine preparations. Mr. Salter, whose practice in cases of diphtheria I know to have been successful to a very remarkable degree, relies mainly upon 'large and frequent doses of tincture of perchloride of iron, a disinfectant gargle of chlorinated soda, and abundance of liquid food and stimulants, especially port wine,' which he believes to be better than brandy. Mr. Thomas Stiles, who has published an interesting account of a violent epidemic of diphtheria at Pinchbeck, in Lincolnshire,[1] mentions as his chief remedies the local application of Beaufoy's solution of chlorine, chlorine gargles, and the internal use of tincture of perchloride of iron. The more I have seen of diphtheria the more convinced I have become that, as in cases of cutaneous diphtheria, so in the more common internal form of the disease, local medication is of primary importance, and I believe that much of the good which appears to result from frequent doses of the tincture of iron is due to its local action on the fauces in its passage to the stomach. Moreover, it seems to me not improbable that, apart from its general stimulant and sustaining influence, the superior efficacy of port wine, as compared with brandy and other kinds of wine, may be partly due to its local astringent action.

I am convinced that the application of strong irritants to the throat is as injurious as the application of leeches or blisters to the skin, and for the same reason—namely, that any breach or abrasion of the mucous or cutaneous surface opens a door for the more ready entrance of the poison.

When the exudation has extended into the air-passages it is of course beyond the reach of gargles; but a chlorine or a sulphurous-acid solution may be applied to the larynx by

[1] *British Medical Journal*, 1858.

means of a bent brush, and the inhalation of disinfectants in the form of vapour or spray may still be useful. For this mode of application I believe the sulphurous-acid spray is preferable to that of chlorine, in consequence of its being less irritating to the lungs, though much, of course, will depend on the relative strength of the solutions. My friend Dr. Joyce, of Cranbrook, writing to me some months since, stated that he had successfully treated his five last cases with sulphurous-acid spray and large and very frequent doses of perchloride of iron; and he ends by saying, 'I shall always for the future try the spray.'

Whatever local remedies are employed should be applied frequently—every hour or two if the patient's condition admit of it, the object being to disinfect the poisonous exudation as soon as it is formed.

When, in consequence of laryngeal obstruction, the question of tracheotomy arises, it is well to bear in mind that, by the prompt and early performance of the operation, we may, in a manner already explained, prevent that extension of the exudation to the air-passages which renders the late resort to tracheotomy an utterly hopeless proceeding. One remarkable but very common result of tracheotomy is so great a loss of the reflex excitability of the larynx that liquids are often permitted to pass through the glottis during the act of deglutition. This may be to some extent prevented by closing the orifice of the canula while the patient is in the act of swallowing.

In all cases of diphtheria frequent supplies of liquid nutriment are necessary, and, in a very large proportion of cases, a liberal use of stimulants is required.

When deglutition is difficult or impossible, as sometimes happens, in consequence of the condition of the throat, every practitioner knows that a patient may be sustained for many days by nutritive and stimulating enemata. Once, in a case of perforation of the stomach, I fed a woman in this way for twenty days, and she got quite well.

It is obviously important, for the sake both of the patient and the attendants, that the sick-room should be thoroughly well ventilated. A neglect of this essential condition may

lead to a relapse, or it may cause the extension of the disease to others. When there is reason to believe that diphtheria has been excited by the insanitary condition of the house in which the patient resides, it may be expedient, when possible, to remove him at once to another house, or to place him in a part of the infected house as far as possible from the probable source of the infection. Some years since I saw with Dr. Julius, near Richmond, a lady who was suffering from a second relapse of diphtheria, the disease having occurred a second and a third time before she had been thought well enough to leave home. Feeling sure that there was within the house an unwholesome atmosphere which prevented recovery, we had her placed, weak as she was, in a close carriage, and driven to a friend's house in London, where her recovery was rapid and complete.

In conclusion, I venture to give a practical hint on the mode of examining the fauces. The examination is much facilitated by having a lamp or a candle held by an attendant by the side of the patient's head, while the operator, having the concave laryngoscope reflector on his forehead, throws the light into the mouth of the patient. Thus, while he has complete control over the light, he has both his hands at liberty, one to depress the tongue, and the other, if required, to apply local remedies. In this way the throat may be thoroughly and speedily examined, with little or no risk to the operator—for he can keep at a safe distance from the patient—and with little or no fatigue to the latter, for it is not necessary to raise his head from the pillow. Then, if it be thought desirable, as it often is, he can introduce the laryngeal mirror and ascertain how far the exudation has extended towards or into the larynx.

CHAPTER XXII.

THE CHIEF CAUSES OF HOARSENESS AND APHONIA, WITH HINTS ON TREATMENT.[1]

Chief Causes of Hoarseness and Aphonia—Tumour on a Vocal Cord—Granular Unevenness of Vocal Cords—Thickening of Inter-Arytænoid Fold—Vocal Cord cut across by an Ulcer—Catarrhal Swelling of False Cords—Diphtheritic Membrane or a Foreign Body pressing on the Cords—Paralysis of the Laryngeal Muscles—Hysterical Aphonia—Muscular Fatigue—Necrosis of Arytænoid Cartilages—Lessened Vital Capacity of the Chest with impaired Muscular Power—Distinction between Aphonia and Aphasia.

THE causes of hoarseness and aphonia are numerous and diverse, but they all resolve themselves into some condition which prevents or impedes the free vibration of the vocal cords. There are five chief modes in which the vibration of the vocal cords may be interfered with. 1. A tumour upon one or both vocal cords, or between them, may prevent their close apposition. 2. A tumour or swelling above the cords or a foreign body may press upon them and so damp their vibrations. 3. There may be inaction of the laryngeal muscles and a consequent want of due tension and approximation of the cords. 4. The arytænoid cartilages, the levers to which the vocal cords are attached and by which they are moved, may be destroyed. 5. A scanty and feeble respiratory stream of air with coincident lessened tone of the respiratory muscles may render the voice feeble and whispering.

A not uncommon cause of hoarseness is a warty or other growth on one or both vocal cords. Such a growth coming between the two cords, prevents their close apposition in vocalising, and the voice is thus rendered more or less feeble and husky. In the fifty-first volume of the *Medico-Chirurgical*

[1] *Medical Times and Gazette*, Jan. 15, 1870.

Transactions I have recorded seven cases in which such growths were removed by the wire écraseur, and in the *Lancet* (January 22, 1881) I have given the history of three additional cases of the same kind.

An unequal thickening and a granular unevenness of one or both cords may interfere with the voice as much as a distinct growth from the surface of a vocal cord. This roughening of the surface of the true cords is a not uncommon result of strumous inflammation in phthisical subjects.

The inter-arytænoid fold of mucous membrane is sometimes so much thickened and indurated by catarrhal or strumous inflammation as to prevent the approximation of the arytænoid cartilages. Thus the closure of the glottis is prevented, and the voice is enfeebled.

In one case the cicatrix of a syphilitic ulcer in the inter-arytænoid fold prevented the close apposition of the cords, and rendered the voice husky; it also prevented the free movement of the arytænoid cartilages and the opening of the glottis during inspiration. Thus the breath was drawn in with a loud noise—inspiration was stridulous.

Syphilitic ulceration sometimes cuts across one of the true cords, and thus makes a large gap in the glottis. This had happened to the right vocal cord of a gentleman whose voice had been reduced to a whisper by syphilitic ulceration within the larynx, many years before.

One of the most frequent causes of hoarseness and feebleness of voice is that which occurs often in cases of catarrhal sore-throat—I mean inflammatory congestion and swelling of the so-called false cords or ventricular bands; that is, the fold of mucous membrane on either side immediately above the ventricle of the larynx. The swollen mucous membrane encroaches upon that little cavity—the ventricle—which in the normal state permits the upward vibration of the true cord; but now the vibrations are damped and impeded by the contact of the thickened mucous membrane.

A chronic catarrhal swelling of the mucous membrane of the larynx is the anatomical condition which exists in many cases of so-called 'dysphonia clericorum.'

The voice may be rendered very feeble and husky by diph-

theritic exudation within the larynx. The false membrane comes in contact with, and sometimes even covers, the cords, and thus prevents or impedes their vibrations.

In like manner, a transient aphonia may result from a piece of viscid mucus within the larynx. A forced expiration or a cough expels the mucus and restores the voice.

A foreign body pressing on the vocal cords will damp their vibrations and cause more or less complete aphonia. A good example of this is afforded by the case before recorded (pp. 341, 342), in which a half-sovereign was lying with its flat surface upon the cords.

Hoarseness or complete aphonia may be a consequence of paralysis of the adductor muscles of the larynx, and this paralysis or weakening of the laryngeal muscles may result from various causes. It is one of the symptoms and results of that rare and fatal disease which Trousseau has described under the name of 'glosso-laryngeal paralysis.' The laryngeal muscles may be paralysed by pressure on the recurrent nerve by a cancerous tumour or by an aneurysm of the aorta. In 1863 a man (F. F——) was under my care in the hospital with signs of extensive cancerous disease within the chest. His voice was reduced to a whisper, and, on inspection by the laryngoscope, I saw that his glottis was open to the extent of about one-eighth of an inch. The arytænoid cartilages were in contact with each other at their tips. There was no movement of the cartilages, and the glottis neither opened wide in inspiration nor closed completely when the patient attempted to vocalise. The inference was that both pneumogastric nerves, and especially the recurrent branches, were implicated in the cancerous growth within the chest, and thus the laryngeal muscles were paralysed. He left the hospital before his death, and the body was not examined.[1]

In some of these cases in which a tumour presses upon the nerves supplying the larynx, there is probably a combination or an alternation of palsy and spasm of the laryngeal muscles —spasm in the early stage, when the pressure is less; palsy

[1] In Chapter XXV. it will be shown that bilateral spasm and bilateral palsy of the intrinsic muscles of the larynx may result from the pressure of an aneurysm or other tumour *on the vagus nerve of one side only*.

later, when the pressure is greater, and the nerve thereby disorganised.

In the case which I just now mentioned the nerves on both sides were probably implicated. There was, in consequence, bilateral palsy of the laryngeal muscles, and both arytænoid cartilages were motionless. When a tumour implicates only one pneumogastric or recurrent nerve, there may be unilateral palsy of the larynx, as shown by the fact that, during attempts at vocalisation, one arytænoid cartilage remains motionless, while the other moves, with its attached vocal cord, in the usual manner. This unilateral palsy on the right side existed in the case of a gentleman whom I lately saw with my friend and former pupil, Mr. Miles A. Wood, jun., of Ledbury. The cause of this one-sided laryngeal palsy, with consequent hoarseness and occasional dyspnœa, was found to be a cancerous tumour at the root of the neck, involving in its substance the right pneumogastric nerve. The tumour pressed upon the œsophagus, causing difficulty of swallowing, and ultimately it made its way into the œsophagus by ulceration, and the patient died from exhaustion. Mr. Wood was early led to an exact diagnosis by observing the unilateral palsy of the larynx, and the fact that the œsophagus towards the root of the neck was so narrowed that a moderate-sized bougie was arrested there.

There are cases of purely nervous or functional, or, as they are sometimes called, hysterical aphonia. The voice may be reduced to a whisper, and even that is sometimes inaudible. On looking into the larynx we see no evidence of structural change, but when the patient attempts to vocalise we find that the vocal cords remain motionless, or that they move very little. This nervous aphonia often comes on suddenly, and it often passes away as suddenly as it came. The best treatment for it consists in the application of electricity directly to the larynx, by means of Dr. Morell Mackenzie's 'laryngeal galvaniser.' The shock excites spasm and a scream, and the cure is sometimes very rapid. I have cured several of these cases at a single sitting, and some by a single shock of electricity. A boy about 12 years of age was much frightened, and nearly drowned, by falling into a pond. From that time he

completely lost his voice, his intellect being unimpaired. When I saw him, he was reported not to have uttered an audible sound for two years. I saw that his larynx was healthy, and I determined to apply electricity. The first shock elicited a loud scream, and at once he recovered his speech.

In most cases it is necessary to apply the electricity again and again, before the cure is complete, and in some cases the treatment is unsuccessful, but these are comparatively rare. When the general health is impaired, treatment should be directed to remedy this, while electricity is being employed.

It is remarkable, but quite intelligible, that when, from any cause—a growth upon one of the vocal cords, or inaction of the muscles—the glottis remains partly open during vocalisation, the air escapes so rapidly during the expiratory act of speaking, that the patient is often compelled to draw in a fresh breath before he comes to the end of a sentence. In consequence of the patulous state of the glottis, there is a rapid escape and waste of air, and the chest is soon emptied.

There is a form of aphonia or weakness of voice which I look upon as the result of muscular fatigue and weakness. I have seen a considerable number of these cases, and most of the patients have been clergymen. The patient begins to speak in a clear and loud voice, and he continues to do so for a variable time; but after speaking or reading aloud for, it may be, a quarter or half an hour, the voice becomes feeble, and it may soon be reduced to a whisper. At the same time there is a feeling of fatigue, and sometimes positive pain in the throat. With these symptoms we may find, on looking into the larynx, no trace of structural change, or only slight congestion and redness, without swelling. A common cause of this form of dysphonia is overwork of the larynx, from frequent preaching and reading in large churches. I have known it to result from over-exercise of the voice in singing, from straining of the voice by the habit of loud talking in the midst of noisy machinery, and from violent efforts in giving the word of command.

In some instances this peculiar form of laryngeal weakness has followed upon an inflammatory attack. It seems probable that inflammation may sometimes extend from the

mucous membrane to the muscular structures beneath, and thus the nutrition and the tone of the muscles may be impaired. This is the more likely to happen if the larynx be much exercised in speaking or singing during an attack of catarrhal inflammation.

The best treatment for these cases consists in rest for the larynx as the organ of speech, change of air and scene, and a general tonic regimen. The daily application to the larynx of a saturated solution of tannin in glycerine often does good. Tannin lozenges, too, may help to give tone to the feeble voice, and the combination of iron and quinine with small doses of strychnine is sometimes useful. I have tried galvanism, but I have hitherto seen no benefit from its use in this class of cases.

Destruction of the arytænoid cartilages is a rare cause of aphonia. Some time since a case of this kind occurred in the hospital. A man was admitted under the care of Dr. Beale with aphonia, stridulous breathing, and urgent dyspnœa. I saw the vocal cords touching each other, and motionless. Mr. Wood opened the trachea, but the man died. On opening the larynx an ulcerated orifice was found in the mucous membrane over each arytænoid cartilage. A small calcareous mass alone was found in the position of each cartilage. It is evident that the cartilages had become ossified and necrosed, and had then been discharged through the mucous membrane. The glottis passively closed, because the levers which move the cords were destroyed and gone.

There is yet one cause of aphonia, or weakness of voice, which requires mention, in order to complete the sketch which I have here given. The larynx is a wind instrument, and for the perfection of its performance it is required not only that there be complete structural and functional integrity of its cartilaginous, membranous, muscular, and nervous apparatus, but the *bellows* must be in working order. There must be a free movement of the ribs and diaphragm, and there must be an unimpeded entrance and exit of air through the trachea and bronchi. When progressive muscular atrophy invades the intercostal and other respiratory muscles, the voice is weakened in proportion to the diminution of muscular power

and of chest movement. When, from any cause, the vital capacity of the chest—in other words, the volume and force of the tidal current of air—is either temporarily or permanently diminished, the voice, as a direct consequence, and in a corresponding degree, is enfeebled. Thus, during a fit of spasmodic asthma, so scanty and feeble is the respiratory current of air, that the voice is annihilated or reduced to the faintest whisper. In cases of advanced emphysema of the lung the voice loses tone and strength in proportion to the diminution of the respiratory power. One of the most curious examples of this form of aphonia is the husky whispering voice which occurs during the collapse of cholera. When discussing the pathology of cholera I expressed my opinion (p. 130) that the feeble voice may be partly due to the small volume of tidal air, corresponding with the partially arrested pulmonary circulation ; but in addition to this it is probable that, in consequence of the defective supply of arterial blood to the tissues, the tone of the entire nervous and muscular system is lessened, and that the impaired vigour of the respiratory muscles in general, and of the laryngeal muscles in particular, is the main cause of the whispering choleraic voice. It is pretty certain, too, that during a fit of spasmodic asthma the power of the respiratory, as of the other muscles, is impaired by the defective blood-supply, consequent on the partial arrest of the pulmonary circulation, during the paroxysm of apnœa.[1]

Aphonia, be it remembered, is quite distinct from aphasia. Aphonia is the result of a structural or a functional defect in the larynx, or of a deficiency in the volume and force of the respiratory stream of air. Aphasia, on the other hand, is the consequence of a break, somewhere high up, in that chain of nervous communication through which the mind is enabled to play upon the vocal instrument.

[1] See *ante*, p. 48, for a comparison of the phenomena of cholera with those of asthma.

CHAPTER XXIII.

THE INDICATIONS FOR TRACHEOTOMY IN CASES OF LARYNGITIS AND DIPHTHERIA.[1]

Tracheotomy more frequently Successful in Adults than in Children, and in Cases of Catarrhal Laryngitis than in Diphtheria—Indications for its Need—If too long delayed Œdema of the Lung and Blood-clotting render it Unsuccessful—Signs of those Conditions—Loss of Reflex Excitability of the Larynx after Tracheotomy.

When the symptoms of laryngitis, whether in a child or in an adult, continue and increase and threaten life, or when, in a case of diphtheria, the extension of the disease to the larynx causes the same threatening symptoms, we ought to have recourse to tracheotomy; and by this operation we may not unfrequently save a life which must otherwise inevitably be lost. The operation is more frequently successful in cases of simple catarrhal laryngitis than in diphtheria—for the reason that, in the latter disease, the exudation often extends into the trachea and bronchi; so that an artificial opening in the windpipe does not counteract the cause of the apnœa. It is more frequently successful in adults than in children, and more frequently in older than in very young children. In adults, I have rarely failed to save life by the timely performance of tracheotomy; but in children I have rarely succeeded. In very young children the trachea is so small that it is scarcely possible to introduce a metal tube; and the operation is, therefore, impracticable. The youngest child that I have seen saved by tracheotomy was one about 2 years old, who, while suffering from inflammatory croup, was operated on by Sir W. Fergusson.

Excluding those cases in which the operation is impossible

[1] *British Medical Journal*, Jan. 15, 1870.

on account of the small size of the trachea, the principles which should guide us in our determination to resort to tracheotomy are the same, whether the patient be a child or an adult.

In general terms, then, it may be stated that, when in spite of prompt and judicious treatment, the obstruction in the larynx and the consequent dyspnœa continue and increase, and when there is commencing lividity of the lips and face, the time for tracheotomy has arrived. When a laryngoscopic examination is practicable, and when, by this means, we discover such an amount of structural change within the larynx as must obviously require several days, and perhaps even weeks, for its removal, the necessity for the operation will be still more apparent.

In considering the question of tracheotomy, it must continually be borne in mind that, if the operation be too long deferred, although it may remove the distressing sense of constriction in the throat, it will not save the patient's life. The reason is, that a prolonged partial apnœa gradually induces a condition of lung and of pulmonary artery which is irremediable and fatal.

The order of events appears to be this. The obstruction in the larynx limits the supply of air to the lungs; the blood in the pulmonary capillaries is imperfectly aërated; and some partially aërated blood passes on into the systemic arteries. At the same time, the minute pulmonary arteries, by their contraction, lessen the supply of blood to the pulmonary capillaries in proportion to the limited access of air. This contraction of the minute arteries is doubtless called into action by a nervous influence transmitted from the capillaries. A message is telegraphed back to the arterial stopcocks, requiring a diminished supply of blood, so long as the respiratory changes are partially suspended. The blood, therefore, accumulates in the trunks of the pulmonary artery, in the right side of the heart, and in the systemic veins. The distension of the superficial veins renders the lips and the skin more or less livid; while the retrograde engorgement of the bronchial veins and capillaries, which belong to the systemic venous system, results in a serous effusion into the bronchial tubes. This serous

exudation gravitates towards the bases of the lungs, filling the air-cells and smaller tubes, and thus still further impeding respiration. Meanwhile, the slowly moving, partially stagnating blood in the pulmonary artery becomes more and more viscid, and at length partially coagulates. Hence, on *post-mortem* inspection, fibrinous coagula, which had evidently been in process of formation for several hours before death, are often found in the pulmonary artery. A state of partial apnœa, therefore, exceeding a certain limit in degree and in duration, results in œdema of the lung, and coagulation of blood in the partially obstructed pulmonary artery; and these changes in the lung and in the artery may, alone, suffice to destroy life. Tracheotomy, then, to be successful in rescuing the patient from fatal apnœa, must be resorted to before the lungs have become highly œdematous, and before the blood in the pulmonary artery has lost its fluidity.

When the laryngeal obstruction has been of recent origin and rapid in its course, it is the more likely that life may be saved by the prompt performance of tracheotomy; but, when urgent dyspnœa has been of long duration, there will always be reason to fear that the lung and the blood in the pulmonary artery may have passed into the condition which I have described.

We may sometimes obtain more positive evidence as to the œdematous condition of the lungs. There may be dulness on percussion over the lower lobes of the lungs, and a fine moist crepitation over the same extent. As a rule, however, when there is great obstruction in the larynx, auscultation teaches us little as to the condition of the lungs. The loud laryngeal stridor completely masks the pulmonary sounds; which, besides, are very feeble, in consequence of the small volume and force of the tidal air in the lungs.

We may suspect that the blood in the pulmonary artery is coagulating, when, with increasing dyspnœa, there is a combination of pallor and lividity of the skin and lips, with extreme feebleness of the pulse. The lividity is a result of over-distension of the systemic veins, while the pallor and the pulselessness are due to a corresponding emptiness of the arteries; the venous fulness on the one hand, and the arterial

emptiness on the other, being direct results of the obstruction in the pulmonary artery.

I have nothing to say as to the mode of performing the operation of tracheotomy. That is a purely surgical question, with which I do not meddle. But I must insist upon the importance of keeping the air of the room warm and moist so long as the patient has to breathe through the opening in the trachea. A neglect of this precaution might result in an attack of bronchitis or pneumonia.

Another point which deserves notice is that, while a patient is breathing through the artificial opening, so much is the reflex excitability of the larynx lessened, that, during deglutition, liquids sometimes enter the larynx, and then escape through the canula. This may, to some extent, be prevented by directing the patient to close the tube with his finger during the act of deglutition.

A gentleman suffering from acute catarrhal laryngitis, whom I attended with Mr. Heckstall Smith and Dr. Allfrey, was rescued from impending suffocation by tracheotomy performed by the latter gentleman. While he was still breathing through the tube, he one day got a piece of bone into his larynx, from the soup that he was drinking. The foreign body caused him much annoyance until it was expelled by a cough. The patient completely recovered, and returned to his work in India. The lesson taught by this and other cases is that, while a patient is breathing through an artificial opening in his windpipe, unless deglutition be performed with care, the food may 'go the wrong way,' and either lodge in the larynx, or, passing through the larynx, it may enter the bronchial tubes.

The physical conditions which result in the thickening and coagulation of the blood during prolonged apnœa are explained in Chapter III.

CHAPTER XXIV.

CLINICAL LECTURE ON SPASM OF THE LARYNX.[1]

Case of Hysterical Laryngismus—Effect of Chloroform and Chloral—Classification of Cases of Laryngeal Spasm—The Value of Chloral as a Remedy—Aneurysm of the Aorta pressing on one Pneumogastric Nerve and causing Laryngeal Spasm—Fatal Spasm of the Larynx from Pressure of a Cancerous Tumour involving the Pneumogastric Nerves—The Diagnostic Value of the Laryngoscope.

GENTLEMEN,—We have recently had in the hospital a woman who, at the time of her admission, presented symptoms which a careless observer might have supposed to indicate the existence of laryngitis. H. W——, aged 24, unmarried, and having no regular employment, was admitted on March 1. She said that a fortnight ago her breathing suddenly became noisy and difficult, and her voice feeble; these symptoms continued until the time of her admission. I saw her in the laryngoscope-room before her admission. She was breathing hurriedly, about thirty times in a minute; both inspiration and expiration were attended with loud stridor; her voice was feeble and whispering. The fact that the breathing was hurried indicated that the laryngeal symptoms were nervous, and not the result of organic disease. When structural disease within the larynx impedes the entrance of air, the respiration is slow and laborious as well as stridulous. With the aid of the laryngeal mirror, I immediately got a good view of her larynx, which I found free from redness, swelling, or other structural change; but I saw that by an irregular action of the muscles the glottis was partially closed during inspiration; hence arose the laryngeal stridor. She told us that on several occasions during the last seven years she had suffered from similar attacks, lasting from one to six weeks. She had also

[1] *British Medical Journal*, May 6, 1871.

experienced other symptoms of a nervous character. Thus four and a half years ago, she had partial loss of power in the left arm, which continued for some weeks, and then gradually passed away. Two years later, she had a similar affection of the right leg, for which she was, after some time, admitted here under my care. She gradually regained power in her leg, but even now it is somewhat weaker than the other leg.

From this history it is evident that our patient is of hysterical temperament, and there could be little doubt that her laryngeal symptoms were the result of spasm. In order to test this still further, we put her under the influence of chloroform; and we found, as we anticipated, and as we have before seen in similar cases, that, as soon as the chloroform took effect, the breathing became quiet and entirely free from stridor. The obstruction in the larynx, having been the result of muscular spasm, was entirely removed by the relaxing influence of the chloroform. We now sent her into the ward and prescribed ten grains of chloral every six hours. After she had taken a few doses of the medicine, the breathing became quiet and the stridor ceased. After the cessation of the laryngeal spasm and stridor, her voice remained feeble and whispering; she had hysterical aphonia. For this symptom we galvanised the larynx. The first application of galvanism did not restore the voice; after the second sitting the voice returned for a few hours, but again it became a mere whisper; and it was not until after a third and fourth application of the galvanism that the voice was permanently restored to its natural tone and strength.

In some cases of nervous aphonia the galvanic stimulus instantly restores the voice. The day on which this patient was admitted a young woman applied to us on account of loss of voice, which had continued for six weeks. Seeing that her larynx was quite healthy, we applied galvanism; she uttered a loud scream, and on the instant her voice was restored.

Now let me remind you that cases of spasm of the larynx arrange themselves in various groups.

1. There are the cases of hysterical laryngismus, of which our patient, H. W——, affords a good illustration.

2. We have laryngismus stridulus, or crowing inspiration

of children, closely allied to which is the laryngeal spasm associated with the epileptic cry and convulsion.

3. Laryngeal spasm, the result of pressure on the pneumogastric nerve or its recurrent branch by an aneurysm or other tumour in the neck or within the chest.

4. Lastly, laryngeal spasm may result from direct irritation of the larynx by the presence of a foreign body, by the inhalation of irritating gases or dust, or by inflammation of its mucous membrane. Much of the distress which results from catarrhal laryngitis in persons of nervous excitable temperament, and especially in children, is a result of laryngeal spasm.

Amongst the remedies for spasm of the larynx, from whatever cause arising, chloral occupies a foremost place. Its operation is similar to that of the vapour of chloroform, but it has the advantage of a much more durable action, so that by regulated doses the patient may be kept continuously under its influence. Chloral is not only the best antidote for the purely spasmodic affections of the larynx, such as hysterical laryngismus, and the laryngismus stridulus of children, but also, combined with other remedies, for the spasm which often complicates and aggravates laryngitis. In cases of inflammatory croup, the combination of chloral with ipecacuanha is most beneficial. As yet I have had no opportunity of giving chloral in a case of spasm excited by pressure on the pneumogastric nerve, but even in these painful cases it will probably be found useful as a palliative.

The case of aneurysm from which the specimen on the table is taken occurred before we had learnt the therapeutic value of chloral. J. H——, aged 31, a hawker, of intemperate habits, was admitted under my care on June 16, 1863. About seven weeks before, he first experienced a sensation of stoppage in his throat, and lost his voice. These throat-symptoms continued until the time of his admission. For eight days prior to admission he had experienced difficulty in swallowing solids. The symptoms on admission were cough, with dyspnœa and loud laryngeal stridor, hoarseness, and difficulty in swallowing solids. On laryngoscopic examination, I found the mucous membrane of the larynx slightly congested,

but there was no swelling, the arytænoid cartilages were freely movable, and the glottis opened wide during inspiration. At the time of the examination the breathing was not stridulous. It was evident, from this examination, that the laryngeal symptoms were not the result of laryngitis or of other structural disease within the larynx. Aneurysm of the aorta was suspected and carefully sought for; but there was no physical sign of aneurysm, no dulness on percussion, no pulsation or abnormal murmur, and the pulse at the two wrists was equal.

On the 18th, two days after his admission, he brought up a few mouthfuls of florid blood.

On the 20th, at 1.15 P.M., he suddenly became faint, and vomited about three-quarters of a pint of florid blood. When I saw him at 1.45 P.M., he was still pale and faint, as if from internal hæmorrhage. No more blood had been vomited, but there was dulness on percussion over the stomach, and it seemed probable that the stomach was becoming filled with blood. He continued to sink gradually, and died at 5 P.M. An aneurysm of the size of a small orange was found at the back of the transverse portion of the arch of the aorta. The aneurysm had opened into the œsophagus, and the stomach was full of blood. The left pneumogastric nerve passed in front of the aneurysm, and was somewhat flattened by pressure, while the recurrent branch passed behind the tumour in its course upwards to the larynx. The appearances are well preserved in the preparation before you.

Many years ago, I was asked by a friend to examine the body of a man who died of what was believed to have been acute laryngitis. I found the larynx quite healthy, but in the anterior mediastinum there was a cancerous tumour, which involved the pneumogastric nerves. The laryngeal symptoms had been the result of spasm, and not of inflammation.

Before the introduction of the laryngoscope, it was often difficult and sometimes impossible to distinguish between the various forms of spasm and of structural disease within the larynx; now, with the aid of the mirror, we rarely have either doubt or difficulty in the diagnosis of this important class of diseases.

CHAPTER XXV.

ON THE LARYNGEAL SYMPTOMS WHICH RESULT FROM THE PRESSURE OF AN ANEURYSMAL OR OTHER TUMOUR UPON THE VAGUS AND RECURRENT NERVES.[1]

Two Cases of Bilateral Palsy of the Laryngeal Muscles from the Pressure of an Aneurysm on the Trunk of one Vagus—Theoretical Explanation—Results of Electrical Stimulation of the Laryngeal Nerves—Contrasted Results of Pressure on one Vagus and on one Recurrent Nerve—Question of Tracheotomy—Summary of Conclusions—Dr. Weir Mitchell and Dr. Morehouse on the Laryngeal Nerves of the Turtle.

Amongst the practical gains which have resulted from the use of the laryngoscope, one of the greatest has been the increased facility which the inspection of the living larynx has afforded for investigating the results of disease implicating the vagus and recurrent nerves.

The main object of the present communication is first to demonstrate and then to explain the fact, that *bilateral spasm* and *bilateral palsy* of the intrinsic muscles of the larynx may result from the pressure of an aneurysm or other tumour *on the vagus nerve of one side only*.

Two cases of bilateral palsy of the larynx, with an aneurysm pressing on one vagus and recurrent nerve, have recently been recorded: one by Dr. Baümler, in the twenty-third volume of the *Pathological Transactions*; the other by myself, in the twenty-fourth volume of the same *Transactions*. My own case[2] was that of a man 45 years of age, who for several months had been suffering from stridulous and difficult breathing, the voice being feeble but tolerably clear.

[1] From vol. lviii. of the *Medico-Chirurgical Transactions*, published by the Royal Medical and Chirurgical Society of London.

[2] *Path. Trans.*, xxiv. p. 42.

Looking into his larynx with the mirror, I saw the vocal cords of their natural colour, nearly touching each other in the middle line, and nearly motionless. There was a slight approximation of the cords during vocalisation. During inspiration the glottis did not expand as in the normal state, but, on the contrary, the cords appeared to be pressed nearer together by the inspiratory current of air, while in expiration, again, the cords were slightly pushed apart by the outgoing stream of air. No swelling or other structural change within the larynx was visible. There was dulness on percussion over the *manubrium sterni*—an impulse was heard there at each systole of the heart, and an impulse was felt by the ends of the fingers, when firmly pressed against the bone. The patient stated that eighteen months before, he had suddenly become hoarse while talking to a friend; the feebleness of voice had continued, and the breathing had gradually become difficult and attended with a noise in the throat. He had, during the last eleven months, been an out-patient at a special throat hospital, where he had been treated by repeated local applications to the interior of the larynx. For some months past the breathing had become so difficult, especially when lying down at night, that he had been unable to sleep for more than a few minutes at a time. I came to the conclusion that the intrinsic muscles of the larynx on both sides were paralysed by the pressure of an aneurysm of the aorta on one or both recurrent nerves.[1]

During the first night after his admission into the hospital he got no sleep, and several times he appeared to be on the verge of suffocation. The following day Sir William Fergusson, at my request, performed tracheotomy. The operation afforded immediate and great relief, and the next night the patient slept for several hours. He had been greatly exhausted by the long-continued dyspnœa and deprivation of sleep. Two days afterwards, symptoms of pleuro-pneumonia set in, and he died on the fourth day after the operation. An aneurysm about the size of an orange projected backwards

[1] The close approximation of the cords was the result of the paralysis of the abductors—the posterior crico-arytænoid muscles—while the feebleness of voice was due to the weakening of the tensors and adductors.

from the transverse aorta. The left vagus nerve passed in front of the aneurysm, and was closely involved in its wall; the left recurrent passed round and behind the tumour, where it was compressed and atrophied, and nearly lost in the wall of the aneurysm. There were some enlarged lymphatic glands near the right recurrent, but the nerve was not pressed upon or even touched by these glands, and both it and the right vagus appeared quite normal.[1]

My friend and colleague Dr. Curnow did me the favour to dissect out the nerves and muscles of the larynx, and he reported as follows: —'The laryngeal muscles on the left side are decidedly atrophied; those on the right side are somewhat larger, but I am inclined to think them atrophied also.'

In Dr. Baümler's case,[2] with the physical signs of aneurysm of the innominate artery, there was dyspnœa with loud laryngeal stridor and aphonia. The laryngoscope showed complete immobility of the right vocal cord, with immobility, almost as complete, of the left cord. After death, which occurred from increasing dyspnœa, the right vagus was found flattened, and in one place almost lost in the wall of an aneurysm of the innominata; the recurrent branch was also flattened and thinner than that on the left side. The left vagus and recurrent appeared perfectly normal everywhere. The intrinsic muscles of the larynx were very pale and flabby, and there was no appreciable difference in the bulk of the muscles on the two sides. The microscope showed granular degeneration of the muscles on both sides to an almost equal extent.

Dr. Baümler mentions first amongst the points of interest in this case 'the absence of changes in the left recurrent, although during life the corresponding muscles showed, on laryngoscopic examination, very great impairment of their function.'

In both these cases of aneurysm the fact of bilateral palsy of the larynx was ascertained by a careful and thorough laryngoscopic examination during life; in both cases the laryngeal obstruction was greater than an affection of the muscles on one side only would account for; in both cases the *post-*

[1] The preparation is in the museum at King's College.
[2] *Path. Trans.*, xxiii. p. 66.

mortem examination was made with the expectation of finding that the recurrent nerve on both sides had been injured, but in both instances it was found that the vagus and recurrent on *one* side only had been compressed; and, lastly, in both cases the intrinsic muscles of the larynx on both sides had undergone atrophic changes. In my own case the muscular atrophy was greater on the side of the compressed nerve, but in Dr. Baümler's case the wasting of the muscles appeared to be equal on the two sides.[1]

For a long time the bilateral palsy of the larynx which unquestionably existed in these two cases appeared to me inexplicable; and I often discussed it with my physiological friends, without obtaining a satisfactory explanation; but at length it occurred to me to suggest the following interpretation of the phenomena.[2]

The aneurysm in each case compressed, not only the recurrent branch, but also the trunk of the vagus; it seems, therefore, not improbable that while the muscles on one side of the larynx were paralysed by direct pressure on the recurrent branch on that side, the paralysis on the other side was the result of a centripetal irritation of the trunk of the vagus, acting on the nervous centre, and through it upon the nerve-supply to the laryngeal muscles on the opposite side; so that the palsy on one side was direct, while that on the other side was the result of a reflex influence.[3]

I now proceed to adduce in support of this theory such facts and arguments as I have been able to gather from clinical observation, and from the results of experiments on living animals.

It is an unquestionable fact that the intrinsic muscles of

[1] Later researches have shown that it would have been of interest to ascertain whether the atrophy of the *abductors* was greater than that of the other laryngeal muscles.

[2] *British Medical Journal*, June 27, 1874.

[3] Since the publication of these two cases there have been recorded three others in which pressure upon, or injury of one vagus has caused bilateral palsy of the laryngeal muscles—one case by Dr. McCall Anderson (*Edinburgh Medical Journal*, July 1881), one by Dr. Whipham (*Path. Trans.*, vol. xxxiii. p. 82), and a third by Professor Sommerbrodt (*Berliner klin. Wochenschr.*, No. 50, 1882). In this case the left vagus was injured by tincture of iodine injected into a strumous tumour in the neck.

the larynx are in pre-eminent degree, bilateral in their action. Everyone who has inspected the living larynx knows how absolutely impossible it is, in the normal condition, to move one vocal cord without, at the same time, moving the other to an equal extent.

According to Dr. Broadbent's ingenious and well-known hypothesis, the muscles which thus act bilaterally must have their central nerve nuclei so closely connected by commissural fibres that the muscles of each side receive their nerve supply from both sides of the brain, in proportion to the completeness of their bilateral action. The muscles of the larynx, which are in an especial degree bilateral in their action, in accordance with this hypothesis, must be, on either side, equally connected with both sides of the nervous centre. The central commissural connection between the nerves supplying the larynx is not a mere hypothesis or a physiological inference, but a demonstrated anatomical fact. Dr. Lockhart Clarke in his elaborate paper 'On the Intimate Structure of the Brain'[1] has described and figured some of the fibres of origin of the spinal accessory nerve, decussating in three places across the median line of the medulla oblongata, and thus connecting the spinal accessory nuclei of the opposite sides. The spinal accessory nerve is known to be the source of the motor fibres in the laryngeal branches of the vagus; and in the structural arrangement thus demonstrated by Dr. Clarke, whereby the spinal accessory nuclei of the two sides are brought into close union with each other, we appear to have the explanation of the normal bilateral action of the laryngeal muscles. This commissural union of the nerve nuclei of bilaterally acting muscles explains certain well-known pathological phenomena.

1. As Dr. Broadbent has shown, it explains the fact that in cases of hemiplegia resulting from a lesion of one hemisphere of the brain, the muscles which act bilaterally are not paralysed. The reason is, that although the motor influence from one side of the brain to these muscles is cut off or lessened, the motor influence from the other side reaches the commissural centre, and thence passes to the muscles on both sides. Dr. Broadbent explains it thus: 'If the centre of

[1] *Philosophical Transactions*, 1868, Part I. pp. 272, 273.

volitional action on one side is destroyed, or one channel of motor power is cut across, the other will transmit an impulse from the motor centre, and this will be communicated to the nerves of the two sides; equally if the fusion of the two nuclei is complete, and there will be no paralysis; more or less imperfectly to the nerve of the affected side if the transverse connection between it and its fellow is not so perfect, in which case there will be a corresponding degree of paralysis.'[1]

The connection between the central origin of the nerves supplying the laryngeal muscles must be most intimate; for in cases of hemiplegia, the result of a lesion of one cerebral hemisphere, however complete may be the palsy of the arm and leg, the muscles of the larynx appear to be quite unaffected by the paralysing influence. The voice, as a rule, is unchanged in these cases; and the laryngoscope shows an equally unimpeded movement of the cartilages and the vocal cords on both sides. In all cases of unilateral palsy of the laryngeal muscles the paralysing lesion must be below the junction of the nerve nuclei.[2]

2. It is evident that the commissural union of the nerve nuclei, which explains the escape of bilaterally-acting muscles from *palsy*, in cases of hemiplegia resulting from unilateral brain disease, renders these associated muscles liable to *spasm* when a morbid motor influence proceeds from either side of the nervous centre. Thus, in cases of hemispasm—spasm, that is, of one arm and leg, starting from a lesion of one cerebral hemisphere—the bilaterally-acting muscles of the chest and abdomen on both sides are liable to be, and are in fact, spasmodically affected; so the epileptic cry which is sometimes heard at the commencement of a convulsive attack, is a result and a proof of bilateral spasm of the larynx. In these cases the morbid motor influence reaches the commis-

[1] *British and Foreign Medico-Chirurgical Review*, April, 1866.

[2] The researches of Krause and of Semon and Horsley on the phonatory centres in the cortex have since corroborated this theory; inasmuch as they have shown that there is an independent representation of the phonatory function in each of the two hemispheres. I have reason to believe that my theoretical explanation of bilateral palsy of the laryngeal muscles in the cases under consideration is now accepted as satisfactory by the leading laryngologists throughout the world.

sural fibres, and thence passes to the nerves and muscles on both sides. Thus the same commissural union which protects bilaterally-acting muscles from the paralysing influence of a one-sided brain lesion, renders them liable to be implicated in convulsive attacks resulting from a disease on either side of the brain.

3. It is obvious that, if the nerve nuclei and the commissural fibres which connect them become diseased, bilateral palsy may result. And this is what probably happens, as a result of a morbific influence conveyed to the nerve centres, when the trunk of one vagus has been, for a long time, irritated by the pressure of an aneurysm or other tumour.

Many cases are recorded—I have myself seen several—in which a tumour, aneurysmal or cancerous, pressing on one recurrent nerve, has caused unilateral palsy of the intrinsic muscles of the larynx. The explanation of these cases is obvious. The conducting power of the efferent nerve is impaired or destroyed, and, as a necessary result, the muscles on that side are paralysed. It is equally obvious that pressure on one recurrent nerve alone—a nerve which is purely motor, and distributed only to the muscles on its own side—will not, either directly or indirectly, paralyse the muscles on the opposite side of the larynx. But it is, to say the least, extremely probable that pressure on the trunk of one vagus may, through an influence conveyed by its afferent fibres to the nerve centre, produce bilateral *palsy*, as in the two cases whose histories I briefly gave at the commencement of this chapter; while in other cases, to which I shall hereafter refer, bilateral *spasm* of the larynx may be excited through the same nervous channels.

It occurred to me that some light might be thrown upon this subject by experiment; and my friend and former colleague, Professor Rutherford, did me the favour to perform some experiments on living rabbits, with results which I will now briefly describe. In all the experiments, the animal being fixed on his back, the larynx and trachea were exposed, and the anterior wall of the trachea just below the larynx was cut away, so that the glottis could readily be seen from below.

The superior laryngeal nerve was exposed and divided, and its central end was stimulated by a faradic current; the immediate result was a strong bilateral adduction of the vocal cords; in other words, the glottis was spasmodically closed. This experiment was performed several times on four rabbits, and with uniform results. After we had performed this experiment, we found that the same results had before been obtained by other experimenters; in particular by Rosenthal,[1] and by Waller and Prevost.[2] The explanation is obvious—a stimulus is sent through the afferent laryngeal nerve to the centre, and thence is reflected by the efferent fibres of the two vagi, through the recurrent branches, to the muscles of the larynx on both sides. This experiment, then, affords a good illustration of the physiological mechanism of bilateral spasm resulting from unilateral irritation.

The principle is probably the same, whether a morbid motor influence passes downwards from one side of the brain, or upwards through the afferent fibres of one vagus, to the common centre of both vagi.

When the recurrent nerve on one side was stimulated, the vocal cord on the same side was abducted, while the opposite cord was unaffected.

Another experiment consisted in cutting across the trunk of the right vagus between the superior and the recurrent laryngeal nerves, and then applying the electrical stimulus to the lower end; the result was abduction of the vocal cord on that side, while the cord on the opposite side remained motionless. Rosenthal found that this experiment performed on a cat caused unilateral adduction of the vocal cord.[3] Whether abduction or adduction result from the stimulation of the recurrent or the distal end of the vagus depends probably upon the relative vigour of the antagonistic intrinsic muscles of the larynx.

When the central end of the right superior laryngeal was stimulated after the trunk of the vagus on the same side had

[1] *Die Athembewegungen und ihre Beziehungen zum Nervus vagus*, Berlin 1862, pp. 224, 225.

[2] *Archives de Physiologie*, tome iii. 1870, p. 196.

[3] *Loc. cit.*, pp. 211, 212.

been divided. the vocal cord on the opposite (left) side was adducted by the reflex nervous current, while the cord on the same side remained motionless, in consequence of the efferent fibres in the trunk of the right vagus being cut across.

When the central end of the divided vagus is stimulated, the result is not adduction of the opposite vocal cord, as when the superior laryngeal is irritated, but Rosenthal observed that the diaphragm and other inspiratory muscles are made to contract, and with this inspiratory action the vocal cord is abducted.[1] Waller and Prevost also obtained the same results from the excitation of the central end of the vagus.[2]

These experiments serve to establish two general principles:—

1. Any movement of the larynx which results from stimulation of the afferent fibres or branches of one vagus is bilateral, except that, of course, division of the trunk of the vagus will prevent the occurrence of the reflex action on that side.

2. Stimulation of the efferent fibres of one vagus—the recurrent branch or the distal end of the divided trunk—causes movement on the one side only of the larynx.

In the course of our experiments we were enabled to verify an observation made long since by Longet[3]—namely, that after the intrinsic muscles of the larynx have been paralysed by the division of all four laryngeal nerves, the glottis is closed by the compressing action of the inferior and middle constrictors of the pharynx during the act of deglutition. When, after all the laryngeal nerves are divided, the action of the constrictors of the pharynx has been suspended by the division of their fibres on one side, the glottis remains motionless during attempts at deglutition. Electrical stimulation of the central end of the superior laryngeal nerve always excites the act of deglutition, as Waller and Prevost[4] have especially pointed out. We observed distinctly, as a result of such stimulation, that the vocal cords moved inwards and rapidly closed the glottis before the slower act of deglutition had commenced;

[1] Rosenthal, p. 214. [2] *Loc. cit.*, p. 196.
[3] *Archives Générales de Médecine*, tome xii. 1841, p. 423.
[4] *Archives de Physiologie*, tome iii. 1870, p. 185.

but in two animals Dr. Rutherford divided the constrictors of the pharynx before the experiments on the superior laryngeal nerve were commenced; the object of this procedure being to enable us the better to distinguish the closure of the glottis by the constrictors of the pharynx during the act of deglutition, from that which is caused by the intrinsic muscles of the larynx.[1]

It is impossible by any experiment upon an animal to imitate all the influences which are in operation when an aneurysm is gradually but constantly compressing the vagus and recurrent nerve in the human subject; but the results of the various experiments to which reference has been made, give support and probability to the theory that a long-continued irritation of the trunk of one vagus may, through its afferent fibres, so disturb and disorganise the common centre of the two vagi, as to cause either bilateral spasm or bilateral palsy of the laryngeal muscles.

Reference may here be made to some well-known facts, illustrating the influence of a peripheral irritation in exciting pathological changes in distant parts through a reflex nervous influence.

Cases of traumatic tetanus afford one good illustration of this. A foreign body in a wound may not only excite a general spasm of the muscles, through an influence conveyed to the spinal cord, and thence reflected through the efferent nerves to the muscles, but in fatal cases, as Dr. Lockhart Clarke has

[1] During the last few years the results of electric stimulation of the laryngeal nerves have been studied by various experimenters. An admirable summary of the results, with a record of numerous and most interesting experiments of their own, has been published conjointly by Dr. Felix Semon and Mr. Victor Horsley, *On an Apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles*, 1886. The authors refer to the results obtained by the American observers, Dr. Hooper and Dr. Donaldson, and they show that in conducting those experiments there are many difficulties and sources of error. The results obtained from animals of different genera, and even from different individuals of the same genus, vary considerably. The opposite results of abduction or adduction of the vocal cord when the recurrent is stimulated, depend upon the strength of the electric current, and also upon the depth of the narcotism caused by etherisation. They conclude by expressing their hope that they have made clear for future investigation the causation of some of the difficulties.

demonstrated, the spinal cord is found to have undergone extensive structural changes.

In the sixth volume of the *Transactions of the Clinical Society* I have published a case in which a piece of flint beneath the skin, in a partially healed wound on the cheek, excited facial neuralgia with facial palsy on the same side, and trismus. These results of a reflex nervous influence all passed away after the removal of the foreign body which had unquestionably been their exciting cause.[1]

During the passage of a renal calculus through the ureter there is often pain in the testicle on the same side, and sometimes there is inflammatory swelling of the painful part. I extract the following from Sir James Paget's *Surgical Pathology*:—'Whoever has worked much with microscopes may have been conscious of some amount of inflammation of the conjunctiva in consequence of overwork. Now, the stimulus exciting this inflammation has been directly applied to the retina alone; and I have often had a slightly inflamed left conjunctiva after long working with the right eye, while the left eye has been all the time closed. I know not how such an inflammation of the conjunctiva can be explained, except on the supposition that the excited state of the optic nerve is transferred or communicated to the filaments of the nerves of the conjunctiva, generating in them such a state as interferes with their nutrition.'

It is a well-known fact that a foreign body lodged in one eyeball may, by a disturbing influence transmitted through the nerves and the nervous centre, excite destructive disease in the other eye; and it is a common and a successful practice to extirpate the primarily injured eyeball, to prevent the induction of a secondary and sympathetic disease in its fellow.

The more intimate the nervous connection between two parts, the greater is the probability that disease in one may excite sympathetic disorder in the other; the probability, then, is very great that irritation of the trunk of one vagus will gradually excite functional disorder, and even structural change, in the nervous centre and in the associated nerve on the opposite side.

[1] For the history of this case see Chapter XVIII. p. 332.

Clinical observation has established the fact that laryngeal symptoms of some kind occur in a large proportion of cases of aneurysm of the transverse aorta. Dr. Sibson has shown, in his elaborate analysis of the symptoms of aneurysm of the aorta, that in cases of aneurysm affecting the transverse portion of the arch, the voice or cough was raucous or whispering, or inspiration was stridulous in 47·5 per cent.[1]

In the history of cases of aneurysm, symptoms are commonly attributed to pressure on the recurrent nerve, which could not possibly result from the implication of that nerve alone.

The recurrent, be it remembered, is an efferent motor nerve. When galvanised, as we saw, muscular contraction occurs only on the one side; no reflex bilateral contraction occurs, as when the afferent superior laryngeal nerve is stimulated. Pressure on one recurrent nerve may cause either unilateral spasm or unilateral palsy of the laryngeal muscles. A unilateral palsy of the laryngeal muscles may render the voice somewhat feeble, but it does not narrow the opening of the glottis sufficiently to seriously impede the breathing. I have seen a considerable number of cases in which the laryngoscope has shown unilateral palsy of the larynx, but in not one has distress of breathing been occasioned thereby. Dr. Bristowe, in an interesting paper in the *St. Thomas's Hospital Reports*,[2] refers to two cases in which unilateral palsy of the larynx was caused by the implication of one recurrent nerve in a cancerous tumour; but he states that in neither case 'was there a trace of dyspnœa, either persistent or paroxysmal.' The loud laryngeal stridor and the urgent dyspnœa which so frequently result from aneurysms and other tumours within the chest may be due to one of three conditions—1st, bilateral spasm of the laryngeal muscles, the result of irritation of one vagus; 2nd, bilateral palsy of the abductors, probably a later result of a tumour pressing on one vagus and recurrent; 3rd, palsy of the abductor on one side, a direct result of pressure on the recurrent, with spasm of the adductor muscles on the opposite side, a reflex result of pressure on the trunk of the vagus.

[1] *Medical Anatomy*, Fasciculus 5.

[2] New Series, vol. iii. p. 205.

When the dyspnœa and stridor are paroxysmal, coming on and going off with equal rapidity, the immediate cause is probably bilateral *spasm* of the larynx; and these attacks bear a striking resemblance to cases of laryngismus stridulus in children. On the other hand, long-continued laryngeal stridor and dyspnœa, are commonly caused by *paralysis* of the abductor on both sides. The laryngoscope affords most valuable aid in the diagnosis of these interesting cases.

It is possible that violent and prolonged spasm of the laryngeal muscles may sometimes be directly followed by a paralytic condition, during which the stridor and dyspnœa continue, and death at length occurs from suffocation. This sequence of pathological events would seem to be analogous to the palsy which follows a fit of epilepsy, when the convulsed limbs are left paralysed. Dr. Hughlings Jackson ingeniously suggests that the violent physiological discharge of nerve-force through the motor fibres during the epileptic paroxysm may, for a time, suspend their functional activity, and thus cause the so-called 'epileptic hemiplegia.'

There is reason to believe that a bilateral affection of the larynx, either spasmodic or paralytic, is a very common result of pressure on the trunk of one vagus. The records of medicine abound with histories of cases in which death has resulted from laryngeal obstruction, and in which a *post-mortem* examination has shown a tumour pressing on one vagus or on the vagus and its recurrent branch. I know of no case in which death has resulted from pressure on *one recurrent* nerve alone; but Dr. John Reid stated that he had in his possession 'a preparation procured from the body of a young man who died very suddenly, with all the symptoms of suffocation, when seated with some companions round a fire, who were chatting and laughing. *Both* recurrents are imbedded in a firm yellowish tumour, through which they cannot be traced.'[1]

It is not to be supposed that a unilateral muscular affection of the larynx could cause suffocation. In all the fatal

[1] *Physiological, Anatomical, and Pathological Researches*, p. 275. In volume xxxiii. of the *Pathological Transactions* (1882, p. 38), Dr. Felix Semon has published a case of bilateral palsy of the abductors, the result of both recurrent nerves being involved in a malignant (?) tumour of the thyroid gland.

cases of this kind, there must have been either a bilateral spasm or a bilateral palsy of the laryngeal muscles.

With reference to the question of tracheotomy in cases of aortic aneurysm, obviously, the main point is to distinguish between laryngeal obstruction, the result of spasm or palsy caused by pressure on the vagus, and narrowing of the trachea, by the direct pressure of an aneurysm on the anterior wall of that tube. In the former class of cases tracheotomy may afford great temporary relief; as in the case recorded at the commencement of this paper, while in cases of obstruction of the trachea near its bifurcation, the operation would be worse than useless.[1]

One fact which we observed in the course of our experiments is worthy of note, on account of its bearing upon the therapeutics of laryngeal spasm. We found, on two occasions, that when a rabbit was under the influence of chloroform, the electric stimulus applied to the central end of the divided superior laryngeal nerve did not excite spasmodic closure of the glottis, although the slight respiratory movement of the cords continued. After the effect of the chloroform had passed away, the same electrical stimulus caused the usual bilateral spasm of the glottis. Waller and Prevost also found that anæsthesia by ether or chloroform arrested the reflex movements which are ordinarily excited by the electrical stimulus applied to the central end of the superior laryngeal nerve in the cat.[2] This antispasmodic effect of chloroform vapour is similar to the undoubted influence which chloral hydrate exerts in preventing or lessening laryngeal spasm—whether in the purely spasmodic attacks of laryngismus stridulus or in cases of laryngitis with spasmodic complication, such as occurs

[1] Dr. Felix Semon has directed attention to the fact that there may be, as he says, 'a double stenosis' (*Path. Trans.*, vol. xxxiii. p. 43). In addition to the obstruction resulting from paralysis of the laryngeal muscles, the trachea and main bronchi may be so compressed by the aneurysmal or other tumour as to cause suffocation, notwithstanding the operation of tracheotomy. He gives particulars of two of his own cases, and refers to another case recorded by Dr Whipham in the same (thirty-third) volume of the *Transactions*, in which, this double stenosis existing, the patients died of apnœa soon after the operation of tracheotomy, which had afforded only partial and temporary relief.

[2] *Archives de Physiologie*, tome iii. 1870, pp. 188, 189.

very frequently in children and sometimes also in adults of both sexes.

The main points which I have endeavoured to establish in this communication are the following :

1. When serious laryngeal dyspnœa results from the pressure of a tumour on the vagus and its branches, the immediate cause is a bilateral affection of the muscles of the larynx. There may be bilateral spasm or bilateral palsy, or palsy on one side with temporary spasm of the muscles on the opposite side.

2. Pressure on one recurrent nerve can affect the laryngeal muscles on the one side only.

3. Pressure on the trunk of the vagus may, through an influence upon the nervous centre, cause either bilateral spasm or bilateral palsy of the larynx ; and this is the true physiological interpretation of the urgent laryngeal symptoms which are often excited by the pressure of aneurysmal and other tumours upon the vagus.

4. The bilateral spasm of the larynx which is excited by the pressure of a tumour on the trunk of one vagus is, probably, the result of a morbidly exalted excitability of the nervous centre induced by irritation of the afferent fibres of that nerve ; and in consequence of this state of exalted sensibility, ordinary stimuli, acting on the afferent nerves of the larynx, may cause bilateral spasm, similar to that which is excited by the extraordinary stimulus of electricity applied to the central end of the divided superior laryngeal nerve.

5. It is probable that the long-continued irritation of the trunk of the vagus may gradually, as in cases of traumatic tetanus, induce such demonstrable structural changes in the nerve-centre as will explain the bilateral palsy, which appears to be one of the results of this chronic nerve-irritation.

In any future cases of this kind that may occur and prove fatal, after the laryngoscope has shown the existence of a bilateral palsy of the larynx, it will be desirable to subject the medulla oblongata and the nuclei of the spinal accessory and vagi nerves to a careful microscopic scrutiny. If the result of such an examination should be the discovery of structural

changes in the nervous tissue, the evidence in support of my theory will be complete.[1]

Postscript.

Dr. Weir Mitchell, of Philadelphia, has done me the favour to send me a copy of a paper published by himself in conjunction with Dr. Morehouse.[2] In this paper the authors describe a peculiar arrangement and distribution of the laryngeal nerves in the turtle, which appear to have an especial interest in relation to the subject of the preceding communication. In the turtle the movements of the glottis are effected by two pairs of muscles; by one pair of muscles the glottis is opened, while by another pair it is closed. The glottic muscles are supplied by two pairs of nerves, a superior laryngeal and an inferior laryngeal or recurrent. The superior laryngeal nerves supply both the opening and the closing muscles of the glottis, while the inferior or recurrent laryngeal are distributed only to the opening muscles of the glottis. The superior laryngeal nerve, in addition to being the chief motor nerve of the larynx—in this respect being analogous to the recurrent in the higher animals—is proved by experiment to be the sensitive nerve of the larynx in the turtle. But the remarkable arrangement which Drs. Mitchell and Morehouse have discovered consists in the existence of a true *chiasm*, or decussation of fibres, between the two superior laryngeal nerves; as a result of which each superior laryngeal nerve contains fibres from both sides of the nervous centre. This nervous chiasm resembles that of the optic nerves, and is, the authors believe, the only example of this arrangement yet observed in extracranial nerves. The authors were led to the discovery of this inter-communication between

[1] Dr. Morell Mackenzie, referring to this subject in his work *On Diseases of the Larynx* (p. 442), says that my 'explanation has not been generally accepted,' and then, offering what he considers a better explanation, he says: 'It is more probable that in such cases as that reported by Dr. Johnson central disease is set up, and the nuclei of the spinal accessory nerve become actually diseased.' It is to be regretted that the author of this suggestion had not done me the justice to read my paper before venturing to correct and to supplement the assumed defect in my theory.

[2] *Researches upon the Anatomy and Physiology of Respiration in the Chelonia. Smithsonian Contributions to Knowledge*, Washington, 1863.

the superior laryngeal nerves by the results of experiment. They found that after both the inferior laryngeal and one of the superior laryngeal nerves had been divided, the movements of the glottis continued unimpaired. They also found that faradisation of the distal end of the divided superior nerve caused bilateral movement of the glottis. This result suggested the existence of some connection between the nerves of opposite sides. The inter-communicating fibres were then sought for and discovered in front of the larynx. It was found that, after dividing the chiasm, division of one superior laryngeal nerve caused palsy of the glottic muscles on the same side. It thus appears that in the turtle the bilateral action of the laryngeal muscles is effected or assisted by such an inter-communication between the two principal motor nerves as gives to the muscles, on either side, an equal nerve supply from both sides of the nervous centre. In the higher animals this union between the motor nerves of the larynx exists at their centre of origin.

Drs. Mitchell and Morehouse observed that, after division of both inferior laryngeal nerves, electric stimulation of the central end of one divided superior laryngeal nerve caused a reflex bilateral movement of the glottis. This shows that in the turtle, as in the higher animals, there is a central connection between the afferent fibres on one side and the efferent fibres on the other.

The correctness of the authors' conclusions as to the function of the laryngeal chiasm, was confirmed by an experiment on one animal, which gave a result, at first sight, contradictory.

In a large turtle, the two inferior laryngeal nerves having been divided, division of the right superior laryngeal caused palsy of the glottis on that side. This result was different from that previously obtained by the like experiment; and on a careful dissection, it was found that the interlateral communicating fibres (the chiasm) were involved in a mass of diseased tissue, which had the same effect as division of the chiasm had been found to have in previous experiments. This observation, which at first appeared to throw doubt upon those which had preceded it, did, in fact, prove the correctness of the authors' previous conclusions.

CHAPTER XXVI.

A CLINICAL LECTURE ON PNEUMONIA.[1]

History of a Case—Explanation of the Symptoms—Blood-poisoning and Fever precede the local Lung Symptoms—Suggestions for cutting short the Disease—Explanation of Pleuritic Pain in Different Stages—Hydrate of Chloral for procuring Sleep and arresting Delirium—Vocal Fremitus over Consolidated Lung—Pneumonia usually limited to one Lobe—Distinctive Signs of Consolidation of an entire Lung and of a copious Pleuritic Effusion.

We have recently had in the hospital a succession of cases of pneumonia. I propose to give you now a condensed history of one typical case, and to make it the subject of some physiological and practical comments.

R. W——, aged 53, cellarman to a wine merchant. Has drunk rather freely, but less so since he lost his wife six months ago.

On June 11, while at work, he felt giddy. This was followed by a sensation of cold, and he had a violent attack of shivering. He went home, and in the evening he had a sudden fit, during which, his daughter says, he was quite unconscious, and foamed at the mouth, but his limbs were not convulsed. The foaming at the mouth continued for about a quarter of an hour, and he remained unconscious for about twelve hours. When he recovered consciousness he was delirious, and continued so until his admission into the hospital on June 14—that is, three days from the commencement of his illness. Here it may be well to mention that, for three days before his illness, he had been working in a cellar which was very cold and damp, in consequence of its walls being covered with fresh cement.

[1] *Medical Times and Gazette*, August 6, 1870.

On the 15th it was noted that he was delirious, and talked incessantly during his sleep. The skin was hot ; temperature 103° ; pulse 104 ; respiration not noted. He had cough with rust-coloured expectoration. There was dulness on percussion, with bronchial breathing and bronchophony over the lower lobe of the right lung. The urine was high-coloured, and contained one-fifth of albumen.

On the 16th the temperature had risen to 104°·2 ; pulse 112. He was perspiring, and still delirious. Physical signs the same. The urine less albuminous—one-sixth.

On the 18th the temperature had fallen to 99°·2 ; pulse 80. Redux crepitation over the right lower lobe. As he continued restless and delirious, he was ordered to take twenty grains of chloral hydrate every four hours.

On the 20th it was noted that he had slept better, and was much less delirious ; crepitation and a creaking friction sound over the right base ; temperature 100°·6 ; pulse 92. To take the chloral three times a day.

On the 22nd.—Slept well ; continues to improve ; no albumen in the urine.

On the 24th the chloral was omitted, and he was ordered to take a quinine and acid mixture. The signs of lung consolidation were passing away, but the friction sound was still audible.

On the 27th the friction sound had ceased, the temperature was normal, and he was convalescent.

It may be well to mention that until the chloral was prescribed he took no medicine. On his admission he had two ounces of brandy in the twenty-four hours ; this was increased to six ounces on the 17th, and again reduced to four ounces on the 22nd. His diet was milk, beef-tea, and eggs.

Now, let us consider briefly the physiological rationale of the symptoms in this case.

I look upon it that before there was any local mischief in the lung, there was blood-poisoning. The precise nature of the blood-poisoning is doubtful, but it may probably have been due to suppressed action of the skin, consequent on the chilling effect of working in a damp cellar. It is possible, too, that the air of the cellar, in addition to being damp and cold,

may have contained organic impurities, the result of defective drainage. Amongst the various acute and serious forms of disease which may be excited by sewer poisons, acute pleuro-pneumonia is well known to be one. The evidence of blood-poisoning in this case consists in the occurrence of rigors, followed by an epileptic seizure, and this again by delirium. Rigors are probably a result of interrupted circulation through the spinal cord, and the immediate cause of epilepsy is a similar interrupted circulation through the brain.[1] To some extent, perhaps, the delirium was a result of his somewhat intemperate habits—it may have been a form of delirium tremens. Another fact which points to blood-poisoning is the occurrence of temporary albuminuria. Again, the high temperature was probably associated with a morbid process in the blood.

One of the most important results of this blood-contamination was the exudation into the lung—the pleuro-pneumonia. Then there quickly followed a fall of temperature.

I have frequently pointed out to you a similar sequence of events in cases of pneumonia, and I have shown you that the exudation into the lung is speedily followed by subsidence of the febrile excitement. The exudation into the lung may be looked upon as analogous to the eruption on the skin in a case of small-pox. In both diseases—i.e. pneumonia and small-pox—a noxious product is thrown out of the circulation, in one case into the lung, in the other into the skin. In both diseases the appearance of the local symptoms is quickly followed by a fall of temperature and a diminution of the general febrile excitement. The structural changes in the lung and in the skin, respectively, give rise to a fresh class of symptoms, and may in various ways cause distress and endanger life.

The great and rapid fall of temperature about the sixth or seventh day of acute pneumonia is thus a part of the natural history of the disease. This fact, however, is not recognised by some practitioners, who occasionally, in publishing cases of the disease, attribute the fall of temperature to the influence of some drug which had been given. In one case I remember,

[1] See Chapters IX. and X.

it was digitalis, and in another ergot, which was supposed to have caused the rapid defervescence.

Now I have a practical object in directing your attention to what I venture to call the physiology of pneumonia, which is not of traumatic origin. A right appreciation of the disease may enable you to prevent or to greatly mitigate the pulmonary symptoms. If we can catch the disease in the stage of the initiatory fever, before the exudation process has commenced, I believe it is possible to prevent the pulmonary mischief, by favouring exudation through other channels. In a large proportion of cases the exciting cause of pneumonia is suppressed action of the skin by a chill or other unknown atmospheric influence, and the main object of treatment should be, as early as possible, to restore the free action of the skin. This may be done in various ways—by a warm-water or hot-air bath, by packing in a wet sheet and blankets, and by the diaphoretic action of repeated small doses of antimony or ipecacuanha, with or without opium. This diaphoretic treatment is often successful in arresting a catarrh, and it may be equally successful in preventing or mitigating pneumonic exudation.

You will observe that vomiting and diarrhœa are amongst the symptoms which occur, not unfrequently, at the commencement of a febrile attack, which results, soon, in inflammatory consolidation of the lung. We may take a hint from these phenomena, and assist in diverting mischief from the lung by an emetic or by a purgative dose of calomel and colocynth, followed by a saline.

The success of this abortive treatment of pneumonia depends mainly upon its early application. It is too late to attempt prevention when the lung tissue is already consolidated by inflammatory exudation. But, inasmuch as before the occurrence of the pulmonary symptoms, the precise nature of the disease must be uncertain, the beneficial results of the treatment will remain doubtful in proportion to the completeness of its success. When the lung tissue has become consolidated by exudation there is no need for active treatment. Expectoration and absorption will, in most cases, more or less rapidly restore the lung to its normal condition. Pleuritic

pain, when present, may be relieved by warm fomentations or poultices, or, if severe, by the application of three or four leeches.

With reference to the pain of pleuro-pneumonia, let me give you one practical hint. The pain may be sharp at the onset of the disease, it may subside entirely when the lung is consolidated, and it may return with some severity when the physical signs indicate that the lung consolidation is passing away. The explanation I believe to be this: the pleurisy which complicates pneumonia is almost always dry pleurisy; there is lymph on the pleura covering the inflamed lung, but as a rule there is little or no serous effusion. The friction of the inflamed and roughened pleural surfaces increases the pain; the consolidation of the lung lessens lung and chest movement; thus friction and pain cease; to return when, with the passing away of the pulmonary consolidation, the lung and chest movements again become free.

You will often observe that with a return of the pleuritic pain, there is a return of the friction sound, which may have been audible during the early stage of pulmonary engorgement, but not during the stage of hepatisation. In the case which I just now read to you the friction sound was heard for the first time when the redux crepitation indicated that the lung consolidation was diminishing. Do not fall into the error of supposing that the redux pain and friction sound indicate a fresh attack of pleuritic inflammation.

This case is one of many in which hydrate of chloral has had a most beneficial influence in allaying delirious excitement and procuring sleep, without, in any way, impeding expectoration or the healing process in the lung. In this respect, therefore, chloral is preferable to any preparation of opium or morphine.

You will remember that in the notes which I just now read, it is stated that there was 'dulness on percussion, bronchial breathing and bronchophony over the lower lobe of the right lung.' My clinical clerk, I find, has omitted to mention whether the vocal fremitus was increased, diminished, or unaltered, as compared with the corresponding lobe of the left lung. It is often stated in text-books that, as a rule, the

vocal fremitus is increased over a lung, or a portion of a lung, consolidated by pneumonia. It is many years since I satisfied myself that this rule is subject to numerous exceptions. The result of my observation is that, while in about half the cases the fremitus over a hepatised lung is increased, in the remaining cases it is either unchanged or it is actually less than over the corresponding part of the healthy lung; and this when a *post-mortem* examination has shown that the pneumonia was uncomplicated by liquid effusion into the pleura. You see, then, that you cannot rely absolutely upon the increase or diminution of the vocal fremitus as a means of distinguishing between consolidation of the lung and liquid in the pleura. Except in the rare case of the lung being kept in contact with the chest wall by firm adhesions, the fremitus is abolished or greatly lessened over a copious liquid effusion in the pleura. On the other hand, over hepatised lung, the fremitus may be increased or lessened, or it may remain unchanged.

Let me remind you of another fact relating to pneumonia, the recognition of which will often assist you in forming a diagnosis. In most cases the disease is limited to one lobe of a lung, and the lower lobe is more frequently affected than the upper. The lower lobe, as you know, is also mainly posterior, and is in contact with the ribs from the spine of the scapula to the diaphragm. The upper lobe extends from the spine of the scapula at the back, and in front, on the left side, from the supra-clavicular region to the diaphragm; while an oblique line extending from the spine of the scapula downwards and forwards separates the upper from the lower lobe in the lateral region. On the right side the position of the lower lobe is the same as on the left, but in front a wedge-shaped portion, cut off, as it were, from the upper lobe, constitutes the middle lobe of that lung. Bearing these anatomical facts in mind, you will find it easy by percussion to define the outline of any one consolidated lobe; and in noting the results of percussion, it may be stated that the extent of dulness corresponds with the upper, lower, or middle lobe, each of which may be separately implicated; though on the right side hepatisation of the middle lobe is generally associated with a similar affection of either the

upper or the lower lobe on the same side. I need not remind you that in some cases the whole of one lung, or even the greater part of both lungs, may be simultaneously inflamed and consolidated.

Suppose, now, that you meet with a case in which you find that over the whole of one side of the chest there is dulness on percussion, by what physical signs could you ascertain whether this dulness is a result of pneumonic consolidation of one entire lung, or of a copious liquid effusion into the pleura? The main points of distinction are these: the pressure of a copious liquid effusion causes a flattening or even a bulging outwards of the intercostal spaces. Liquid in the left pleura displaces the apex of the heart to the right, while effusion on the right side pushes the heart further to the left; and the downward pressure on the diaphragm depresses the liver on one side and the stomach on the other. On percussion it will often be found that the dulness extends beyond the middle line and the edge of the sternum, consequent on displacement of the mediastinum. An increase of the vocal fremitus on the dull side, or an equally pronounced fremitus on the two sides, would indicate solid lung and not liquid in the pleura, by which the fremitus is abolished, except in the rare case of a portion of the lung being maintained in contact with the chest by adhesions. I have before told you that in some, not rare cases, the vocal fremitus is much lessened over a consolidated lung. In such cases auscultation will at once enable you to form a diagnosis. For while, over a hepatised lung, bronchophony and bronchial breathing are everywhere heard, either with or without moist sounds, the effect of a copious effusion into the pleura is to abolish all vocal and respiratory sounds, except such as may be heard indistinctly and, as it were, at a distance, over the root of the lung near the spine.

CHAPTER XXVII.

CLINICAL LECTURE ON A CASE OF PLEURISY.[1]

Brief Abstract of a Case of Pleurisy with copious Effusion—Thoracentesis—Quick Recovery—Two Modes in which a copious Pleuritic Effusion impedes the Absorption of the Liquid—Explanation of the thick Layers of Fibrine in Cases of Pleurisy and Pericarditis—Practical Application of the Doctrine—Albuminous Expectoration after Thoracentesis: its probable Cause.

T. Long, aged 16, an errand-boy, was admitted under my care on June 18. On June 9 he left off his waistcoat, and, in consequence, he got a chill. On the 12th, he first felt pain in the left side, of a dull aching character. The pain was increased by exertion and by a deep breath; and, being unable to continue his work, he went home to bed. The pain continued; he lost his appetite, and felt weak. On the 18th, when admitted into the hospital, the left side of the chest had a rounded form, the intercostal spaces bulged, and the ribs were nearly motionless. The left side measured 14¼ inches, the right 14 inches. The heart was seen and felt beating to the right of the sternum. The whole left side was dull on percussion from base to apex. No respiratory sound was audible, except an indistinct and distant blowing near the spine. Vocal fremitus was absent. On the right side there were normal resonance and puerile respiration. Respirations 34; pulse 120; temperature 101°·4; urine normal. The boy had a pale, delicate, and emaciated look, and a malar flush on the face. Here we obviously had to deal with a very copious liquid effusion into the left pleura; and, after watching the case for a few days, I determined, for reasons which I will presently explain, to have the liquid withdrawn.

On June 26 the house-surgeon, Mr. Duncan, introduced

[1] *British Medical Journal*, Oct. 25, 1873.

a fine canula through the ninth intercostal space, in a line below the angle of the scapula, and drew off with an aspirator forty-one ounces of opalescent fluid. The wound was then closed, and no air was admitted into the pleura. The admission of air into the pleura, if it do not increase the risk of suppuration within the cavity, certainly tends to compress the lung, and so to impede, if not entirely to prevent, its expansion after the removal of the liquid. Within a few minutes after its removal, the liquid formed a firm gelatinous coagulum. The withdrawal of the liquid was attended with immediate relief to the breathing.

Two days afterwards, the left side of the chest had regained its normal form and size, and its movement was nearly as free as that of the right. From that time, his progress towards recovery was continuous and rapid; the normal resonance and respiratory sounds gradually returned; and our last note of him, on July 21, just before he left the hospital, was to the effect that the only remains of abnormal physical signs were some dulness on percussion and feeble respiration below the angle of the left scapula, the result, probably, of false membranes over that part of the lung.

Now, I wish to point out to you that there are two conditions which greatly impede the absorption of the serous effusion of pleurisy. These are—1, so copious an effusion of liquid as to distend the pleural cavity; 2, a thick layer of unorganised fibrine covering the surface of the pleura. A very copious liquid effusion impedes absorption, partly by obstructing the flow of blood through the compressed lung, thereby causing a general fulness of the systemic veins, including, of course, the bronchial veins; partly by directly compressing the subpleural veins, thus retarding the return of blood, and causing capillary engorgement beneath the pleura. When the pressure of liquid is sufficient to cause bulging of the intercostal spaces, such as occurred in this case, it is obvious that the intercostal venous circulation must be seriously impeded. The mechanical withdrawal of a sufficient amount of the liquid effusion to relieve tension of the cavity, and thus to remove pressure from the lung and the veins beneath the pulmonary and the costal pleura, will usually be followed by a quickened absorption of the liquid

which remains in the pleura. In like manner, when anasarcous swelling of the legs has rendered the skin so tense as to impede the return of blood by the veins, and thus to favour the increase of the dropsical swelling, the discharge of some liquid through a puncture in the skin is usually followed by the absorption of a further portion of the dropsical effusion, which, entering the circulation, exerts a diuretic influence upon the kidney, and excites a copious flow of urine.

There is no difficulty in understanding that a thick layer of fibrine covering the surface of the pleura, and therefore interposed between the subpleural vessels and the liquid effusion, must greatly impede the absorption of the liquid. In some cases of pleurisy, with copious liquid effusion, the pleura has been covered by firm unorganised fibrine, from a quarter to half an inch in thickness. Now I wish to show you that an exact knowledge of the mode in which a thick layer of fibrine is formed upon an inflamed pleura affords a powerful argument for early tapping in cases of pleurisy, with copious liquid effusion. The explanation which I am about to give you applies to the formation of false membranes upon the pericardium and peritoneum, as well as upon the pleura. In the first stage of inflammation, the serous membrane is roughened by a thin layer of exuded lymph; in the next stage there is an effusion, more or less copious, of an albumino-fibrinous liquid. Then the subsequent thickening of the false membrane on the pleura occurs by successive deposits of fibrine from the liquid effusion, upon the previously exuded and deposited lymph. The process is exactly analogous to that which occurs on the surface of an inflamed cardiac valve. The endocardium being roughened by a scanty exudation of lymph, there occurs a subsequent deposit of fibrine from the blood upon the damaged valve, and thus the so-called warty vegetations are formed. The fibrine coagulates and is deposited upon any part of the endocardium, which has been roughened by inflammation, as it coagulates and concretes upon a wire or other foreign body introduced within the vessels of a living animal.[1]

[1] See chapter xxxiii., *On the Morbid Anatomy and Pathology of Acute Endocarditis.*

The liquid effusion of pleurisy may be looked upon as blood minus its red corpuscles. We have seen that the liquid drawn from our patient's chest quickly clotted into a gelatinous mass; and this coagulation, which occurs rapidly after the removal of the fluid from the chest, often takes place gradually within the chest, upon the roughened surface of the inflamed pleura. The explanation which I have given you of the process by which fibrine is deposited from the liquid effusion of pleurisy is in accordance with the fact that, while in cases of dry pleurisy the effused lymph is comparatively scanty, very thick and firm false membranes occur only in association with a copious liquid effusion.

And now for the practical application of this pathological doctrine. It is obvious that, the longer the liquid effusion of pleurisy remains within the chest, the greater is the probability of a copious deposit of fibrine upon the roughened surface of the pleura, and the thicker this fibrinous deposit, the less is the probability that the liquid will be absorbed. These considerations, then, suggest the expediency of early tapping in all cases of pleurisy with a copious liquid effusion; and I have no doubt that the timely performance of thoracentesis, in the case of our patient, greatly promoted his recovery. If the liquid had not been pumped out of the chest, it is probable that the pleura would have become coated over by successive layers of fibrine deposited upon its surface; the result would have been a tedious convalescence, and ultimately an incomplete recovery, with more or less contraction of the side, the lung being bound down, and its expansion prevented by a thick and firm false membrane over its surface.

During the last few months,[1] the French medical societies and journals have been much occupied with the discussion of the 'albuminous expectoration' which sometimes occurs after the operation of thoracentesis for pleurisy. In two leading articles in the *British Medical Journal* of October 4 and 11, 1873, you will find a clear and able summary of the discussions, and an account of the various theories to which this interesting subject has given rise. The expectoration of a frothy albuminous, sometimes blood-tinged fluid, may begin within a period

[1] This was said in October, 1873.

varying from ten minutes to an hour after the withdrawal of the pleuritic fluid; and its duration varies from a few hours to a whole day. Referring you to the articles in question for an account of the various theories relating to the subject, I may state now that the explanation which appears to have met with the most general acceptance amongst the French pathologists is, that the re-admission of air into the previously compressed lung, acts as an irritant to the air passages, and so excites active pulmonary congestion, which results in a serous transudation through the capillaries into the air-cells and bronchi. But I doubt whether the re-admission of air could exert any such irritant influence upon the pulmonary tissues as the theory in question assumes.

My friend and colleague Dr. Duffin has published the following ingenious explanation of the phenomena in question.[1] He says that 'we have, in a limb compressed by Esmarch's bandage and rope, a condition closely analogous to that of a lung compressed by an extensive effusion. When the apparatus is removed, it is often observed that a burst of redness diffuses itself over the part. Sometimes, but by no means invariably, a troublesome hæmorrhage will then ensue from the cut surface. The reason probably is, that the pressure has not only emptied the vessels, but has also compressed the nerves of the limb, and among others the vaso-motor nerves. When the blood returns to the extremity, it finds the vessels not only empty, but lax, owing to vaso-motor paralysis. A similar condition exists in a lung which has just been relieved from the pressure of a considerable effusion. Its vaso-motor nerves have been compressed. As the lung again expands, the tension thrown upon its small vessels is not only sudden, but very great. When the nerves and vessels have had a few hours to recover, their controlling action again asserts its influence and the arterial tension becomes normal. The rapid and violent onset and the short duration of the albuminous expectoration, are thus rationally accounted for. The amount of expectoration has generally been proportionate to the degree of previous compression and the extent and rapidity of the subsequent expansion of the lung.'

[1] *British Medical Journal*, March 21, 1874, p. 372.

Dr. Duffin's theory appears to me to give a satisfactory explanation of the facts; and a consideration of the injurious results of long-continued pressure upon the vessels and nerves of the lung affords an additional argument for early thoracentesis in cases of copious pleuritic effusion.

CHAPTER XXVIII.

THE TREATMENT OF CATARRH AND BRONCHITIS.[1]

Treatment of Catarrh—Hot Bath of Water or Air—Wet Pack—Opium—Morphine with Antimony—Ammonia—Camphor—Treatment of Bronchitis—Diaphoretics—Inhalation of Steam—Bronchitis Kettle—Fomentations and Poultices to Chest—Leeches—Cupping—Venesection—Alcoholic Stimulants—Tonics—Expectorants—Emetics—Caution as to Opiates—Bronchitis with Bright's Disease—Chronic Bronchitis—Inhalation—Change of Air and Climate.

An ordinary catarrh, although not a dangerous or a very serious disease, is yet, with many persons, an oft-recurring malady, which occasions a great amount of discomfort and annoyance both to the patient and to his associates; and, as treatment has considerable influence upon the progress of the disorder, it is worth while to give the subject careful consideration.

The exciting cause of a catarrh, in a great majority of cases, is a chill, or some unknown atmospheric influence, which tends to suppress the action of the skin; and the most successful plan of treatment consists in the employment of means for restoring the free action of the skin. The popular domestic treatment consists in the use of a hot foot-bath at bed-time, a fire in the bed-room, a warm bed, and some hot drink taken after getting into bed, the diaphoretic action being assisted by an extra amount of bed-clothes. Complete immersion in a warm bath is more efficacious than a foot-bath; but the free action of the skin is much more certainly obtained by the influence of hot-air—most surely and profusely, perhaps, by the Turkish bath. The Turkish bath, however, is not always to be had, and, even when available, its use in the

[1] *British Medical Journal*, Oct. 23, 1869.

treatment of catarrh is attended with some inconvenience. In particular, there is the risk of a too speedy check to the perspiration after the patient leaves the bath. On the whole, the plan which combines in the greatest degree efficiency with universal applicability consists in the use of a simple hot-air bath, which the patient can have in his own bed-room. All that is required is a spirit-lamp with a sufficiently large wick. Such lamps are made of tin, and sold by most surgical instrument makers.

The lamp should hold sufficient spirit to burn for half an hour. The patient sits undressed in a chair with the lamp between his feet, rather than under the chair. An attendant then takes two or three blankets and folds them round the patient from his neck to the floor, so as to enclose him and the lamp, the hot air from which passes freely round his body. A macintosh cloak made for the purpose is often used instead of the blankets. In from a quarter to half an hour there is usually a free perspiration, which may be kept up for a time by getting into bed between hot blankets. I have myself gone into a hot-air bath suffering from headache, pain in the limbs, and other indications of a severe incipient catarrh, and in the course of half an hour I have been entirely and permanently freed from these symptoms by the action of the bath.

Another simple and efficient mode of exciting the action of the skin, consists in wrapping the undressed patient in a sheet wrung out of warm water, then, over this, folding two or three blankets. The patient may remain thus 'packed' for an hour or two, until free perspiration has been excited.

I may mention, in passing, that the hot-air bath and the wet packing are very useful in the treatment of many forms of disease. I constantly employ both in the treatment of renal disease; and not long since I believe that by the wet packing I saved the life of a lady, in whom very alarming symptoms were associated with the imperfect outcoming of the rash of scarlatina.

Now, to return to the treatment of bronchial catarrh, let me impress upon you that the sweating plan of treatment, to be successful in cutting short the disease, must be adopted

early—I mean within a few hours from the commencement of the symptoms.

Another mode of treating catarrh, which is very successful with patients who are tolerant of opium, consists in giving a dose of opium, or morphine, at bedtime. Within half an hour after the opiate is taken, it frequently happens that the unpleasant coryza, and all other symptoms of catarrh, have passed away. If the patient can avoid exposure on the following day, the cure may be complete, and there is no need to repeat the dose.

It is probable that the good effect of the opiate is partly due to its diaphoretic action, which may be increased by combining it with ipecacuanha; but, besides its action upon the skin, there must be some direct influence on the nerves and vessels of the inflamed mucous membrane to explain the speedy relief from discomfort which follows the opiate dose. The opiate treatment of catarrh is not so generally applicable as the sweating plan, for the reason that many persons are intolerant of opium, or they cannot take it without suffering from headache, nausea, and other distressing symptoms, which render it an undesirable remedy for them. In any case the opiate treatment, like the diaphoretic method, is more successful in proportion as it is resorted to early in the attack.

[After the publication of this lecture, Dr. Styrap, in a paper which appeared in the *British Medical Journal*, December 9, 1876, strongly recommended repeated small doses of morphine combined with antimony for the treatment of incipient nasal and bronchial catarrh. He gives the two following formulæ, three or four doses of either of which often suffice to arrest an incipient catarrh:

1. ℞ Liq. morphinæ (P.B.), ♏xl.; vini antim., ♏xxx.; potassæ cit., ℈iv.; syr. aurant., ʒij.; aquæ ad ℥iv. M. A fourth part every three or four hours.

2. ℞ liq. morphinæ, ♏xl.; vini antim., ♏xxx.; liq. am. cit., ℥j.; potass. cit., ℈iv.; sp. chloroformi, ʒij.; aquæ ad ℥iv. M. A third part every three or four hours.

I can bear testimony to the usefulness of these formulæ.]

In some persons, repeated doses of ammonia have the effect of lessening the coryza and other distressing catarrhal symp-

toms. Five grains of carbonate of ammonia, or a drachm of the aromatic spirit, may be taken in water every three hours. A single dose of ammonia at bedtime is an efficient and useful diaphoretic, its action being aided by external warmth. Some catarrhal patients experience great relief from an occasional dose of spirit of camphor. The usual dose of the Pharmacopœia preparation is from ten to thirty drops in a wineglass of water.[1] In ordinary catarrh, as a rule, no change of diet is required, but a catarrh which has gone on unchecked for a few days is sometimes much mitigated by a generous diet and an extra glass of wine.

Those who are especially liable to catarrh should be careful to keep their feet warm and dry; and they should be warmly clothed, wearing woollen next the skin. But they should avoid excessive wrapping up; since this, with even gentle exercise, tends to overheat the body, and so to increase the risk of a subsequent chill. The practice of wearing a hare-skin or washleather over the chest is to be condemned, as at once uncleanly and unwholesome.

It may be well to remind catarrhal subjects that the nose is a natural respirator, so that, in passing from a hot room into the open air, if the mouth be kept closed, the air, in its passage through the nostrils, has its temperature raised before it enters the chest.

There is reason to believe that the daily use of a cold sponge-bath, or a shower-bath, by those whose circulation is sufficiently vigorous to effect a speedy reaction and glow on the surface, has a wholesome hardening influence upon those who adopt it, and that it renders them less liable to attacks of catarrh.

Treatment of Acute Bronchitis.—Acute bronchitis is an exaggerated catarrh; the two diseases are essentially the same, and they require the same principle of treatment, only modified according to the character of the symptoms.

In the early stage of acute bronchitis, when the mucous membrane is dry and swollen, the hot-air bath or the wet packing may be employed once or oftener with advantage.

[1] In Chapter XVI., *On Cases of Poisoning by the Homœopathic Concentrated Solution of Camphor*, I have pointed out the danger of employing that poisonous preparation for the treatment of catarrh.

Another very useful remedy in this stage is tartar emetic, in doses of one-sixth of a grain, combined with liquor ammoniæ acetatis, and sometimes with a small dose of morphine. This mixture exerts a diaphoretic action both upon the skin and the mucous membrane of the air-passages; thus it brings on the stage of secretion, and with this a mitigation of the vascular engorgement. The patient should remain in bed, and the temperature of the room should be maintained at from 60° to 65° Fahr., the air being kept moist by steam from the spout of a kettle, or from a special boiler with a long spout—a so-called 'bronchitis kettle.' The inhalation of steam, repeated several times in the course of the day, is often very soothing and beneficial. Hot fomentations may be applied to the front and back of the chest by means of spongio-piline, or flannels covered with macintosh. A mild mustard poultice to the front of the chest is a good remedy for a sense of tightness and dyspnœa; but I advise you not to excite painful inflammation of the skin by mustard or turpentine, or by any other means.

When dyspnœa, with a feeling of tightness and oppression at the chest, is urgent and distressing, the application of a few leeches to the chest, or a moderate abstraction of blood by cupping, often affords prompt, decisive, and permanent relief. Venesection is very rarely required, though in the case of a plethoric subject suddenly seized with general capillary bronchitis, and threatened with death from apnœa, venesection may prove a life-saving remedy. Milk and beef-tea form the most suitable diet during this stage of the disease. Stimulants and opiates are to be avoided, as a rule, on account of their tendency to increase the congestion and dryness of the inflamed mucous membrane. In the second stage, when a free secretion has been established, antimony and acetate of ammonia are to be discontinued. At this period, a combination of carbonate of ammonia with spirit of chloroform is useful as a stimulating expectorant and antispasmodic. Brandy or wine in moderate quantities may now be required to sustain the strength. When, in the advanced stages, there is a profuse purulent secretion, with copious perspirations, the ammonia mixture may be replaced by one containing, in each dose, a grain of sulphate of quinine, two grains of sulphate of zinc, and twenty minims

of aromatic sulphuric acid. This combination often checks very rapidly the excessive secretion from the bronchial mucous membrane. The stimulating expectorants are sometimes useful at this stage of the disease—I mean senega, squills, ammoniacum, and the compound tincture of benzoin. If, as sometimes happens, the stimulating expectorants suddenly check secretion, tighten the breath, and increase dyspnœa, their employment must at once be discontinued. When the secretions accumulate and threaten suffocation, the patient being blue, and cold, and drowsy, and the cough nearly or quite ceasing, an emetic of sulphate of zinc, or if that fail twenty or thirty grains of carbonate of ammonia, is often wonderfully efficacious in clearing the air-passages.

Here I must give you an especial warning with regard to opium. A patient who has been sitting up in bed, labouring for breath day and night, naturally craves for sleep, and begs for an opiate. Now a small dose of opium given in such a case has caused fatal narcotism in numberless instances. The opiate stops the cough, and, of course, the expectoration; the patient sleeps more and more heavily; meanwhile the secretion accumulates, and causes fatal apnœa. Never, therefore, give an opiate to a bronchitic patient who has the slightest blueness of the lips. When the expectoration is quite free, and the lips are florid, you may sometimes venture to give a small opiate with antimony or ipecacuanha, or you may give a drachm of the compound tincture of camphor, or twenty minims of chlorodyne. The good effects of a few hours' sleep thus procured are sometimes very manifest.

When bronchitis is associated with blood-contamination consequent on Bright's disease, diaphoretics, purgatives, dry cupping, and poultices over the loins, are amongst the most useful remedies.

The treatment of *chronic bronchitis* is essentially the same as that of the acute form of the disease. They merge into each other by imperceptible degrees. An acute attack may subside into a chronic condition, and exposure to cold will quickly convert chronic into acute bronchitis.

Amongst other remedies in the chronic stage, the inhalation of the vapour of creasote, oil of turpentine, or terebine, is often

beneficial. These vapours facilitate expectoration at the same time that they tend to check the profuse purulent secretion. The abundant secretion may sometimes be checked by inhaling, in the form of spray, a solution of tannic acid.

In treating the diseases of the air-passages by the inhalation of vapours, bear in mind that these vapours rapidly pass beyond the lungs; they are quickly absorbed and enter the circulation, causing, in some instances, headache and other discomforts. The necessary contamination of the blood by the inhalation of vapours renders this mode of medication less generally useful than it otherwise might be, in the treatment of bronchial inflammation and catarrh.

Change of air, and, in particular, a residence in a mild, dry, and equable climate, are amongst the most important remedial and preventive measures in cases of chronic bronchitis.

CHAPTER XXIX.

A LECTURE ON VESICULAR EMPHYSEMA OF THE LUNGS.[1]

Definition—Anatomical Characters—Physical Signs—Causes, Pathology, and Mechanism—Relation of Emphysema to Bronchitis—General Symptoms and Results—Prognosis—Treatment.

Laennec applied the term Emphysema to two different conditions of lung. *Vesicular emphysema* consists in dilatation of the air-cells. The walls of the cells often become ruptured, so that several contiguous cells unite to form a single cavity. This is a very common and very interesting form of disease. *Interlobular emphysema* is characterised by an infiltration of air into the connective tissue between the lobules; sometimes, too, beneath the pleura, and into the areolar tissue of the mediastinum and of the body generally. This is a comparatively infrequent accident.

I shall speak now only of *vesicular emphysema.*

Emphysema, as you know, is Greek for *inflation,* and the term is correctly employed by surgeons to designate accidental inflation of the subcutaneous areolar tissue; such as occasionally happens after a wound of the lung by a fractured rib, or during the operation of tracheotomy.

Inflation of the air-cells of the lungs is a normal condition; but, in the so-called vesicular emphysema, the cells are *dilated*; and Dr. Stokes uses the term 'dilatation of the air-cells' to designate Laennec's vesicular emphysema. The disease consists essentially in dilatation of the air-cells with rupture of their walls.

Anatomical Characters.—The cells are much increased in size, and very irregularly so. The greater number, perhaps,

[1] *British Medical Journal,* June 27, 1868.

equal the size of a millet-seed; while some attain the magnitude of a hemp-seed, a cherry-stone, a French bean, or even a still greater size. The larger cells are probably formed by the union of several, through rupture of their intermediate partitions. The larger cavities often form projections on the surface of the lung when the chest is opened. The air does not readily escape from the dilated cells, partly perhaps on account of some obstruction in the bronchi which lead to them, partly from loss of elasticity of the walls of the dilated cells. When the lung is inflated by blowing into the trachea, the tissue round the projecting vesicles rises to their level, and the surface of the lung becomes even.

A good way of showing the dilatation of the vesicles is, as Laennec directs, to make thin sections of a lung which has been inflated and then dried. It may thus be seen that not only are the cells dilated, but that many of their septa are more or less completely destroyed.

Emphysema may affect either a part or the whole of one or both lungs. It is either partial or general. The emphysematous dilatation is usually most advanced and conspicuous in those portions of the lungs which receive least uniform support from the walls of the chest and the surrounding viscera; e.g. the apices of the lungs where they project above the clavicles, and where they sometimes form a conspicuous soft swelling at the root of the neck; the anterior margins which lie behind the somewhat yielding cartilages of the ribs, and which are to some extent deprived of direct pressure by the intervention of the heart and great vessels.[1]

Again, the base of the left lung is less firmly supported by the stomach than the corresponding part of the right lung by the liver; and, accordingly, Louis found that the lower lobe of the left lung was twice as often the seat of emphysema as the lower lobe of the right; while the apex of the right lung was more frequently emphysematous than the apex of the left.[2]

When the whole or a great part of the lung is affected, the lung does not collapse as usual when the chest is opened. The organ appears to be enlarged, and almost to project

[1] Sir William Jenner, *Med.-Chir. Trans.*, vol. xl. p. 31.

[2] Jenner, *loc. cit.*, p. 32, *note*.

beyond the margins of the divided ribs. When compressed, the lung does not crepitate like healthy lung, but, as Laennec describes it, 'the sensation conveyed by pressing the parts is very like that produced by handling a pillow of down.'[1] Emphysematous lungs float with great buoyancy on water. They are usually more or less of a *pale grey* colour, and they have a peculiar *dry* feel. The pale colour and the dryness are results of the small quantity of blood which they contain. The capillaries, many of them, are obliterated. The posterior parts of the lungs are sometimes engorged.

In fatal cases of emphysema, the anatomical signs of bronchitis are commonly found: congestion and thickening of the mucous membrane of the bronchi; obstructed bronchi, with here and there collapsed lobules; dilated bronchi; and, more rarely, bronchial abscess.

The Microscopic Characters of Emphysematous Lungs.— Mr. Rainey has published[2] an interesting account, with illustrations, of the microscopic appearances in an emphysematous lung. He describes the pulmonary membrane which forms the walls of the vesicles, as being perforated or cribriform; the perforations being well defined, of an oval or circular form, of various sizes, and more or less numerous, according to the stage of the disease. The membrane in other parts is studded over with oil-globules, either single or clustered; and Mr. Rainey believes that this oily degeneration precedes the perforation of the membrane. In an injected specimen, the capillaries are seen to be much attenuated, while the meshes of the capillary network are expanded and dilated. These changes in the capillaries are proportioned to the dilatation of the air-cells; and they appear to indicate that there has been a considerable stretching and expansion of the membrane, together with its capillary network.

The oily degeneration of the walls of the air-cells is not a constant phenomenon. I have repeatedly looked for it without finding it; and, not unfrequently, dilatation and rupture of the air cells, with expansion and partial obliteration of the capillary network, are the only definite structural changes

[1] Dr. Forbes's translation, p. 144.

[2] *Med.-Chir. Trans.*, vol. xxxi. p. 297.

which are visible in an emphysematous lung. I agree with Dr. Waters[1] that 'it is quite possible that the elasticity of the yellow fibres may become impaired, or even destroyed, without any structural alteration resulting, such as could be appreciated even with the highest powers of the microscope; and it is also equally possible that changes may occur in the blood-vessels, giving rise to malnutrition of the pulmonary tissue, yet that we may be unable to distinguish them.'

Amongst the most constant of the *post-mortem* appearances are dilatation of the right cavities of the heart, with hypertrophy of the walls. Less frequently there is some hypertrophy and dilatation on the *left* side.

Physical Signs.—Such being the anatomical characters of emphysema of the lungs, we may next consider the *physical signs* of the disease.

In cases of advanced and general emphysema the whole chest appears enlarged. There is an unnatural bulging and prominence both in front and at the back. The chest becomes 'barrel-shaped.' There is an unusual fulness above and below the clavicles. A few months since, I was consulted about a lady who was supposed to have a tumour at the root of the neck on one side. I found that the swelling was produced by the bulging of an emphysematous lung upwards into the neck above the clavicle. In some cases, the chest is irregularly and unsymmetrically prominent on one or both sides. The *semicircular measurement* of one or of both sides of the chest is frequently increased. The disease, however, may be highly marked without such increase. This is especially common in persons beyond middle age, whose costal cartilages are ossified. The *movements* of the ribs are very limited. Their position is that which they ordinarily occupy at the end of a deep inspiration; and now, during even forced breathing, the whole chest rises in a mass, but there is very little movement of individual ribs. The breathing is chiefly diaphragmatic and abdominal. The lower part of the chest sometimes sinks in during inspiration. The *duration* of the *expiratory movement* is seen to exceed that of the *inspiratory*. This is a result of the diminished elasticity of the lung.

[1] *Diseases of the Chest.*

Percussion.—There is increase of resonance, an exaggerated pulmonary resonance, sometimes of a tympanitic character, but never so purely tympanitic as in cases of pneumothorax, or distension of the stomach by air. The excessive resonance extends over the cardiac region; the heart being overlapped and pushed downwards by the enlarged lungs. The resonance extends, on both sides, lower than its normal limit, owing to the pushing down of the diaphragm. The liver is thus made to occupy a lower position than normal, and may sometimes appear to be enlarged when it is only misplaced. Exaggerated resonance is not constant; sometimes the arched form of the ribs renders them less yielding, and therefore less resonant on percussion, more especially in old persons, when the cartilages are ossified.

Auscultation.—The chief auscultatory sign of emphysema is feebleness of the respiratory murmur. This is a result of limited expansion of the chest, in consequence of which the air enters the lungs with less force, and therefore with less noise. The respiration over certain parts of the chest is sometimes harsh and exaggerated; for the same reason that it is puerile over the sound lung, when one is rendered more or less impervious to air. The *rhythm* of the sounds is altered. The sound of inspiration is comparatively short; that of expiration is prolonged. Very commonly, when the expiratory sound is much prolonged, it is a sibilus that is heard rather than the natural sound of expiration. All the auscultatory signs of *bronchitis* are frequently heard, and also the fine crepitation of œdema of the lung. Laennec described a sound, which he called *a dry crepitant rhonchus with large bubbles*, which, he said, resembles the sound produced by blowing into a dried bladder. This he considered pathognomonic of vesicular and interlobular emphysema, but more distinct and constant in the latter. Some writers assert that they have heard such a sound on rare occasions. Others—and amongst them Sir Thomas Watson and Dr. Stokes—state that they cannot distinguish between this sound, if it really exists, and the crepitations which accompany bronchitis. It is probable, as Sir Thomas Watson suggests, that the sound in question 'is really large crepitation in the neighbourhood of the dilated cells.'

Laennec himself says : 'We have a sound like this in the common subcutaneous emphysema, on pressing interruptedly with the ear on the stethoscope, or with the fingers, on the affected part.'[1] This I have several times heard; it is like the fine crepitation of pneumonia, and is evidently produced by air being pressed through the moist meshes of areolar tissue; it is a *moist*, not a *dry* sound.

Laennec also describes a rubbing (*frottement*) of ascent and descent, which he believed to be a result of the friction of sub-pleural vesicles against the costal pleura, in cases of interlobular emphysema. A friction-sound probably never occurs except as a result of pleurisy; in which disease Laennec overlooked it.

To recapitulate then. The chief physical signs of vesicular emphysema are: local or general bulging of chest; diminished expansion; respiration chiefly abdominal. Increased resonance on percussion, not constant. Resonance extending over the whole of the cardiac region. Feeble respiratory murmur, not constant; prolonged expiration. Often the signs of catarrh and bronchitis. The *vital capacity* of the chest is small. An emphysematous patient can blow but little air into the spirometer. The heart beats in the epigastrium.

The Causes, the Pathology, and the Mechanism of Vesicular Emphysema.—Most writers on emphysema have assumed that this condition of lung is a consequence of bronchitis; but they have differed in their explanation of its mechanism.

Laennec's theory was that, as a result of catarrh, there is plugging of some bronchi by secretion, and a consequent accumulation of imprisoned air, which, being expanded by the high temperature of the surrounding lung, causes dilatation of air-cells. He erroneously supposed that inspiration is more powerful than expiration, and so conveys air beyond the obstruction, which expiration fails to expel.

Dr. Williams maintains the catarrhal basis of Laennec, but supposes that the air-cells communicating with the plugged bronchi escape distension, while the surrounding air-cells dilate, in consequence of the extra work and pressure thrown upon them.

[1] Page 55.

Dr. Gairdner maintains that emphysema is a result of the pulmonary collapse, which he has shown to be a common consequence of bronchitis and obstruction of the bronchi. Dr. Gairdner's theory of emphysema is that it 'is an increase in volume of those portions of the lung to which the air has access, to supply the place of diminished volume in those parts from which it is excluded.' [1] He denies that the chest is enlarged in emphysema, and says: 'There is great reason to believe that it is usually smaller than natural; the arching of the front and increase of the antero-posterior diameter being more than counteracted by a diminution in all the lateral diameters, particularly at the base of the chest.' [2] This denial of the enlargement of the chest is contrary to my own observations, and to those of most physicians who have directed attention to the subject. Dr. Gairdner's theory appears sufficient to explain the emphysema which occurs in the neighbourhood of lung, collapsed after obstruction of the bronchi, or condensed and atrophied by a tubercular or an inflammatory deposit; but it affords no satisfactory explanation of cases of emphysema affecting the whole lung, and causing general enlargement of the chest. Moreover, in many cases of this kind, the history clearly shows that the emphysema has preceded the symptoms of bronchitis. Since my attention has been particularly directed to the relation between emphysema and bronchitis, a period now of many years, I have met with a considerable number of persons, both in hospital and in private practice, who, presenting all the physical and general signs of emphysema in a very marked degree, have had no bronchitic symptoms up to the time of their coming under my notice, or they have had these symptoms so recently as to preclude the theory that the bronchitis had caused the emphysema.

The fact that emphysema sometimes precedes the symptoms of catarrh has been noticed by several authors. Thus, Laennec states that 'he has some reasons for believing that, in certain cases, the dilatation of the cells is the *primary* affection, and the catarrh *consecutive*.' [3] Louis affirms that,

[1] *Brit. and For. Med.-Chir. Rev.*, April 1853, p. 472. [2] P. 470.

[3] Forbes's translation, p. 148.

in many instances, catarrh has not shown itself until one or more years after the commencement of oppressed breathing and other symptoms of emphysema.[1] And, more recently, Dr. Waters mentions, amongst other reasons for believing that emphysema depends on a primary degeneration of the lung-tissue, 'the high degree of development which the disease often reaches without any previous history of violent or long-standing cough.'[2]

I shall presently explain in what way emphysema acts as a powerful predisposing cause of bronchitis; but I must first indicate the chief circumstances under which the disease occurs. These are very various; but there is reason to believe that, in all cases of general emphysema, there has been an impaired nutrition of the lung-tissue, a loss of its normal elasticity and cohesive power, and a consequent expansion and rupture of the air-cells.

1. First, there is a class of cases in which the tendency to this disease appears to be inherited. Several members of the same family are found to suffer from the malady, and successive generations of the same family. When the disease is hereditary, it sometimes manifests itself at an early age—even as early as between the ages of 20 and 30—and the chest soon becomes much enlarged.

2. There are cases of what may be called *senile emphysema*. Advancing age brings on the disease in those who have no hereditary tendency to it. As the arterial and other tissues degenerate and decay with advancing years, so does the elastic tissue of the lung. In these cases of senile emphysema, there is usually no visible enlargement of the chest; for, the cartilages of the ribs being ossified, the chest-walls are incapable of being much expanded. Emphysema, without enlargement of the lung and of the chest, is sometimes called *atrophic emphysema*; while the enlarged emphysematous lung is designated *hypertrophic*. But there is really no hypertrophy of the lung. We do not call a simply dilated bladder hypertrophied; neither do we speak of the uriniferous tubes of the

[1] 'Recherches sur l'Emphysême des Poumons,' *Mém. de la Soc. Méd. d'Observation*, Paris, 1837, tome i.

[2] *Researches on Emphysema of the Lungs*, p. 34.

kidney as being hypertrophied, when they are dilated into cysts.

3. In another class of cases, the excessive use, or rather abuse, of alcoholic liquors has probably been the exciting cause of the disease. This, at any rate, seems in some instances to afford a sufficient explanation of that impaired nutrition of the pulmonary tissues which is the immediate precursor of the dilatation and rupture of the air-cells. In some instances, we find that the emphysematous patient has had one or more attacks of gout; and it seems probable that the gouty dyscrasia may contribute to the malnutrition of the pulmonary tissue.[1]

4. In a fourth class of cases, the emphysema has supervened upon repeated attacks of bronchitis. In such cases, it is probable that the nutrition and the elasticity of the pulmonary tissues have been impaired by the recurring bronchitic attacks, while the air-cells have been dilated and ruptured by the forcible efforts of coughing.

5. In another class of cases, valvular disease on the left side of the heart appears to have been the cause of the emphysema. The association of emphysema with disease of the mitral or aortic valves is so frequent that it cannot be a mere casual coincidence. For many years past I have observed this association of emphysema of the lungs with valvular disease on the left side of the heart, and I have very frequently demonstrated it in the wards of the hospital. In these cases, we often find a history of rheumatic fever dating some years back; then, after a time, increasing shortness of breath and palpitation; and, on examination, the physical signs of emphysema and of old valvular disease are observed. In these cases, it is probable that the impaired nutrition and the loss of elasticity of the lung-tissue result from the impeded circulation consequent upon the defective valve.

6. There is a class of cases to which I desire to direct your especial attention—cases in which emphysema is associated with an excessive accumulation of fat in the subcutaneous tissue, in the abdomen, and about the heart. Many of these cases would come under one or other of the classes to which I

[1] Dr. Greenhow *On Chronic Bronchitis.*

have already referred. In not a few cases, the emphysema and the tendency to accumulate fat are hereditary; and in some, the same tendency is a result of intemperance in eating and drinking. But these cases require a separate and particular mention, on account of their peculiar and distinctive features, and on account of the important influence of diet in the prevention and mitigation of their distressing symptoms. What, then, is the connection between emphysema of the lungs and an excess of fat throughout the body? We find that, in a large proportion of cases, this excess of fat is associated with what Dr. Quain[1] has very appropriately designated 'fatty growth' about the heart; and this fatty growth is always most abundant on the right side of the heart. The effect of this growth is to weaken the muscular walls of the right cavities. The result is that the circulation through both the pulmonary and the bronchial vessels is impeded. There will be a diminished flow of blood through the pulmonary artery, in proportion to the impaired contractile power of the right ventricle; and the backward pressure of blood from the distended right cavities will impede the return of blood from the bronchial, as from the other systemic veins. The impeded circulation leads to impaired nutrition and lessened elasticity of the pulmonary tissue, and thus the lung becomes emphysematous. An excess of fat, therefore, in particular a fatty growth on the right side of the heart, may be a primary cause of emphysema. Then emphysema, having reached a certain stage, undoubtedly increases the tendency to obesity, and that, too, in a very intelligible way—by impeding respiration, and so lessening the combustion of hydro-carbon. Exercise lessens the accumulation of fat in the system by increasing respiration and combustion. An emphysematous patient cannot take active exercise, because he is short-winded; and, whether he takes exercise or not, the pulmonary circulation is so much impeded, and the vital capacity of the chest so far diminished, that the respiratory changes are always in arrear of the needs of the system. Unburned hydro-carbon, therefore, accumulates in the form of fat; and many of these unhappy patients carry about with them a great weight of

[1] *Med.-Chir. Trans.*, vol. xxxiii.

material which is simply an encumbrance to them. This great accumulation of fat, again, mechanically interferes with the movements of the chest and of the diaphragm, so that the respiratory changes and the pulmonary circulation are still further impeded, while the heart is embarrassed by the growth of fat upon its surface; and thus, by these mutual reactions, the evil grows steadily worse.

7. There is yet another class of cases, quite distinct from any of those to which I have before referred; I mean cases in which emphysema has supervened upon repeated attacks of spasmodic asthma. However purely spasmodic asthma may be at its commencement, there is a continual tendency to the development of structural changes in the lungs; and by far the most common of these changes is emphysema, with its usual catarrhal complications, and with hypertrophy and dilatation of the right cavities of the heart. The following I believe to be the explanation of the emphysema which results from spasmodic asthma. During the paroxysms of asthma there is a partial apnœa,[1] caused by spasmodic narrowing of the bronchial tubes. This partial apnœa involves, as its necessary correlative, a partial asphyxia—that is, pulselessness. The movement of blood through the lungs is checked by the stopcock action of the minute pulmonary arteries, in proportion to the degree in which the respiratory changes are limited by the bronchial spasm. The arrest of blood in the lungs causes engorgement of the right cavities of the heart, and of the whole systemic venous system. This retrograde engorgement, extending to the bronchial veins and capillaries, has, for its invariable sequence, mucous exudation from the bronchial mucous membrane. The earlier attacks of asthma leave no permanent structural changes in the lungs. With the cessation of the bronchial spasm, the freedom of the circulation is restored, and the lungs are left structurally sound; but, when the asthmatic paroxysms have been frequent, severe, and prolonged, the lungs become emphysematous. The frequently recurring spasmodic apnœa, with the consequent blood-stasis

[1] In the Lecture on the *Physiology of the Circulation* I have stated the reason for the use of the terms *apnœa* and *asphyxia* in the sense in which they are here employed (p. 22, *note*).

and engorgement of the bronchial veins and capillaries, causes an impaired nutrition of the pulmonary tissues, a loss of elasticity, and then dilatation of the air-cells. During the violent respiratory efforts, too, the air-cells will be subjected to an excessive pressure.

It will be seen, then, that emphysema consequent on disease of the mitral or aortic valve, emphysema the result of fatty growth on the right side of the heart, and emphysema the result of spasmodic asthma, have this in common, that the impaired nutrition of the lung-tissue, and the consequent dilatation of the air-cells, are the result of impeded pulmonary and bronchial circulation; so that, while the cause of the impediment is different in each of these three classes of cases, the ultimate result is the same.

Some difference of opinion has existed upon the question whether the dilatation of the air-cells occurs during the act of inspiration, or during expiration. Most writers have insisted chiefly upon the inspiratory distension of the air-cells as the cause of their dilatation; but Sir William Jenner [1] has directed attention to the influence of violent expiratory efforts—such as occur in coughing, &c.—in distending and dilating the cells, particularly in those portions of the lung which do not receive uniform support from the walls of the chest—namely, the apices and the anterior margins of both lungs, and the base of the left lung. The air-cells in these portions of lung are not unfrequently dilated and torn suddenly during the violent respiratory movements which occur when the entrance and egress of air are impeded, as in cases of croup, diphtheria, and hooping-cough.

To sum up, then, the immediate physical causes of vesicular emphysema:

1. It may be a result of increased strain and pressure upon air-cells which are previously healthy, as in the cases just now referred to, in which acute emphysema occurs during the progress of diseases which involve a great impediment to the entrance and exit of air. Violent and prolonged gymnastic exertion is an occasional cause.

2. It is a result of over-distension acting upon cells in the

[1] *Med.-Chir. Trans.*, vol. xl.

immediate neighbourhood of portions of lung collapsed, during the course of bronchitis, or condensed and contracted by a tuberculous or an inflammatory deposit.

3. Lastly, when emphysema comes on slowly, and affects the greater part of both lungs, it is a result of previously impaired nutrition and diminished elasticity of the pulmonary tissue.

The Relation of Emphysema to Bronchitis.—A local and a very partial emphysema may result from lobular collapse of the lung, which is one of the accidents of bronchitis; a more general emphysema may be induced by repeated attacks of bronchitis, as I have already explained; but whence comes it that general emphysema, when not preceded and caused by bronchitis, is associated with a continual liability to catarrh and bronchitis? I look upon it as certain, that this tendency to bronchitis is a *consequence* of the emphysema. I had arrived at this conclusion from a careful observation of the order in which the various phenomena of the disease occur, before I saw any explanation of their relation to each other; and my opinion is now confirmed by what I consider to be the rational interpretation of well-ascertained facts. We have seen that one of the chief anatomical features of emphysema is the obliteration of a considerable portion of the capillary network of the lung; hence arises an impeded pulmonary circulation, and hypertrophy with dilatation of the right side of the heart. The systemic veins are always more or less distended; and the bronchial veins, which are tributary to the systemic veins, are partakers in this distension. The bronchial veins and capillaries, therefore, being in a constant state of passive engorgement, there is a continual tendency to serous transudation through the bronchial mucous membrane, which is manifested by the wheezing sound audible on a deep inspiration; and a catarrh from exposure to cold, which, occurring to an individual with sound lungs, would be only a slight and transient ailment, is apt to become a severe and chronic bronchial inflammation. The impeded circulation through an emphysematous lung, acting backwards, through the right side of the heart and the vena cava, upon the bronchial veins, predisposes to bronchitis, and prolongs and aggravates it when

it occurs, just as varicose veins in the legs act as a predisposing cause of inflammation of the skin, and often render its cure difficult. This, then, is the explanation of the fact that general emphysema of the lungs is almost constantly associated with a tendency to winter-cough and bronchitis. This explanation of the relationship between emphysema and bronchitis receives additional confirmation from the fact before referred to and accounted for—namely, the invariable sequence of bronchial catarrh upon every paroxysm of spasmodic asthma; the catarrh being, as a rule, profuse and prolonged in proportion to the duration and the intensity of the previous bronchial spasm and the consequent blood stasis.

It can scarcely be doubted that attacks of bronchitis tend to increase the emphysema, partly by causing a yet greater impairment of the nutrition of the lung, and partly by the forcible distension and tearing of the air-cells during the paroxysms of coughing and dyspnœa.

The Symptoms of Emphysema.—Dyspnœa is the most frequent. At first it is slight, and felt only on unusual exertion, such as walking fast or running up-stairs. It gradually increases, and is subject to great variations of intensity. It is liable to occasional aggravations through bronchial spasm, flatulent distension of the stomach and bowels, and intercurrent attacks of bronchitis.

With the dyspnœa, there is often more or less of *palpitation* and a painful sense of distension at the bottom of the sternum. We have before seen that, in consequence of the impeded pulmonary circulation, the right cavities of the heart become distended and dilated, and their muscular walls hypertrophied. The more or less extensive obliteration of pulmonary capillaries in an emphysematous lung, impedes the passage of blood from the right side of the heart as effectually as a constricted pulmonary artery or a diseased valve. The circulation through the coronary veins, too, must be impeded by the distension of the right cavities; and this impeded circulation must be a source of cardiac weakness and distress.

Cough, as a symptom, ranks next in frequency and importance to dyspnœa. It is not an essential symptom of emphy-

sema alone; but it is a constant attendant on the catarrhal and bronchitic complications of emphysema.

Expectoration, more or less abundant, and of variable character, is associated with the cough during the bronchitic periods. Some patients expectorate the semi-transparent pearly material which is characteristic of Laennec's 'dry catarrh.'

Hæmoptysis is a more frequent symptom than is generally supposed. Its occurrence often excites a suspicion of phthisis, but it is notorious that patients who are in a marked degree emphysematous rarely become tuberculous. The blood expectorated is usually of dark colour, and more or less mixed with bronchial mucus. Its source is the over-gorged veins and capillaries of the bronchial mucous membrane; and the immediate exciting cause is often a fit of coughing, which throws back the blood with great force into the bronchial veins and capillaries.

The *countenance* is usually more or less puffy, congested, and dusky—a result of defective aëration of blood and venous obstruction. *Headache* is a common result of the same state of blood and circulation.

The *conjunctivæ*, too, are often congested, and the eyes have a glistening watery appearance.

Emaciation is not usually observable, except in the advanced stages of the disease, or as a result of severe attacks of bronchitis. In consequence of the enlargement and air-distension of the chest, the *buoyancy of the body in water* is increased; so that a patient finds, to his surprise, that he has acquired an increased facility of swimming, notwithstanding his loss of muscular power and his increasing dyspnœa on exertion.[1]

The *pulse* and breathing are usually not quickened, except on exertion, or when bronchitis is present. Very commonly, the breathing is remarkably slow and heaving, and attended with a wheezing sound.

The *voice* loses tone and strength; and the patient is incapable of a sustained note, or of long or loud speaking. Sometimes the voice becomes husky from congestion and œdema of the mucous membrane of the larynx.

[1] Dr. Walshe on *Diseases of the Lungs*.

Œdema of the ankles, either with or without congestion of the kidneys and albuminuria, may occur as a result of obstructed pulmonary circulation and consequent dilatation of the right cavities of the heart.

The *appetite* and *digestion* are commonly impaired, and the patient is often distressed by flatulent distension of the stomach after food. This impedes the descent of the diaphragm, and tends to cause or to increase dyspnœa. It is not difficult to explain the flatulent distension of the stomach. The process of digestion depends upon the solvent action of the gastric secretion upon the food. The secretion of this gastric fluid is checked by the impeded circulation of blood through the stomach consequent upon the obstruction in the lungs. In like manner, the secretion of urine is checked by the impeded circulation through the kidney. In consequence of the deficient secretion of the gastric solvent, the food ferments, decomposes, and gives off gases which distend the stomach.

Prognosis.—The prospects of an emphysematous patient are by no means favourable or pleasant. He may live for a considerable number of years ; but he must expect a continual increase of his malady, with its attendant dyspnœa and liability to bronchitis. Emphysema, although in its early stages it is attended with no immediate danger, involves much chronic suffering, and tends materially to shorten life. The most unfavourable cases are those in which the disease is hereditary, and manifests itself at an early period of life. The immediate danger to life is usually associated with the bronchitic complications, the increasing obstruction to the circulation, and the consequent renal congestion with albuminuria, a scanty secretion of urine and dropsy, passive engorgement of the bronchial veins and capillaries, and a resulting serous effusion into the bronchi, which renders the lungs œdematous.

Treatment.—In the treatment of emphysema, we have to consider, first, the emphysema itself ; and, secondly, the bronchitic and other complications. When once the lungs have become emphysematous, that condition is permanent, and we can do nothing effectual for its removal ; but a knowledge of the various causes which tend to bring on the emphysema will

enable us to do much to ward off the disease, and to prevent its increase. Now, it is manifest that amongst the determining causes of emphysema there are some over which we have more control than over others. Hereditary tendency to the disease, and the degenerative changes which occur with advancing years, are little influenced by treatment. But we can treat spasmodic asthma with more or less success. By diet and various medicines we can for a time prevent the mischievous reaction of a damaged valve upon the muscular walls of the heart, and the consequent hindrances to the pulmonary and systemic circulation; and we can warn our patients against the abuse of alcoholic stimulants, and in each of these cases our advice and our assistance may be productive of more or less benefit. But the class of cases in which, by timely warning, we may render the most valuable aid, are those in which there is an increasing tendency to the accumulation of fat beneath the skin and in various other parts. These subjects should be warned of what is in store for them if this fat-making disposition be not checked and counteracted; and then they should be taught that their chief safeguard consists in active exercise in the open air, and a carefully regulated diet. There is no rule without exceptions; but it may generally be assumed that very fat men and women are hearty and indiscriminate feeders; and however active they may have been in early life, in proportion as they increase in adipose bulk, they are the less disposed to active exercise. Now, if these subjects will escape the miseries attendant upon emphysematous lungs, they must restrict themselves to a diet somewhat after the fashion of that which is popularly known as Bantingism. The late Mr. Harvey, of Soho Square, conferred a great benefit upon Mr. Banting by cutting off his favourite bread and milk, and putting him on a judicious course of diet. Mr. Banting, zealous and grateful, appealed to all his fellow-sufferers from excess of fat to change their diet and improve their figures. Many of his disciples, acting without medical advice, did themselves serious harm by a too sudden and rigorous abstinence from fat and farinaceous food; they, in fact, starved certain tissues which require those hydro-carbon compounds for their nutrition, and thus they brought suffer-

ing upon themselves and discredit upon Bantingism. Moderation is to be observed even in abstaining. Each case may require some special directions, but, as a general rule, these fat-making subjects should be directed to take the fibre of fish, flesh, and fowl liberally; to eat sparingly of fat, butter, sugar, and farinaceous substances, including, of course, vegetables, bread, and pastry; to abstain entirely from malt liquors; and to take no more wine or spirit than is absolutely needful for health and comfort. This plan of diet, with daily walking or horse-exercise in the open air, will do much to check, although it may not entirely arrest, the tendency to accumulate an excess of fat.

I have said that these emphysematous patients are commonly dyspeptics, and suffer from flatulent distension of the stomach after food, and I have given the explanation of these symptoms. A complete cure of this form of dyspepsia can be effected only by removing all impediments from the way of the circulation—an impossible task. Yet something may be done to mitigate the symptoms. A combination of tincture of perchloride of iron and tincture of digitalis is sometimes beneficial; the iron acts as a tonic, while the digitalis as a diuretic tends to lessen venous engorgement, and so to increase the freedom of the circulation. A combination of mineral acids —hydrochloric or nitro-hydrochloric—with vegetable bitters, quinine, cascarilla, or calumba with strychnine, and tincture or syrup of ginger, is often very useful in assisting digestion and preventing flatulence. One great object of medical care is to prevent catarrh and bronchitis, and to cut short these attacks when they occur. Amongst the most important means of prevention are warm clothing, protection from cold and wet, and, during the winter, a residence in a warm, dry, and equable climate. The treatment of catarrh and bronchitis is fully discussed in the previous chapter (xxviii.).

CHAPTER XXX.

A LECTURE ON HÆMOPTYSIS: ITS CAUSES, RESULTS, AND TREATMENT.[1]

Hæmoptysis often Accidental and Harmless—Chief Causes—Tubercular Disease—Heart Disease—Apnœa—Pneumonia—Bronchitis—Plastic Bronchitis—Cancer of Lung—Emphysema—Excessive Exertion—Foreign Bodies in Air-passages—Mechanical Injury of the Chest—Aneurysm—Scurvy—Purpura—Uræmia — Excess of Alcohol—Vicarious — Hysterical — Hæmorrhagic Diathesis—Pulmonary Hæmorrhage a Cause of Phthisis—Case of Recovery after Copious Hæmoptysis—Treatment.

Since the time of Laennec, it has been very generally assumed that hæmoptysis, when not associated with valvular disease of the heart, or vicarious of the catamenial discharge, is almost always a result and an indication of tubercular disease of the lung. I have long known and taught that this doctrine, thus broadly stated, involves a large amount of error. I have seen a considerable number of cases of hæmoptysis, in which there has been no evidence of structural disease within the chest, either at the time or for months and even years afterwards. Spitting of blood is a symptom, which at the best is sufficiently alarming; there is no need to aggravate its terrors by the erroneous assumption that it is almost invariably associated with serious organic disease either of the heart or the lungs.

Let a man who has once spat blood apply to an insurance office, and the probability is that he will be rejected, or required to pay a large increase of premium; yet it may be that the blood-spitting was as much the result of a harmless accident as if the nose had been the source of the bleeding. The lung is, by far, the most vascular organ in the

[1] *British Medical Journal*, Feb. 12, 1870.

body. In addition to its own nutrient bronchial vessels, the whole of the blood, from every other organ, passes through the pulmonary capillaries. These two systems of vessels in the lung, in consequence of their relation to each other, to the heart, to the movements of the chest, and to the function of respiration, are more liable to sudden strain and pressure than the blood-vessels of any other organ; and it would be indeed marvellous if bleeding did not frequently occur from their accidental rupture, without previous disease.

Now let us pass in review the chief known causes of hæmoptysis; and by hæmoptysis in this lecture, I mean hæmorrhage from the air-passages, excluding cases of bleeding from the gums or fauces.

First, then, tubercular disease of the lung is a frequent cause of hæmoptysis. Certain of the pulmonary capillaries are compressed by a tubercular deposit; the surrounding vessels are subjected to increased strain; they consequently give way, and hæmorrhage occurs. In a more advanced stage of the disease, when softening of the deposit is in progress, blood-vessels may be opened by ulceration. In some instances, considerable branches of the pulmonary artery in tuberculous lungs become aneurysmal; and the rupture of such an aneurysm may be a source of sudden, copious, and even fatal hæmorrhage.

Disease on the left side of the heart is a well-known cause of hæmoptysis. Suppose an incompetent mitral valve: the left ventricle drives a portion of blood forcibly backwards into the lungs, while the right ventricle continues to propel the blood onwards. The pulmonary capillaries, strained by the increased pressure upon their walls, give way, and the result is hæmoptysis and sometimes pulmonary apoplexy.

Disease on the right side of the heart is comparatively rare; but, when it does exist, it not unfrequently causes bronchial hæmoptysis. In the chapter *On some Results of a Retrograde Engorgement of the Blood-vessels* (p. 51), I have described the case of a woman whose sole organic disease was great dilatation of the tricuspid orifice. The whole systemic venous system was much engorged, in consequence of the reflux of blood through the tricuspid orifice. The bronchial

veins and capillaries were partakers in this engorgement, and the result was rupture of bronchial capillaries and hæmoptysis. In cases of congenital narrowing of the pulmonary artery, hæmoptysis has been a frequent symptom. This, again, is a result of a retrograde engorgement of the bronchial veins and capillaries. These patients, too, have frequently become phthisical. I shall presently refer to the occurrence of phthisis as a *consequence* of pulmonary hæmorrhage.

Bronchial hæmoptysis, resulting from disease on the right side of the heart, will help us to understand the hæmoptysis of apnœa. Thus hæmoptysis, to a variable extent, is an occasional accident in cases of spasmodic asthma. What is the explanation of this ? Bronchial spasm limits the supply of air to the pulmonary capillaries. In consequence, the minute pulmonary arteries, by their contraction, check the onward movement of the blood. The systemic arteries are comparatively empty, while the whole systemic venous system is distended. The bronchial veins and capillaries share in this distension ; and hence bronchial mucous exudation, and occasionally bronchial hæmorrhage.

Active inflammatory congestion of the pulmonary and bronchial vessels is a frequent source of hæmorrhage. In most cases of pneumonia, the expectoration is more or less blood-tinged. The mucous expectoration of bronchitis is not unfrequently mixed with blood. In both pneumonia and bronchitis, profuse hæmoptysis is an occasional occurrence, quite unconnected with tubercular disease.

Plastic bronchitis is a rare disease. I have met with it only twice. When it does occur, hæmoptysis is a frequent symptom.

In cases of primary cancer of the lung, the sputa are more or less blood-tinged, and not unfrequently assume the appearance of red-currant jelly.

In several cases of hæmoptysis that have come under my observation, emphysema of the lung has been the only structural change that I could discover. In most of these cases, the exciting cause of the hæmoptysis has been either an attack of bronchitis or over-exertion. In one case, a fat lady with emphysematous lungs, while suffering from an attack of

catarrh, had profuse hæmoptysis after walking up-stairs. She died some years afterwards of Bright's disease, but had no symptom of any other pulmonary disease than emphysema, with occasional catarrh.

A former clinical clerk, a powerful muscular man, who has over-strained his lungs and rendered them emphysematous by excessive gymnastic exertion, has twice had hæmoptysis after playing at football. He remains in good health.

Without doubt, excessive exertion may cause rupture of pulmonary vessels and hæmoptysis in persons whose lungs and heart are perfectly sound; but it is obvious that vesicular emphysema is a predisposing cause of hæmoptysis from over-exertion. Vesicular emphysema is associated with more or less obliteration of pulmonary capillaries, and with hypertrophy of the right ventricle consequent on the impeded circulation through the lungs. Therefore, when the heart's action is excited by active exercise, the pulmonary capillaries are injected with great force by the strong right ventricle, and the result may be rupture of vessels and hæmorrhage.

In a considerable number of recorded cases, the irritation of the air-passages by a foreign body introduced through the larynx has been attended with bronchial hæmoptysis.

It is obvious that a blow on the chest, either with or without fracture of the ribs, and a consequent wound of the lung, may rupture vessels, and cause hæmoptysis. So an aneurysm of the aorta, or of one of its primary branches, may open into the air-passages, and cause blood-spitting.

In all the cases of hæmoptysis to which I have referred, the hæmorrhage results from various forms of structural disease or injury, either in the lungs, or heart, or blood-vessels; but pulmonary hæmorrhage may occur unassociated with any appreciable structural change within the chest. In some of these cases, deterioration of blood, and consequent weakening of the walls of the vessels, are the probable causes of the hæmorrhage. Thus hæmoptysis is one of the forms of hæmorrhage which is not unfrequently associated with scurvy, with purpura, and with uræmia. I have repeatedly seen both profuse hæmoptysis and epistaxis as results of an habitual excess of alcohol and a deficiency of nutritious food.

Hæmoptysis is occasionally vicarious of the menstrual discharge. In cases of this kind, we must guard against deception. I once had a patient whose blood-spitting was supposed to have this origin. On examination, however, I discovered the source of the bleeding in numerous scratches which she had made with a needle in the roof of her mouth. I believe that this case is a type of some other supposed cases of vicarious hæmoptysis.[1]

Lastly, there are cases in which there is no apparent cause for the hæmoptysis; and, to conceal our ignorance, we speak of the hæmorrhage as resulting from a *hæmorrhagic diathesis*. The following case, recently in the hospital, is an example of this.[2]

John Herring, aged 37, a blacksmith, was admitted December 18, on account of hæmoptysis, which began ten days before his admission. He said that, for a fortnight before he began to spit blood, he had suffered from pain in the right side of the chest. The hæmoptysis began on December 8, and had continued daily until his admission. One day, December 11, he brought up as much as three-quarters of a pint. The blood, he said, had varied in colour, sometimes being dark, at other times bright red. He said that, twelve years ago, he had pleurisy; that five or six weeks after this he first spat blood, and that he had since had frequent attacks of hæmoptysis. He had sometimes passed nine months without an attack; at other times he had had two attacks within a week. The blood-spitting had generally continued from twenty-four to thirty hours. If it continued longer, he usually stopped it by a dose of elixir of vitriol. Three years ago he had inflammation of the lungs, and was laid up for six months, but during this illness he had no hæmoptysis. He said that he had always been temperate. His father died of old age (upwards of 80); his mother from 'change of life, and something wrong in her head.' He has three brothers and a sister in good health; none have died; his grandparents all lived to old age. On admission, he was a fairly nourished man; but his face and lips were pallid. He complained of a

[1] See Chapter XI., *On Hysteria*.

[2] *Hospital Case Book*, vol. xxxix., p. 75.

troublesome cough, and he expectorated a quantity of dark yellowish-brown blood-tinged material, having a fœtid odour; his breath, too, was fœtid, and there was some pain over the right side. Tongue coated, appetite bad, bowels costive, urine normal; the skin was moist; at night, sometimes profuse perspiration; pulse, 66; temperature, 99°·8. Percussion gave natural resonance over the whole front of the chest; behind, there was some dulness on percussion on the right side, from the spine of the scapula to the base of the lung; over the dull space there was rather large crepitation, with diffused blowing expiration; the vocal resonance and vibration were unchanged over the dull space; elsewhere the respiratory sounds were normal, both front and back; the heart's sounds and action were normal. He was ordered to take ten grains of gallic acid every three hours; to inhale the vapour of turpentine and boiling water night and morning. On December 22, the expectoration had ceased to be blood-tinged; it was of a dark-greenish colour, and still had an offensive odour. Temperature, 98°·5; the dulness and crepitation over the lower lobe of the right lung were diminished; he was ordered to inhale creasote instead of turpentine. He continued steadily to improve; the expectoration gradually ceased; and he left the hospital on January 15. The day before his discharge it was noted that there was normal resonance over the right lower lobe; inspiration was vesicular and free from crepitation. The only abnormal sound was somewhat prolonged expiration over the right back.

Now here is a case in which a frequently recurring hæmoptysis, extending over a period of twelve years, is pretty certainly not associated with organic disease of the lung, either as a cause or a consequence. When he came in, the physical signs indicated that the lower lobe of the right lung was partially consolidated, probably by blood, which had been driven into the extreme bronchi and air-cells, where it appears to have decomposed and become fœtid. This blood was gradually expectorated; the lung has recovered its normal condition; and the man has been restored to his usual state of health.

Although I have long known that hæmoptysis not un-

frequently occurs unconnected with phthisis, it is only recently[1] that I have learnt that pulmonary hæmorrhage is an occasional exciting cause of phthisis. Laennec taught us to believe that, when profuse hæmoptysis is quickly followed by the symptoms of a rapid phthisis, tubercles were latent in the lung and caused the hæmorrhage. Niemeyer has lately revived the doctrine, which was accepted before the time of Laennec, that a copious pulmonary hæmorrhage, in a person previously healthy, may be an exciting cause of phthisis.[2] Niemeyer's doctrine is, that a portion of the blood, being driven down to the ultimate bronchi and air-cells, acts as a foreign body, and sets up inflammatory and degenerative changes in the lungs, from which the patient may slowly recover, or which may result in cheesy deposits, excavations, and fatal phthisis.

In the second volume of the Clinical Society's *Transactions* there are two interesting papers: one, by Dr. Christian Bäumler, on 'Cases of Hæmoptysis followed by Inflammatory Changes in the Lungs;' another, by Dr. Hermann Weber, on 'Hæmoptysis as a Cause of Inflammatory Processes and Phthisis.' These papers are confirmatory of Niemeyer's doctrine; and I am sure that in the main the doctrine is true, for it has rendered intelligible to me some facts in my past experience, which before I could not comprehend.

On May 10, 1867, I first saw a married lady, aged about 25, who, a month before, during a violent fit of coughing, had brought up a large quantity of blood. Since the hæmoptysis, she had been weak and ill, but not confined to bed. I found all physical signs normal at the upper and front part of the chest; but, over the right base at the back, there were some dulness on percussion, and rather loose and large bubbling rhonchi. The family history indicated no tendency to phthisis. The history and the physical signs were not those of tubercular disease, of ordinary pneumonia, or of ordinary bronchitis. I have no doubt that, consequent on the pulmonary hæmorrhage, blood had been driven into the lower

[1] This was said in 1870.

[2] See Niemeyer's *Text-Book of Practical Medicine*, translated by Drs. Humphreys and Hackley, of New York.

lobe of the right lung, and had there excited inflammatory changes. I saw this lady occasionally for some months, during which she gained strength, but the physical signs remained unchanged. I have heard quite recently (January 1870) that her health has continued to improve; but her medical attendant in the country says there is still dulness and crepitation over the right base, and still absence of abnormal physical signs over the upper and front part of the chest. There is reason to hope that ultimately she may recover from the accidental results of the pulmonary hæmorrhage, though the long continuance of the physical signs gives the case a somewhat serious aspect.

[This prediction has been completely verified: the patient's health continued to improve, and when I last saw her, on June 26, 1874, she had grown quite stout. The dulness at the right base had disappeared, the respiratory murmur was rather feeble there, but there were no moist sounds. In 1875 she went to India with her husband, and I hear from a relative (January 1887) that, with the exception of occasional slight ailments, she has been in good health and has grown very stout.]

In Chapter XXXVII., on *Thrombosis and Embolism*, will be found the case of a lady, aged 40, in which the fatal consequences of a pulmonary hæmorrhage were of an unusual character.

You see, then, that pulmonary hæmorrhage is a subject which demands a careful and diligent study. Each case requires a minute and accurate investigation before we can venture to give an opinion as to the cause or the probable consequences, or the appropriate treatment. There are few cases in which an off-hand, inconsiderate opinion is more likely to be erroneous, and, therefore, unjustifiable, than in these alarming cases of blood-spitting. The fact that hæmoptysis is often associated with serious organic disease, either as a cause or a consequence, renders it the more important that the antecedents and the circumstances of its occurrence should, in every case, be thoroughly investigated.

The *treatment* of pulmonary hæmorrhage will vary somewhat according to the nature of the exciting cause. There

are, however, certain general rules which are applicable to all cases. The patient must remain as absolutely at rest as possible. Bodily exertion or emotional excitement, by increasing the force and frequency of the heart's contractions, is apt to increase the bleeding, or to provoke a return. The patient should lie still, and neither move, speak, nor cough, more than is absolutely necessary. A cough is a powerful provocative of pulmonary hæmorrhage, and it is sometimes desirable to allay irritation and cough by morphine, which not only quiets the cough but which, there is reason to believe, excites contraction of the muscular arterioles, and so tends to arrest hæmorrhage. Do your utmost to prevent the patient from being alarmed and excited by the sight of the blood. Let him have a plentiful supply of cool fresh air. Let him keep lumps of ice in the mouth and swallow the cold water. Sometimes the application of ice to the chest has a powerful effect in arresting the bleeding. It should not be continued long enough to chill the patient; and it is more likely to be effectual in exciting sympathetic contraction of the deeper arteries when applied for a short time over different parts of the chest, both front and back, than when applied continuously over one part. Gallic acid I believe to be one of the best styptics in cases of pulmonary hæmorrhage. It may be given in doses of ten grains, in the compound infusion of roses, every three hours, if the bleeding be copious, and less frequently as the bleeding subsides. Another useful styptic is the liquid extract of ergot. This, too, may be given every three hours, in doses of half a drachm. It may be given alone or combined with the gallic acid mixture. The oil of turpentine in 20-minim doses sometimes succeeds when other remedies fail; and I have seen the bleeding quickly arrested by the inhalation of turpentine vapour. We directed our patient, Herring (p. 460), to inhale, first turpentine, and subsequently creasote, for two reasons: first, to correct the fœtor of the expectoration and the breath; and, secondly, to promote the expulsion of the decomposing blood from the lung, and thus prevent the risk of mischief from its retention. You will find the inhalation of turpentine vapour an excellent expectorant in this and other cases where the object is to expel accumulated material from the air-passages.

An emetic of sulphate of zinc may sometimes be given for the same purpose. Dr. Weber, in the paper before referred to, recommends a combination of antimony and ipecacuanha as an emetic, partly to expel blood from the lung, and partly to arrest bleeding.

CHAPTER XXXI.

CLINICAL LECTURE ON PHTHISICAL PERFORATION OF THE PLEURA, WITH PNEUMOTHORAX.[1]

Case of Phthisis with Perforative Pneumothorax—Physical Signs at Different Stages of the Case—*Post-mortem* Inspection—Clinical Commentary—Case of Perforative Pneumothorax in a very Early Stage of Phthisis—A similar Case—Explanation of Absence of Fluid in the Pleura—Splashing Sound on Succussion in Cases of Hydro-Pneumothorax—A Case of sudden Perforative Pneumothorax with rapid and complete Recovery—Reference to other Cases—Case of Pneumonic Perforative Pneumothorax with Dry Pleurisy on the Opposite Side—Ultimately Paracentesis and Drainage—Complete Recovery.

I HAVE now (January 7, 1878) under my care a case in which there is very conclusive evidence that a softened tuberculous deposit in one lung has caused perforation of the pleura, with resulting pneumothorax—that is, an escape of air into the cavity of the chest. The symptoms and the physical signs which occur in connection with this serious accident are very remarkable and very interesting; and I propose now to give you a brief history of the case, as recorded by my clinical clerk, Mr. Macdonald, with some practical and explanatory comments thereupon.

G. G——, aged 26, an Italian stonemason, employed at the new Law Courts, was admitted into Sambrooke ward on December 26, 1877. He came to this country two months ago. For about six weeks he has had cough, with expectoration of phlegm and occasional slight blood-spitting. Two days before his admission, he was suddenly seized with pain in the right side of the chest, and great difficulty of breathing, which confined him at once to his bed. On admission (December 26), the respirations were 54, pulse 140, temperature 105°. He complained of pain below the right

[1] *British Medical Journal*, Feb. 23, 1878.

nipple, extending through to the back. There was frequent short cough, with scanty muco-purulent expectoration. On inspection, it was seen that, while the left side of the chest moved freely, the right side was nearly motionless. The apex of the heart was seen and felt beating in the sixth interspace, external to the left nipple. The liver was felt extending at least two inches below the margin of the ribs on the right side. On percussion, the whole right side of the chest was more resonant than the left, though the resonance had not a tympanitic character. Over the whole left side there was loud puerile respiration; while on the right side the first impression was that respiratory sounds were entirely absent; but, on careful auscultation below the nipple, I discovered at each inspiration a distinct so-called 'metallic tinkling;' and some of you will remember that I then expressed my opinion that a tuberculous deposit near the surface of the lung had softened, and perforated the pleura; so that, at each inspiration, air passed into the cavity of the pleura, giving rise to the condition called *pneumothorax*. The inspiratory metallic tinkling is caused by bubbles of air bursting through a small opening on the surface of the lung into the air-filled cavity of the pleura, and there giving rise to a resonant echo. The air enters the cavity of the pleura more freely than it passes out (no expiratory sound was heard). The reason of this is that, during inspiration, the partial expansion of the lung renders the perforation patulous, while during expiration the elasticity of the collapsing lung partially closes the aperture, and thus retards the escape of air from the pleural cavity. The consequence is, that the right side of the chest has become distended with air, which pushes the heart to the left, and the diaphragm and liver downwards.

There is yet another remarkable physical sign. When the patient speaks or coughs, the voice and the cough are heard to resound with what is called an amphoric echo: a similar echo to that which is heard when we speak with the mouth over the orifice of an empty decanter, or over the bung-hole of an empty wooden cask.

The prognosis was very grave. The past history—in particular the hæmoptysis—rendered it nearly certain that

there was tuberculous disease of the lung; and now the right lung, being compressed by the air upon its surface, had collapsed, and was thus rendered useless; in addition to this, some pus and softened tuberculous matter had escaped into the cavity of the pleura, and acute pleurisy had been the result.

The right side of the chest was ordered to be covered by a linseed poultice, and 15 minims of liquor morphinæ to be given three times a day.

On December 31, when I saw him for the second time, the metallic tinkling sound, or amphoric crepitation, as it is better called, was replaced by a loud inspiratory amphoric blowing, most distinctly heard below the right mamma in front, and behind in the right inter-scapular region. The amphoric echo of the voice and cough remained as before. The change from amphoric crepitation (metallic tinkling) to amphoric blowing is probably to be explained by the enlargement of the opening in the pleura permitting a fuller current of air to pass at each inspiration into the cavity of the pleura. The sound is heard only during inspiration, because the rush of air into a large cavity is essential for its production, while the slower passage of air from the pleural cavity into the lung in expiration is unattended by audible sound. The sound is exactly imitated by blowing sideways into a decanter, or into a large metal tube, such as a gun-barrel.

On January 3 the physical signs on the right side remained the same, except that there was some dulness on percussion extending to near the angle of the scapula, indicating liquid effusion into the pleura. On the left side, near the angle of the scapula, a dry friction-sound was heard on inspiration. Inflammation had extended from the right to the left pleura, which was now roughened by lymph.

On January 27 (the day on which this lecture was given), the auscultatory signs had again undergone a remarkable change. The amphoric blowing was no longer audible, and no respiratory sound of any kind was heard over the right side of the chest; while the amphoric echo of the cough and voice continued. What is the explanation of this change? The most probable interpretation is, that the orifice in the pulmonary

pleura has been closed by a recent exudation of lymph, by which the passage of air from the lung into the cavity of the pleura is prevented. The notes indicate that the temperature has ranged from 101° to 103° until within the last two days, when, with increasing weakness, it has fallen to 98°; the respirations have risen from 36 to 54; and the pulse, from 120 to 140. There has been a rapid decline of strength, and it is evident that the disease must soon terminate fatally.

Subsequent History, with a Report of the Inspection of the Body.—Death occurred on January 8, within twenty-four hours after the preceding lecture was given; and the following is a condensed report of the inspection, which was made by the Pathological Registrar, Mr. Barrow, thirty-three hours after death.

On opening the abdomen, the liver was found to be pushed down to within an inch of the umbilicus. The convexity of the diaphragm on the right side was on a level with the ninth rib, while on the left it extended as high as the fifth or sixth rib. The heart was displaced entirely to the left of the median line. On puncturing the right side of the chest, a quantity of air escaped. The right pleural cavity contained about six pints of greenish sero-purulent liquid. The right lung had completely collapsed; its entire surface was covered by a thick layer of recent lymph; and at its base were two masses of soft lymph, the larger one being as large as a kidney. When the lung was inflated by blowing air into the trachea, no air escaped from the surface until the soft lymph had been carefully scraped off, when a circular opening large enough to admit the blunt end of an ordinary probe was discovered, at about the middle of the posterior surface of the lower lobe of the lung. Through this opening, after the removal of the recent exudation, air escaped freely when the lung was inflated. On incising the lung at that spot, the opening was found to communicate with a cavity about the size of a large walnut; the cavity being partly filled by a purulent secretion, and surrounded by condensed lung-tissue. The apex of the lung was consolidated, and contained several caseous masses; but in no other part had softening and excavation occurred. The posterior surface of the left lung was

partly covered by a thin layer of recent lymph. The apex of the left lung was condensed, of a greyish colour, and having a shotty feel, with here and there a small mass of caseous deposit. No other noteworthy morbid appearance was discovered.

Subsequent Clinical Commentary on the Case.—It will be seen that, in this interesting case, the *post-mortem* appearances corresponded very exactly with the physical signs observed during the various stages of the disease; and in particular my bedside interpretation of the replacement of the amphoric crepitation (metallic tinkling) by amphoric blowing, and the final cessation of the latter auscultatory sign, seems to be completely confirmed. The phenomena of this case are entirely in accordance with Fournet's doctrine that, in cases of pneumothorax from perforation of the pleura, the phenomena of metallic tinkling and amphoric blowing will cease, if complete closure of the opening be effected. Without doubt, a crepitating echo (metallic tinkling) may occur in a closed pleura containing air and liquid, as a result of the agitation of the liquid by coughing or movement of the body (succussion), or the fall of drops of liquid from the upper to the lower parts of the pleural cavity; and perhaps even, as Dr. Walshe suggests,[1] 'the echo of rhonchi in the adjacent bronchial tubes may cause tinkling;' but, for the production of a true amphoric blowing, I am firmly persuaded that a free current of air into a large cavity is essential. In all the cases in which the phenomenon of amphoric blowing has come under my observation, this physical sign has been associated with one or other of the three following conditions: 1. A large cavity in the lung communicating with a bronchus; 2. Perforation of the lung, with an inspiratory current of air into the cavity of the pleura; 3. Pneumothorax, with a fistulous opening in the chest-wall, and a consequent inflow of air during inspiration.

Some of you will probably remember a boy named Ratcliffe who was under my care in Sambrooke ward in the summer of 1875. After being tapped for empyema, he had a fistulous opening in the chest-wall for several weeks, but ultimately

[1] *On Diseases of the Lungs*, 4th edition, p. 156.

recovered completely. While the fistulous opening with pneumothorax continued, we heard sometimes amphoric crepitation (metallic tinkling) caused by air-bubbles bursting through the fluid secretion of the wound into the air-containing cavity of the pleura; at other times, amphoric blowing, when a fuller current of air was inspired through the fistulous opening nearly or quite free from fluid. These phenomena could at any time be temporarily suspended when the fistulous orifice was closed by the pressure of a finger or a plug of lint.

In the great majority of cases of perforation of the pleura with pneumothorax, the diagnosis is attended with little or no difficulty. The physical signs which I have before referred to, occurring in a patient who has been suddenly seized with hurried and difficult breathing and pain in one side of the chest, are in the highest degree conclusive. In some cases, however, the physical signs are not so unequivocal as in that which I have here recorded. Some years since, I had in the hospital two cases in which there was a somewhat perplexing inconsistency of physical signs. In one case, with tympanitic resonance over the lower and back part of the chest, amphoric blowing and amphoric echo of the voice and cough, there was dulness on percussion over the upper and front part, with moist rattling sounds. This was explained after death by a pretty extensive adhesion of the upper lobe of a tuberculous lung to the chest-wall. As these adhesions between the pulmonary and the costal pleuræ are the chief protection against perforation, so the forcible tearing asunder of an adhesion during a fit of coughing is the most frequent immediate cause of the accident. The other case of pneumothorax to which I referred, was one in which, with increased resonance over the whole of the left side, without amphoric blowing, a distinct, though feeble, respiratory murmur could be heard down to the very base of the lung. On inspection, it was found that the centre of the base of the lung, over a surface about two inches in diameter, was firmly adherent to the diaphragm. This adhesion prevented the complete collapse of the lung, and the descent of the diaphragm in inspiration drew a limited amount of air into the lung.

The case which we have been considering affords an illustra-

tion of the fact that perforation of the pleura, with resulting pneumothorax, may occur in an early stage of phthisis. Many years ago, I saw, with my old friend the late Mr. Lavies, a case in which this fatal accident occurred at a yet earlier stage. An adult male prisoner in the Westminster House of Correction, having had a cough for a few weeks, was suddenly seized with pain in the right side and distressing rapidity of breathing. There was excessive resonance on percussion over the affected side, while the normal respiratory sound was replaced by a loud amphoric blowing. The diagnosis of perforation of the pleura with pneumothorax was obvious. The man died in a few days, and in the apex of the lung was a scanty caseous deposit, the only part that had undergone softening being a piece not larger than a pea, immediately beneath the perforated pleura. In all cases, perforation of the pleura with pneumothorax is attended by the sudden onset of symptoms of more or less intensity. The collapse of the lung and the consequent sudden great diminution of the vital capacity of the chest always cause more or less urgent dyspnœa. The pain and the other symptoms associated with the onset of acute pleurisy are, I believe, mainly influenced by the amount of purulent secretion which escapes from the lung into the cavity of the pleura. When a pus-secreting cavity of considerable size opens into the pleura, the escape of its contents rapidly excites intense inflammation in the serous cavity; while, on the other hand, a perforation which does not communicate with a cavity is attended with little or no pleuritic inflammation. On looking back to some notes of cases seen when I was physician to the Public Dispensary, I find that, in the case of a man whom I saw in March 1847, although he lived for five days after the occurrence of perforation with pneumothorax, no liquid in the pleura or other evidence of pleurisy was found after death. In that case a very small perforation had occurred at the apex of the left lung, in contact with a piece of softening tubercle, but not communicating with a cavity; and this, as I believe, explains the absence of pleuritic complication.

In that case, there was absence of amphoric blowing, explained, as I then believed and noted, by the extreme smallness of the aperture in the pleura. It is probable that the

collapse of the lung, after the occurrence of perforation may have entirely closed the aperture through which the air first escaped. The diagnosis of perforation with pneumothorax was based upon the sudden onset of pain and dyspnœa in a phthisical subject, exaggerated percussion-resonance over the left side, very feeble breath-sounds, and crepitation which was attended with amphoric echo, beneath the clavicle. From this, I inferred that the apex of the lung was partially adherent to the chest-wall; and this diagnosis was verified by the *post-mortem* examination.

You are aware that when there is a mixture of air and liquid in the pleura, a sudden jerk (succussion) of the patient's body when he is standing or sitting, causes a splashing sound, which is easily heard when the ear is applied over the affected side. This physical sign has been known to the profession since the time of Hippocrates. We did not seek for it in our patient's case. We had abundant diagnostic evidence without it; and the patient's feebleness and suffering were so great that it was undesirable to add to his distress by the rather rough procedure which is requisite to develop the phenomena in question. An over-zealous auscultator is in some danger of forgetting that a minute and prolonged, and especially a rough, examination of the chest may be a source of fatigue, distress, and even danger to the patient.

APPENDIX TO CHAPTER XXXI.

A Case of Sudden Perforative Pneumothorax, with rapid and complete Recovery.[1]

On November 15, 1881, I saw at Woolwich, in consultation with Surgeon-Major Godwin, Master H. A——, æt. 17, whose history was as follows:—He had been at school at Gosport, working hard and successfully and in good health.

On November 2 he ran about twenty miles in a paper-chase, when he was so much fatigued that he dropped on the ground. He, however, quickly recovered, and during the two following days he was in his usual good health, doing his school

[1] Reprinted from Vol. XV. of the *Clinical Society's Transactions.*

work and exercising himself in the gymnasium. On the third day after the paper-chase, November 5, soon after his midday dinner, and immediately after running up-stairs, he was suddenly seized with severe pain in the left side of the chest, urgent dyspnœa, and extreme prostration. Mr. H. S. Wharton, of Gosport, who was immediately sent for, found the boy lying on his back gasping for breath, and, as he says in a note with which he has favoured me, 'Knowing him to be an emotional lad, who was likely to faint from very slight causes, I thought a distended stomach was at the bottom of his troubles. I ordered him to bed, and on my next visit put my ear to his chest in a casual way, not dreaming that there was anything amiss there, as he had no pain or cough leading me to suspect thoracic mischief, when to my surprise I could hear no respiratory murmur on the left side; but in its place the most positive evidence that the pleura was distended with air, there being amphoric resonance over the whole left side and the heart displaced to the right.' The diagnosis was confirmed by Dr. Miller, of Southsea, who saw the patient in consultation with Mr. Wharton.

About four or five hours after the onset of the attack the pain and dyspnœa, which had been urgent, entirely passed away, and so long as he remained at rest did not return.

After a few days' rest in bed, his father took the patient to his home at Woolwich, on November 14, where, on the following day, I saw him with Dr. Godwin. He was in bed, but declared that he was quite well, and wished to know when he might go back to his school work. He had a slight short and dry cough, but no pain or dyspnœa. Pulse 60; respiration 24; temperature 97°. The physical signs of perforative pneumothorax were unequivocal. Over the whole left side, which was comparatively motionless, there was hyper-resonance on percussion, except in the interscapular region, where, in the probable position of the collapsed lung, the resonance was rather less than at the corresponding point on the right side; over the same limited space there was a feeble and, as it were, a distant sound of respiration. Elsewhere over the whole left side, the normal respiratory murmur was replaced by loud amphoric blowing and an occasional crepitating echo ('metallic

tinkling'). There was amphoric echo of the cough and voice. There was no evidence of liquid in the pleura; no dulness at the base; no splashing sound on succussion. The heart was felt and heard beating in the epigastrium, and to the right of the sternum.

As there were no symptoms requiring treatment, it was agreed to keep him in bed on simple diet, without medicine of any kind.

On November 27, Dr. Godwin was good enough to write to me, and his report indicated considerable improvement; the main points being 'increase of movement on the left side, absence of amphoric blowing and metallic tinkling, distinct respiratory murmur down the left side of the spine; the heart's impulse felt to the left of the sternum; general health excellent in all respects.'

Again, on December 23, Dr. Godwin reported the result of a conjoint examination made three days before by his colleague, Dr. Stevenson, and himself, to this effect:—'Vesicular murmur is now plainly audible over the whole left chest; it is weaker over the lower lobe than the upper, but yet quite distinct; the heart is well over to the left side. His general health excellent. About a fortnight ago he had a return of pain similar to that which caused him such agony when he was first attacked, but much less intense. On examination at that time I found that the heart had made a decided move towards its natural position. The pain lasted only a few hours. He has now gone off to Brighton with his parents.'

On January 11, 1882, his parents brought him to me. He then had no cough or other sign of illness, and the only noticeable difference on a careful comparison of the two sides of the chest, was a somewhat doubtful flattening, with diminished inspiratory movement, in the left subclavian region. The respiratory and vocal phenomena were everywhere quite normal. He has since returned to school, and his first medical attendant there, Mr. Wharton, writes to me:—'As far as I can determine, he is perfectly cured of his pneumothorax.'

The question arises, What was the cause of the breach in the texture of the pleura which resulted in the pneumothorax?

His parents reminded me that I had once before been

consulted about him, and on referring to my case-book I found that on May 19, 1879, when he was 15 years of age, he was brought to me on account of a cough, which I attributed to enlargement of the tonsils. I made no note of the condition of the chest, but I have no doubt that I examined it with a negative result.

A younger brother has lately died, after a long illness from scrofulous disease of the hip and vertebræ. I think it probable that at some former period there had been a slight structural change at the apex of the left lung, resulting, as so commonly happens, in the formation of local adhesions between the pulmonary and costal pleuræ; and that, as a result of the violent exertion which he had recently undergone, these old adhesions became stretched and partially ruptured, so as ultimately to cause a rent in the texture of the pleura, with consequent pneumothorax. Then, as there was no purulent or other morbid secretion which, by escaping into the cavity of the pleura, would excite inflammation and empyema, the ruptured pleura was soon repaired, the escaped air was gradually absorbed, the lung again expanded, and the heart resumed its normal position.

Perforative pneumothorax, being usually associated with phthisis, is almost invariably fatal. I have seen a considerable number of cases of phthisical pneumothorax, but this is the first case of recovery that has come under my observation. I am indebted to Dr. Stephen Mackenzie for a reference to a case of recovery published by himself.[1] The patient was a man, æt. 50, who had for many years suffered from cough and shortness of breath. The pneumothorax, which occurred suddenly, was supposed to have resulted from the rupture of an emphysematous air-vesicle. The air was pumped out of the chest by an aspirator. The symptoms and physical signs passed away, and the patient left the hospital in three weeks, feeling better than he had done for months. Dr. Mackenzie also referred me to a short paper by Dr. Wilks,[2] in which three cases of recovery from simple pneumothorax are briefly recorded.

[1] *Lancet*, August 19, 1871, p. 259.

[2] *British Medical Journal*, Feb. 2, 1874, p. 770.

Two of the cases occurred in Guy's Hospital, one being a young man, the other an elderly man, under the care of the late Dr. Hughes, who suggested that the pneumothorax had resulted from rupture of a 'bullous portion of an emphysematous lung.' Both patients made a complete recovery. The third case was that of a lady, æt. 30, who in the eighth month of her pregnancy, after a slight cold, had acute perforative pneumothorax. The symptoms gradually subsided; she was soon delivered of a healthy child, and her recovery was complete. In none of these three cases was any operative procedure resorted to.

Postscript.

Case of Pneumonic Perforative Pneumothorax, with Dry Pleurisy on the Opposite Side. Complete Recovery.

A very remarkable case of complete recovery from perforative pneumothorax with effusion, was one which I attended with my lamented friend, the late Dr. Baxter, in 1882. The temperature charts and the bed-side notes were afterwards sent to Dr. Baxter, and unfortunately have been lost. I regret, therefore, that I can give only a general outline of the case.

H. C——, æt. 19, a tall and not very muscular youth, consulted Dr. Baxter at the beginning of January 1882, on account of a cough which had troubled him for about a week. Dr. Baxter sent him home to bed, and in a few days he presented, with a moderately high temperature, the physical signs of pneumonia at the base of the left lung. A few days later there was a sudden access of dyspnœa, and with this the physical signs of pneumothorax on the left side. At this stage of the case Dr. Baxter asked me to see the patient with him. There was tympanitic resonance over the whole left side of the chest, with amphoric blowing, and the heart was displaced to the right. In about another week there were physical signs of liquid in the left pleura, dulness on percussion, with splashing sounds in succussion; and about the same time he was rather suddenly seized with severe pain over the right side of the chest, where a dry pleuritic friction sound was extensively

heard. The patient's condition seemed desperate, with one lung collapsed and useless, while the other was embarrassed by the agonising pain which attended every inspiration. The distress of breathing was extreme, and the pulse very rapid and feeble. He was well supplied with nourishment and stimulants, and the painful side was poulticed and fomented; but the main reliance was placed upon repeated doses of morphine. He had a drachm of the liquor morphinæ twice, and occasionally three times, in the twenty-four hours. By degrees the pain, and with it the urgent dyspnœa, subsided. Meanwhile, the fluid in the left pleura was found to have increased, while the amphoric blowing had ceased, and his general condition was much improved.

On March 29 he was removed to a private hospital in Fitzroy Square, and on the following day, my friend Sir Joseph Lister drew off fifty-four ounces of clear serum, without a trace of opacity. I am indebted to Sir Joseph for the following notes of the further progress of the case: 'A large drainage-tube was inserted in the free space obtained by removing a portion of one of the lower ribs in the axillary line, and an antiseptic dressing was applied. The fluid remained serous to the last, a good illustration of the different results of antiseptic from ordinary treatment. In the early part of April, the discharge having become very slight, the tube was removed, and the wound was allowed to heal. But on returning from a stay of about three weeks in the country, I found that the fluid had re-accumulated, and on May 4 I opened up the cicatrix and re-introduced a tube. The quantity of fluid let out on the second occasion happened to be exactly the same as on the first—fifty-four ounces—and again it was purely serous. This time I took care to retain the tube longer. It was finally removed on June 3, and he left convalescent on the 14th of that month.'

The youth has remained well since his serious illness. In a letter dated January 28, 1887, he says: 'I am as strong now as I ever was, and have never had the slightest cold or illness since my return home.' The most critical period of the case was during the two or three days of agonising pleuritic pain

on the right side, while the left lung was entirely collapsed and useless. There can, I think, be no question that the repeated doses of morphine enabled him to weather the storm. The subsequent antiseptic drainage of the pleura was most successful.

I have lately (1887) heard from the father of the patient whose case is recorded at p. 473, that the young gentleman has been with his regiment through the late campaign in the Soudan, and that he has not once been on the sick list.

CHAPTER XXXII.

ON CERTAIN DIFFERENCES IN THE PHYSICAL SIGNS BENEATH THE CLAVICLES, ON THE TWO SIDES OF THE CHEST, IN HEALTHY SUBJECTS.

Physical Signs of Phthisis in an Early Stage—Table Showing the Signs in the Infra-clavicular Regions in Fifty Healthy Men—The Great Frequency of increased Vocal Resonance and Vibration on the Right Side—Flattening under one Clavicle from Lateral Curvature of the Spine.

When making a physical examination of the chest in cases of suspected phthisis, attention is especially directed to the apex of the lung, and to a comparison of the respiratory and vocal sounds over the corresponding portions of the two lungs. Any inequality of the physical signs on the two sides is looked upon with suspicion, and may be, in fact, an indication of structural change in the lung.

There are certain auscultatory signs which may result from a slight deposit in the lung—so slight as not to cause an appreciable difference in the percussion note. The chief of these are a feeble inspiratory sound, with harsh and prolonged expiration, also a comparative increase of the vocal resonance, and of the vocal vibration or fremitus on one or other side. Now it is important to know and to bear constantly in mind that there may be a marked inequality of these physical signs in the subclavicular regions of perfectly healthy subjects.

Many years ago, I was one of a committee of four who undertook a careful investigation of this subject, and the results of the inquiry are given in the following tabular statement, which was published in Dr. Sibson's *Medical Anatomy* p. 72:—

THE SIGNS PRESENT IN HEALTH OVER THE UPPER LOBES.

The following observations were made by a Committee consisting of Dr. Baly, Dr. George Johnson, Dr. Quain, and Dr. Sibson, and named by a private Society for the study of Diseases of the Chest.

The observations were made on fifty healthy men, their ages ranging from eighteen to thirty, selected by Dr. Baly from among the inmates of Millbank Prison.

(Published with the sanction of the Society.)

Signs over the First and Second Ribs below the Clavicles.

1. Respiratory Movements:—Number of Observations 47

Equal	45	
Greater on the Right . . .	1	
Greater on the Left . . .	1	

2. Sound on Percussion:—

A. Over 1st and 2nd Ribs:—Number of Observations 50

Equal	39	Including 1 doubtful.
More resonant on the Right	4	All slightly
More resonant on the Left .	7	1 decidedly. 4 slightly. 2 very doubtful.

B. Over 1st and 2nd Cartilages:—Number of Observations . . . 20

Equal	1	Others probably not recorded.
More resonant on the Right	3	
More resonant on the Left .	16	7 decidedly. 9 slightly.

3. Breath Sound during Inspiration, ordinary:—Number of Observations 50

Equal	26	13 Sound distinct. 7 Sound feeble. 3 Sound scarcely audible. 1 Sound inaudible. 2 Undecided as to equality.
Louder on the Right . . .	7	1 decidedly. 3 slightly. 3 predominance somewhat doubtful.
Louder on the Left . . .	17	2 much. 6 decidedly. 8 slightly. 1 slightly, but very faint.

N.B. — In one case no sound heard on right.

4. Breath Sound during Inspiration, forced:—Number of Observations 50

Equal	20	17 decidedly. 2 equality doubtful. 1 scarcely audible.
Louder on the Right	10	6 decidedly. 3 slightly. 1 predominance doubtful.
Louder on the Left	20	14 decidedly. 5 slightly. 1 doubtful.

5. Breath Sound during Expiration, ordinary:—Number of Observations 50

Equal	28	3 distinctly. 6 scarcely audible. 19 inaudible.
Louder on the Right	21	2 much. 7 decidedly. 10 slightly. 1 slightly, but very faint. 1 predominance doubtful.
N.B.—In seven cases no sound heard on left. Louder on the Left	1	Decidedly.

6. Breath Sounds on Expiration, forced:—Number of Observations . . 49

Equal	6	4 more or less loud. 1 feeble. 1 scarcely audible.
Louder on the Right	41	6 much. 23 decidedly. 12 slightly.
Louder on the Left	2	Decidedly.

7. Sound of Whisper:—Number of Observations 42

Equal	0	
Louder on the Right	42	5 much. 29 decidedly. 8 slightly.
Louder on the Left	0	

8. Sound of Voice:—Number of Observations 50

Equal	3	
Louder on the Right	47	4 much. 37 decidedly. 6 slightly.
Louder on the Left	0	

9. Vocal Vibration:—Number of Observations 50

Equal	3	2 distinct. 1 feeble.
Stronger on the Right	47	5 much. 37 decidedly. 5 slightly.
Stronger on the Left	0	

10. Heart's Sounds, especially Second Sound:—Number of Observations 50

Equal	14	
Louder on the Right	29	1 much. 21 decidedly. 6 slightly. 1 predominance doubtful.
Louder on the Left	7	3 decidedly. 4 slightly.

Breath Sounds over the Back.—Supra-spinous Fossa.

11. Breath Sounds during Inspiration, forced:—Number of Observations 16

Equal	7	
Louder on the Right	4	Decidedly.
Louder on the Left	5	2 decidedly. 3 slightly.

12. Breath Sounds during Expiration, forced:—Number of Observations 16

Equal	0	
Louder on the Right	16	12 decidedly. 4 slightly.

For convenience of reference I have numbered the different sections.

The most striking and important inequalities on the two sides are those relating to the vocal resonance and fremitus. Thus, in sections 8 and 9, it is shown that while in only 3 out of 50 healthy men were the vocal resonance and fremitus equal in the two subclavicular regions, in the remaining 47 cases the resonance was louder and the vibration stronger on the right side. In not one case out of the 50 was the vibration greater on the left. In section 7 it will be seen that the sound of the whispered voice was more audible on the right side in every one of the 42 subjects examined. In sections 3 to 6 the results of comparing the respiratory sounds on the two sides are given. It appears that the respiratory sounds

are much more frequently equal on the two sides than are the vocal resonance and vibration. It also appears that an inequality is more frequently observed with forced than with ordinary respiration. When there was a difference in the sound of inspiration on the two sides, in the majority of cases the sound was more feeble at the right side; or, as appears in the table, it was louder on the left.

On the other hand, the expiratory sound, when unequal on the two sides, was, in the great majority of cases, louder on the right side; and with forced expiration this was more frequent than with ordinary expiration, the numbers being: out of 49 cases 6 equal, 2 louder on the left, 41 louder on the right.

The facts here recorded, which have the most important bearing upon the physical signs in the early stage of phthisis, are the great frequency of comparative increase of the vocal resonance and vibration, and of the expiratory sound on the right side. These physical signs, therefore, when observed at the left apex, would be much more probably associated with a slight deposit in the lung than when they occur on the right side.

The occasional differences in the inspiratory sound on the two sides are of less importance.

The fact recorded in section 2, that in 16 out of 20 men examined the resonance on percussion over the first and second costal cartilages was greater on the left than on the right side, is probably due to the position of the ascending aorta and the descending cava nearly behind the right cartilages. The sounds of the heart, especially the second sound, as will be seen from section 10, are in the majority of cases heard more distinctly under the right than the left clavicle, the probable reason being that the aortic valves, which are the main element in the production of the second sound, are nearer the right than the left clavicle.

With reference to the diagnosis of disease at the apex of the lung, it is often noted that an inflammatory or tuberculous deposit there causes a visible flattening in the subclavicular region. It should, however, be generally known that such flattening, unassociated with other physical signs,

often results from an entirely different cause. In most cases of lateral curvature of the spine the two sides of the chest become unsymmetrical—one side of the chest will commonly be more rounded than the other at the back, while the relative rotundity and flattening are reversed in front. And a slight flattening thus resulting in one infra-clavicular region might, if not rightly interpreted, be supposed to result from a deposit in the apex of the lung. If such a deposit were the cause of the flattening, there would be diminished resonance on percussion over the flattened region. On the other hand, it will usually be observed that when, in the absence of lung disease, the two sides of the chest are unsymmetrical, the flattened side is slightly the more resonant of the two; the explanation being that a nearly straight rib when percussed yields more to the blow, and gives out, therefore, a louder sound than a more arched rib, over which the force of the blow becomes more equally diffused.

A consideration of the facts above recorded may sometimes have the good result of preventing the unfair and uncalled-for rejection of candidates for life insurance.

CHAPTER XXXIII.

A LECTURE ON THE MORBID ANATOMY AND PATHOLOGY OF ACUTE ENDOCARDITIS.[1]

Roughening of the Endocardium—Fibrinous Deposits—Rarely Ulceration—Occasional Rupture of Tendinous Cords—Changes Chiefly on Left Side of Heart—Origin of the Fibrinous Vegetations—Experiments of Sir John Simon, Dr. Mackenzie, and Sir Joseph Lister—Double Festoon on Aortic Valves—Mechanical Wound of Valve covered by Fibrine—Sources of Embolism.

I PROPOSE now to describe and explain to you the anatomical characters of acute endocarditis.

The lining membrane of the heart, when inflamed, becomes red, either uniformly or in patches. The surface, deprived of its natural polish, feels more or less rough. The membrane becomes opaque, thick, and soft, and can be stripped or scraped off in patches; its tissue sometimes cracks, and then fibrinous coagula form on the margins of the resulting fissures.

Fibrinous concretions or wart-like vegetations form on the free surface of the membrane, sometimes in patches, here and there, in the interior of the auricle or ventricle; more commonly on the mitral or aortic valves; much less frequently on the tricuspid valves; with extreme rarity, if ever, on the pulmonary valves. These fibrinous vegetations are often scattered over the surface of the valves, both mitral and aortic; but they are commonly most abundant near the *margins* of the valves, forming bead-like fringes on the margins of the mitral valve, and often, as Sir Thomas Watson particularly points out, a double festoon on each semilunar valve, the fibrine being collected most abundantly at the line of junction between the thinner and thicker portions of the valve.

[1] *British Medical Journal*, March 5, 1870.

It should be observed that the vegetations are usually confined to the *under* or cardiac aspect of the aortic valves, and that they rarely occur on the upper or arterial surface. I will presently give you what seems to be the explanation of this fact.

Sometimes, though rarely, rheumatic inflammation leads to *ulceration* and *destruction* of one or more of the semilunar valves. The ulcerative process may extend into the contiguous muscular tissue, and it may even go so far as to perforate the septum of the heart. Sir Thomas Watson relates one case in which this occurred, and another in which an abscess as large as a hazel-nut had formed in the septum immediately opposite the disorganised valve. The hearts of these two patients are on the table. Both patients were young women—one aged 21, the other 22. Both were admitted for a first attack of rheumatic fever. Both had acute pleurisy, and endocarditis, without pericarditis. One died in three weeks; the other, within a month after admission.

Sometimes large and firm coagula are found in the cavity of the auricle or ventricle.

One of the accidents of endocarditis is the rupture of one or more tendinous cords of the mitral valve, rendered brittle by the inflammatory process. The broken ends of the cords then become covered by warty vegetations.

Some interesting questions relating to the morbid anatomy of endocarditis we will consider now, before passing on to the symptoms.

1. Why are the morbid changes so often limited to the *left side* of the heart? To this question the reply is, that arterial blood probably contains the morbid element of acute rheumatism, which is the most frequent cause of endocarditis, more abundantly than does the venous blood; and the morbid arterial blood, by its contact with the endocardium of the left cavities, acts as an irritant, and thus excites inflammation. An experiment of Dr. Richardson's affords support to this explanation. He injected a solution of lactic acid into the peritoneum of a dog. The result was endocarditis, with fibrous deposit on the *tricuspid valve*. The acid absorbed by the veins first came into contact with the right side of the heart, and

there excited disease; and a bellows sound was heard on auscultation over the heart.

Acute rheumatism, although the most frequent, is not the sole cause of endocarditis. Amongst other exciting causes of the disease may be mentioned uræmia the result of acute or chronic renal disease, pyæmia, erysipelas, scarlet fever. If in all cases of scarlet fever the heart were examined as systematically as it is during the progress of rheumatic fever, the occurrence of endocarditis would be found to be more frequently associated with that disease than is now generally supposed.

2. What is the origin of the fibrinous vegetations? Why are they so often found on the valves alone? and why especially on the margins of the valves? Some pathologists, and amongst others Sir John Simon,[1] deny that these vegetations are of inflammatory origin. His theory is, that they are fibrinous precipitations from an overcharged solution of fibrine; the valves encrusting themselves with fibrine, just as a stick in certain streams coats itself with a calcareous envelope. And he explains the preference of these vegetations for the left side of the heart by the fact that arterial blood is more prone than venous blood to precipitate its fibrine, either from containing more of it, or from containing it in some more separable form. Sir John Simon supports his theory by the results of experiment. By means of a very fine needle, he passed a thread transversely through the artery and vein of a living dog, leaving it there, so that it might cut the stream. The thread was allowed to remain during a period of from twelve to twenty-four hours. The vessels experimented on were the femorals, the carotid and jugular, the aorta and cava, in different animals. The result was, that the thread, where it traversed the artery, presented a considerable vegetation on its surface, sometimes as large as a grain of wheat, always of pyramidal shape—its base attached to the string; its apex in the direction of the blood-current. Mr. Simon remarks upon this: 'In the artery, one might say that the thread whipped the blood, just as one whips blood in a basin to get the fibrine out of it; but with this trifling difference, that, instead of the

[1] *Lectures on Pathology*, p. 55.

rod beating the fluid, the fluid ran over the rod, and precipitated its fibrine there.' In the vein, the thread seemed to operate only obstructively, never coating itself with fibrine, but sometimes delaying or stopping the circulation with a voluminous black clot, chiefly collected on that side of the thread most remote from the heart.

Sir John Simon's theory, supported as it is by his ingenious experiments, contains a very important element of truth; but it is not the whole truth with regard to the pathology of endocarditis. Without doubt he is right in his opinion that the fibrinous vegetations are chiefly, if not entirely, a deposit from the blood, and not solely or chiefly an exudation from the inflamed lining membrane of the heart. Admitting with him that acute rheumatism is a disease in which there is an excess of fibrine in the blood; in which, too, 'almost certainly there are other conditions besides *quantity*, making the fibrine easy of precipitation,'[1] there is conclusive evidence that *the immediate cause of the fibrinous deposit, which constitutes the vegetations of endocarditis, is the altered physical condition of the lining membrane, which results from its being inflamed.*

There are facts and experiments which afford much support to this proposition. The fibrine of the blood has a tendency to coagulate on any parts of the interior of the vascular system which have become roughened or otherwise structurally changed. Thus, in the interior of an aneurysm, the fine laminated coagula form layer after layer. In one series of experiments, designed by the late Dr. F. W. Mackenzie to illustrate the pathology of phlegmasia dolens, he first excluded the blood from the jugular vein of a living dog; then he irritated the lining membrane by the application of a solution of nitrate of silver. When the blood was readmitted, it speedily coagulated throughout the entire tract of the vein so treated.[2] This affords a striking illustration of the influence exerted by inflammation of the lining membrane of the vascular system in causing coagulation of the blood.

In Mr. (now Sir Joseph) Lister's Croonian Lecture on the

[1] P. 57.

[2] *Medico-Chir. Trans.*, vol. xxxvi. p. 201.

Coagulation of the Blood he adduces facts and arguments in opposition to the theory that ammonia is the essential cause of the blood's fluidity, and shows that the contact of a foreign body will determine the coagulation of the blood when the escape of ammonia would have been impossible. Thus blood coagulates on a coil of wire inserted into the carotid of a horse; and it coagulates in capillary tubes immersed in ammonia. He also found that blood coagulated on parts of vessels which had been inflamed by contact with ammonia.

The explanation of the fibrinous vegetations, therefore, appears to be this. The endocardium is inflamed and thickened, and its surface consequently roughened by exudation. Upon the surface so roughened, the fibrine becomes deposited, and agglutinates in wart-like masses. The valves are a favourite seat of these fibrinous deposits from the blood, because their streamward surfaces and their margins are continually exposed to the friction of the blood-current; so that if the whole interior of the ventricle and the surfaces of the valves were uniformly inflamed and roughened by exudation, the vegetations would be more abundant over the valves, on account of the mechanical facilities which there exist for the coagulation and deposit of the fibrine.

If, as I have before suggested, the contact of the morbid blood with the endocardium is the exciting cause of the inflammation, it is obvious that the orifices of the heart and the valves, in proportion as they are more exposed to this morbific influence than the general surface of the cavities of the heart, are, in the same degree, more liable to be the seats of inflammation.

Sir Thomas Watson explains the double festoon of fibrine on each semilunar valve by supposing that the exuded lymph is mechanically pressed away from the thinner portions of each valve, where the two surfaces come into contact when the valves are closed; and consequently the lymph is 'heaped up in a ridge along the boundary lines of contact, just as a thin layer of butter on a board would be displaced and heaped up in a little curvilinear ridge by the pressure of one's thumb.' It is probable that a deposit on the under or cardiac aspect of the thinner margins of the valves might be displaced by the

pressure of the opposed surfaces; but this theory does not explain the absence of fibrinous deposits on the upper or arterial aspect of the valves. I believe that the distribution of the fibrinous vegetations on the semilunar valves is explained by the fact that the thicker, fibrous, and more rigid portion of each valve has its margin and its cardiac surface projected against the onward current of blood, and thus receives a more abundant deposit of fibrine.

My explanation of the fibrinous vegetations is essentially the same as that given by Rokitansky,[1] who says that, in the great majority of cases, the vegetations are only in part to be regarded as inflammatory products. The lowest layer alone can be considered as an exudation; whilst the greater number have been formed in a secondary manner by a deposit of fibrine from the blood.

As fibrine is thus deposited upon surfaces roughened and structurally changed by inflammation, so in like manner fibrinous vegetations are apt to occur on the margins of any fissure of the endocardium, on the torn extremities of a papillary tendon, on the margin of an acute aneurysm of the heart, on the inner wall of a chronic aneurysm of the heart, and, beyond the heart, on rough and uneven spots on the inner surface of the vessels. In each of these cases, the deposit of fibrine is determined by the blood coming into contact with an abnormal surface.

Sir Thomas Watson mentions a curious fact which bears upon this subject. In one of Dr. Hope's experiments, performed with the view of ascertaining the cause of the second sound of the heart, the aortic valves of an ass were held back by a wire inserted into the vessel. The animal had previously been rendered insensible by a narcotic, and the circulation was kept up by artificial respiration. Upon the final cessation of the heart's motions, the organ was removed from the body, and examined; when the valve that had been mechanically injured was found studded with wart-like masses of fibrine. The brief duration of this experiment precludes the possibility of an inflammatory exudation. It is manifest that the mechanical injury of the surface of the valve had the

[1] *Pathological Anatomy*, vol. iv. p. 222.

same influence in causing a deposit of fibrine, as the inflamed membrane has in the case of endocarditis.

The facts to which I have referred indicate with sufficient clearness the manner in which warty vegetations are formed during the progress of acute endocarditis. The lining membrane of the heart is physically changed by the inflammatory process; and, as a result of this, fibrine is deposited upon the altered surface, as it is deposited upon a foreign body, such as a thread or a wire, introduced within the vessels; and the fibrine is deposited most abundantly on those parts of the interior of the heart which, being inflamed, are also most exposed to the friction of the stream of blood, especially the margins of the valves and their streamward surfaces.

A knowledge of the fact that these wart-like masses of fibrine are mechanical deposits from the blood, having no organic union with each other or with the tissues beneath, renders intelligible the readiness with which the fibrinous concretions become detached from the valves, and carried away with the stream of blood, thus giving rise to the various phenomena of embolism.

CHAPTER XXXIV.

A LECTURE ON VALVULAR DISEASE OF THE HEART.[1]

SECTION I.

The Anatomical Results of Chronic Valvular Disease upon the Walls and Cavities of the Heart—The Physical Signs and Diagnosis of Disease affecting the different Valves—Valvular Disease without Murmur—Bellows Sound without Organic Disease—Inorganic Murmurs.

In my lecture to-day, I propose to describe to you first the effect of chronic valvular disease upon the walls and cavities of the heart—in other words, the anatomical results of valvular disease; we shall then pass on to the consideration of the physical signs and diagnosis of each form of valvular disease.

The effect of valvular disease upon the walls and cavities of the heart depends greatly upon the situation and the character of the disease. An almost constant result of all forms of valvular disease is to impede the onward movement of the blood; and this result occurs whether the valvular disease be what is called obstructive or regurgitant; whether it be such as to cause narrowing of one of the orifices, and so a direct impediment to the onward passage of blood; or whether the valve, being rendered incompetent to close its orifice, permit a reflux or regurgitation of blood. The tendency of valvular disease is to cause dilatation of those cavities which lie behind the seat of disease. Disease of the aortic valves tends to cause dilatation of the left ventricle; and this dilating effect upon the ventricle is much greater in cases of regurgitant disease at the aortic orifice than when the disease is simply obstructive. The influence of incompetence of the aortic valves in causing dilatation of the left

[1] *British Medical Journal*, Jan. 13, 1872.

ventricle is intelligible if you bear in mind that in these cases, during the diastole of the ventricle—that is, while the muscular walls are relaxed, the cavity becomes rapidly filled and distended, partly by the onward stream of blood through the mitral orifice, but chiefly by a reflux current forcibly driven through the unclosed aortic orifice by the elastic resiliency of the arterial walls. This unnatural and forcible distension of the relaxed ventricle tends gradually to dilate its cavity. The dilatation is a destructive process; it tends to weaken the heart's walls, and it may so widen the mitral orifice as gradually to render its valve incompetent to close it. The tendency to dilatation is checked by a compensatory and conservative process of hypertrophy of the muscular walls of the cavity. The muscular fibres, stimulated to increased exertion by the excessive pressure of blood, grow firmer and thicker, and this conservative hypertrophy of the muscular walls tends to check the destructive dilatation of the cavity.

We shall find, when we come to study the symptoms of valvular disease, that for a period which varies in different cases, hypertrophy of the heart's walls may entirely counteract the injurious effect of a narrowed orifice or a defective valve. So that, with the physical signs of valvular disease, life may be prolonged for many years with no indication of disordered circulation or of impaired health. By degrees, however, the result of valvular disease is in most cases to cause structural changes in those cavities, which, in the order of the circulation, lie behind the seat of disease. Thus disease of the aortic orifice tends to dilate the left ventricle, so as sometimes to render the mitral valve incompetent, and then to dilate the auricle. Disease of the mitral valve, whether obstructive or regurgitant, acts directly upon the left auricle, and then retards the pulmonary circulation. Obstruction at either the aortic or the mitral orifice tends gradually to act backwards through the lungs upon the right cavities, and to dilate them: thus the ultimate result of a defective aortic or mitral valve may be to dilate the right ventricle and auricle, and to render the tricuspid valve incompetent.

The general tendency of valvular disease, then, is to cause

hypertrophy with dilatation, combined in various degrees. When with hypertrophy of the muscular tissue the thickness of the walls and the size of the heart's cavities retain their normal relative proportions, the case is said to be one of *simple hypertrophy*. It is spoken of as *eccentric or dilated hypertrophy* when, with thickening of the muscular walls, there is a disproportionate dilatation of the cavity, such as occurs in the left ventricle as the ultimate result of incompetence of the aortic valves. On the other hand, the term *concentric or contracted hypertrophy* is applied to those cases in which thickening of the walls is associated with contraction of the cavity. This may be seen in the left ventricle in some cases of mitral disease. In cases of simple hypertrophy, the form of the heart remains unchanged; but the effect of dilated hypertrophy is to render the heart somewhat globular in form; and in some instances its transverse exceeds its longitudinal diameter. The size and the weight of the heart are also greatly increased. In cases of dilated hypertrophy, the result of aortic valve incompetence and consequent regurgitation, the weight of the heart may be three and even four times the normal weight—the excess of weight being a measure of the increased growth of muscular tissue.

One of the most interesting and important ultimate results of valvular disease is impaired nutrition of the muscular walls of the heart, consequent upon an impeded circulation through their tissue. The tendency of valvular disease of the heart is to lessen the blood-stream in the systemic arteries, and to increase, in a corresponding degree, the fulness of the systemic veins. The circulation through the walls of the heart is interfered with by a defective valve in the same way as the circulation through every other organ which receives its blood from the aortic system of vessels. Thus there is a defective supply of blood through the coronary arteries and an impeded return of blood by the coronary veins. Since every organ and tissue requires for its nutrition a free movement of blood through its capillary vessels, it is manifest that a defective blood-stream in the *afferent* arteries and an obstructed current in the *efferent* veins will equally, although differently, impair the nutrition of an organ. A defective

mitral valve, whether causing a direct impediment to the onward flow of blood into the ventricle, or permitting a reflux from the ventricle into the auricle, tends to diminish the flow through the aortic orifice and the supply to the coronary arteries. Disease at the aortic orifice—more especially such as permits regurgitation from the aorta—has an equal tendency to lessen the supply of blood to the nutrient arteries of the heart. The influence of insufficiency of the aortic valves upon the circulation through the coronary arteries requires a few words of explanation. It has sometimes been asserted that the orifices of the coronary arteries are so covered by the upper margins of the aortic valves during the systole of the ventricles that no blood can enter these vessels until the diastole occurs. But numerous careful observations have shown that in the great majority of the hearts of both man and animals, the orifices of the coronary arteries are clearly above the margins of the valves.[1] While, therefore, it is certain that in most hearts the trunks of the coronary arteries are injected during the ventricular systole, it is probable that this condition is universal, and it has been proved by physiological experiment that the pulse in the coronaries is synchronous with that in the other arteries. It is probable, however, that the flow of blood through the terminal branches occurs mainly during the diastole, for it has been observed that while the exposed heart of a living animal becomes paler during the systole, when the minute vessels are compressed by the contracting muscular walls, it regains its red colour during the diastole. It appears, therefore, that during the diastole of the ventricle, when the aortic valves are closed, the forcible elastic reaction of the aorta drives the blood through the terminal branches of the coronary arteries into the muscular tissue of the heart. Now imperfect closure of the aortic valves must obviously influence the circulation through the coronary arteries; for, in direct proportion to the reflux of blood into the ventricle, the tension of the aorta and the consequent pressure of blood in the coronaries will be lessened. Then, in process of time, the continual over-distension of the aorta by

[1] See Henle's *Handbuch der Anatomie des Menschen*, 1876, vol. iii. part 1, page 87.

the action of the dilated and hypertrophied left ventricle tends to dilate the vessel, and to cause degeneration of its walls, with consequent impairment of their elasticity. Here, then, is another cause of lessened blood-supply through the coronary arteries. Thus the overworked left ventricle, receiving a diminished and a progressively diminishing supply of nutrient blood, begins to degenerate, becomes soft, pale, and fatty; and death occurs from a rapidly increasing failure of the circulation, or perhaps suddenly from syncope.

Thus far we have considered the effect of a diminished supply of *arterial* blood to the walls of the heart. The results of *venous* obstruction are equally striking and important. Sir William Jenner has directed attention to this subject in an interesting paper published in the *Medico-Chirurgical Transactions* (vol. xliii). When the right cavities of the heart are over-distended in consequence of obstruction on the left side of the heart, the coronary veins are gorged, and the muscular tissue of the heart is kept in a state of passive congestion. The result is that, after a time, the walls of the heart become indurated, tough, and leather-like. When examined microscopically, the muscular striæ are generally found to be indistinct. Between the fibres there are innumerable fine granules, some of which, as well as the damaged muscular fibres, have undergone an oily transformation. When the congestion has been extreme and long continued, the coronary veins are often found dilated. Amongst the results of this passive venous engorgement are a considerable accumulation of dropsical serum in the sac of the pericardium, and minute hæmorrhagic spots beneath the serous membrane on the surface of the heart. Less frequently these specks of blood are found beneath the endocardium on the inner surface of the heart. These hæmorrhagic spots are similar to those which occur as a result of the systemic venous engorgement during choleraic collapse.[1]

Physical Signs.—We will next consider the physical signs of chronic valvular disease. These, of course, vary according to the seat and the nature of the valvular defect. [On the following diagram the chief auscultatory signs of the various

[1] See p. 112, &c.

forms of valvular disease and the character of the pulse will be found briefly stated.]

Form of Disease	Morbid Sound: When heard	Morbid Sound: Where best heard	Character of Pulse
Aortic obstruction . .	Systolic.	Second right intercostal space.	Normal or small.
Aortic insufficiency or regurgitation . . .	Diastolic.	Ditto.	Collapsing; locomotive.
Mitral insufficiency or regurgitation . . .	Systolic.	At apex of heart and at back of chest.	Small; irregular.
Mitral Obstruction . .	Presystolic.	At apex of heart, rarely heard at back of chest.	Ditto.
Tricuspid regurgitation	Systolic.	At bottom of sternum.	Normal.
Pulmonary obstruction	Systolic.	Second left intercostal space.	Normal.
,, regurgitation	Diastolic	Ditto.	Normal.

We will first consider the case of *aortic obstruction*, the result of narrowing of the aortic orifice, or rigidity of one or more of the semilunar valves. In these cases there is a systolic murmur heard most distinctly at the base, over the second intercostal space, close to the right edge of the sternum. The sound, when loud, may be heard along the arch of the aorta, at the top of the sternum, and again over the back, at the left vertebral groove opposite the second, third, and fourth dorsal vertebræ, but rapidly ceasing downwards. The heart's impulse is increased through hypertrophy of the ventricle. The pulse may be normal in volume, force, and frequency, or small and feeble, when great obstruction is associated with a feeble ventricle.

Aortic insufficiency or regurgitation is indicated by very striking physical signs. The most constant is a diastolic murmur at the base, taking the place of the normal second sound. This murmur is often loud, having a cooing or a harsh sawing character. It is produced by the passage of a reflux current of blood over the free margins of the incompetent valves. In a large proportion of cases, there is some obstruction at the orifice as well as incompetence of the valves. In these cases there is a systolic combined with a diastolic murmur at the base; but, although both sounds originate in the same orifice, the systolic murmur, passing onwards with

the blood, will be most distinctly heard at a point somewhat higher than the point of greatest intensity of the diastolic sound, which appears, as it were, to be carried downwards with the regurgitating blood-stream. The sound is carried by the current of blood, just as the sound of a bell is conveyed by the wind, so as to be heard louder and farther in the direction of the wind than in the opposite direction.

The left ventricle being much dilated and hypertrophied, a strong heaving impulse may be felt and seen over a large space. The extent of cardiac percussion-dulness is increased, and the apex beats below and to the left of its normal position, in the sixth or even in the seventh intercostal space.

The *pulse* is quite characteristic, and is called a *collapsing*, *jerking*, or a *locomotive* pulse. The cavity of the left ventricle, as we have already seen, becomes much dilated, while the walls are hypertrophied. The large ventricle receives at each diastole not only the onward stream of blood from the auricle, but also a reflux current from the aorta. The large cavity, thus distended, empties itself forcibly into the aorta; the result is that, for a moment, the whole arterial system is distended, and the pulse feels full; then instantly, with the diastole of the ventricle, the regurgitant current lessens the tension of the arteries, and the pulse sinks away beneath the finger. The pulse, therefore, is well described as *collapsing*, or *jerking*. The jerking character of the radial pulse is intensified by raising the arm into a vertical position. The explanation of the term *locomotive*, as applied to this kind of pulse, is simply this: the superficial arteries, being alternately distended and elongated by the injecting force of the ventricle, and then rapidly contracting during the diastole, may plainly be seen to move beneath the skin. This is especially remarkable when a bend in an artery has its curve increased during the distended and elongated state of the vessel, returning to its former position when the distension ceases. The throbbing of the arteries in cases of aortic incompetence is often distinctly seen by the aid of the ophthalmoscope in the arteries at the fundus of the eye. Another peculiarity of the pulse is that, as felt at the wrist, it is, in most cases, notably behind the systole of the ventricle. The probable explanation of this

retardation of the pulse is to be found in the fact, that at the commencement of the systole the artery wants the usual tension and fulness, in consequence of the previous reflux of blood into the ventricle; the pulse-wave, therefore, is propagated more slowly than normal.

Mitral insufficiency or regurgitation is indicated by a systolic murmur heard most distinctly at the apex of the heart. The sound may be heard at the back, near the angle of the left scapula, and somewhat lower than the point at which a systolic aortic murmur is best heard. A murmur in the mitral orifice is sometimes heard more distinctly over the back than at the apex of the heart in front. Still more rarely it may be heard at the back when it is quite inaudible in front. In most of these cases, owing to the distension of the pulmonary artery, on listening over the second and third left costal cartilages, the pulmonary valves may be heard to close with an intensified sound; while the sound of the aortic valves to the right of the sternum is comparatively feeble, owing to the diminished tension of the aorta. The second sound is said to be *accentuated* over the pulmonary valves.

The *pulse* is small in proportion to the amount of regurgitation through the mitral orifice, and the consequent deficiency of blood in the systemic arteries. Not unfrequently, too, the pulse is irregular and intermitting.

Mitral obstruction consequent upon narrowing of the orifice, has the same effect upon the pulse and upon the relative intensity of sound produced by closure of the pulmonary and aortic valves, as regurgitant disease at the mitral orifice. The diagnostic physical sign of mitral obstruction is a *presystolic* murmur heard best at the apex. The murmur does not take the place of the normal second sound, as in the cases of aortic regurgitation. It occurs later, and is, in fact, synchronous with the active contraction of the auricle which immediately precedes the systole of the ventricle. The murmur continues while the blood is being driven by the systole of the auricle through the narrowed mitral orifice; and it ceases suddenly when the influx of blood into the ventricle is stopped by the ventricular systole. With reference to the auricle, then, the sound has been well designated by Dr.

Gairdner *auriculo-systolic*. As regards the ventricle, it is *presystolic*. The sound is usually of a soft character, immediately preceding the systole of the ventricle, and closing abruptly with the systole. The murmur is accompanied by a thrill or fremitus, felt by the hand pressed firmly over the apex. The first sound which follows the murmur is unusually short, in consequence of the rapid contraction of the partially filled left ventricle. Without doubt, mitral obstruction is more common than this direct mitral murmur, although the murmur itself is by no means rare. There are two facts which help to explain the comparative infrequency of a direct mitral murmur —first, the blood, propelled only by the auricle, enters the ventricle in a somewhat feeble stream; and, second, during the diastole, the apex of the heart not being in contact with the wall of the chest, a feeble sound originating in the mitral orifice is less likely to reach the ear.

Some time since I had under my care in the hospital a case of mitral stenosis with an interesting modification of the presystolic murmur. Eliza M——, æt. 36, had suffered from palpitation and shortness of breath since an attack of rheumatic fever sixteen years before. When admitted, there was some dulness on percussion, with mucous râles over the bases of both lungs. The pulse was small and feeble. The extent of cardiac dulness was normal; the impulse moderately strong and sharp, and *preceded* by a distinct thrill. On auscultation over the apex, a soft blowing sound was heard, commencing immediately after the systole, therefore diastolic. This sound was continuous until, immediately before the succeeding systole, it was intensified, and then ceased with the sharp systolic impulse, and the short, but normal, first sound. There was, therefore, a soft *diastolic* murmur, continued into a louder *presystolic*, and ceasing with the ventricular systole. The second sound was normal, but accentuated over the pulmonary valves to the left of the sternum.

The phenomena of this case confirm the usually accepted explanation of the presystolic mitral murmur—namely, that it is a result of constriction of the mitral orifice (mitral stenosis), that it is synchronous with the contraction of the auricle (auriculo-systolic), and that the forcible driving of

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the blood through the constricted orifice by the auricular contraction is the main cause of the presystolic murmur and thrill. It seems probable that the soft diastolic murmur which commenced immediately after the systole, was caused by the blood flowing into the ventricle, under the influence of the elastic resiliency of the distended pulmonary veins; while the presystolic intensification of the sound was caused by the increased rapidity of the current resulting from the auricular contraction. In most cases of mitral stenosis no murmur is audible during the ventricular diastole, until the force and speed of the blood-current are increased by the auricular systole; so that there is a silent interval between the first sound and the subsequent presystolic murmur; while, in the case which I have here briefly noted, a soft diastolic murmur, commencing immediately after the first sound, was continuous with the presystolic intensification.

Although the cases in which there are both obstruction and regurgitation at the mitral orifice are sufficiently common, *a double mitral murmur* is comparatively rare. In a few cases, I have satisfied myself that such a murmur existed. The heart from one of these cases is on the table.[1] It will be seen that, while there is extensive disease of the mitral valve, with a large fibrinous coagulum on its auricular surface, there are some small fibrinous fringes on the aortic valves, which would in no degree impair their efficiency or impede the blood-stream. The double sound heard during life must have originated at the mitral valve.

Hitherto we have been discussing the physical signs of disease affecting the valves on the *left* side of the heart—the aortic and the mitral valves. In the vast majority of cases, valvular disease occurs on the left side of the heart, and on the left side only. Of the two sets of valves on the right side, the tricuspid is much more frequently defective than the pulmonary. The curtains of the tricuspid valve are sometimes thickened by rheumatic inflammation, or by atheromatous degeneration. More frequently, however, the valve is rendered incompetent by dilatation of the auriculo-ventricular orifice; and this dilatation is usually a result either of valvu-

[1] Case of Maria Johnson, K. C. H., ix. p. 89.

lar disease and obstruction on the left side of the heart, or of extensive emphysema of the lungs. Incompetence of the tricuspid valve may exist without audible sound to indicate its presence. We infer the existence of such incompetence when, at each contraction of the ventricle, a reflux wave is visible in the superficial jugular veins. The auscultatory sign of *tricuspid incompetence and regurgitation* is a *systolic* murmur heard most distinctly at *the bottom of the sternum*, and becoming less distinct or inaudible at the apex of the heart. I have told you that dilatation of the tricuspid orifice, and consequent insufficiency of the valve, is a not uncommon result of valvular disease on the left side of the heart; accordingly, we sometimes find that, by careful auscultation, we can recognise the physical signs of aortic or mitral disease, together with those of tricuspid regurgitation, in the same patient.

Contraction of the tricuspid orifice is an occasional congenital defect, and sometimes a result of disease in after-life; but it is infinitely less common than dilatation; and I am not aware that I have ever heard a *diastolic*, or *presystolic*, or *direct tricuspid murmur*, the result of narrowing of the right auriculo-ventricular orifice.

A remarkable case of both tricuspid and mitral stenosis is recorded by Dr. Balfour.[1]

A *murmur* originating in the *pulmonary orifice* is the rarest of all cardiac sounds. The most frequent causes of such a murmur are—first, congenital malformation and narrowing of the pulmonary artery; second, aneurysm at the origin of the aorta, pressing upon the pulmonary artery, and displacing its valves. Upon the table there is a preparation showing a small aneurysm of the aorta which has displaced the pulmonary valves, so as evidently to have rendered them incompetent to close the artery. The late Dr. Hope, in his book on *Diseases of the Heart*, has given a drawing of a specimen exactly like the one on the table. In Dr. Hope's case there was a loud superficial diastolic murmur heard most distinctly to the left of the sternum, over the pulmonary artery. This sound was not heard in the carotids or over the

[1] *Clinical Lectures on Diseases of the Heart*, p. 112.

back. The pulse was normal. There was not the collapsing pulse which is so characteristic of aortic regurgitation.

In some recorded cases of malformation of the pulmonary artery, there has been a loud double murmur (systolic and diastolic), most distinctly heard to the left of the sternum, not conducted along the aortic vessels, and not associated with the collapsing pulse of aortic regurgitation.

Now, having discussed at sufficient length the physical signs of valvular disease, let me impress upon you the fact that serious *valvular disease may exist without abnormal sound over the heart.* In cases of old disease of the mitral valve with extreme narrowing of the orifice, it sometimes happens that, although the symptoms of impeded circulation are very manifest, there is no audible abnormal murmur. Excessive narrowing of the orifice, with thickening of its rigid margins, and the very rapid action of the heart which usually accompanies great mitral contraction, are all unfavourable for the production of an audible murmur. On the table are two specimens of extreme narrowing of the mitral orifice. Both cases occurred in my hospital practice; and in neither case was a murmur heard, although carefully sought for.

When, with incompetence of the aortic valves, the margins of the valves become covered with fibrinous deposit, a regurgitant current may exist without audible murmur. Fibrinous warty vegetations are sometimes found on the surface of the mitral and aortic valves when no abnormal sound has been heard during life. Mitral regurgitation may so lessen the force and volume of the blood in the aorta as to prevent the generation of sound by an obstructive disease at the aortic orifice. Mitral regurgitation may also lessen or quite destroy the characteristic collapsing pulse of aortic regurgitation.[1]

To illustrate the diagnosis of valvular disease in the absence of auscultatory signs, I may refer briefly to the case of Jane H——, aged 42, who was admitted into Twining ward on January 25, 1865,[2] with a very remarkable history. She had twice had rheumatic fever; the second time five years

[1] See the following clinical lecture *On the Diagnosis of Coexisting Aortic and Mitral Incompetence.*

[2] *Hospital Case Book*, vol. xxii. p. 277.

before her admission. She had been in fairly good health until eleven weeks before, when, after a hard day's work, while running fast along Fleet Street, and being much out of breath, she was stopped by a sudden intense pain over the heart, with palpitation. She was able to walk slowly home, but the palpitation and dyspnœa continued, and were soon followed by other symptoms of impeded circulation; amongst others by hæmoptysis and dropsy. On her admission, in addition to the dropsy, there were signs of pulmonary engorgement and great lividity of the surface. The heart's action was rapid and irregular; the pulse 165, but so small and feeble that it could scarcely be counted. On auscultation I thought that I heard an occasional systolic murmur at the apex, but I was doubtful, and no one else could hear it. In the absence of auscultatory signs, there was evidence of great obstruction at the left side of the heart; and, connecting this with the sudden onset of the symptoms eleven weeks before, and the extremely small pulse, I expressed my opinion that one or more of the tendinous cords of the mitral valve had been ruptured while she was running. She died five days after her admission, and my diagnosis was found to be correct. One of the tendinous cords had been broken near its attachment to the middle of the anterior flap of the valve. And that the tear had been recent was proved by the presence of a small bead of fibrine on the surface of each broken end.[1] The curtains of the valve and all the tendinous cords were opaque and thickened, and the mitral orifice was somewhat narrowed. The poor woman had been hard at work and free from cardiac symptoms until the occasion before mentioned; and there can, I think, be no question that the breaking of one of her heart-strings was the cause of the distress which began so suddenly, and which continued to the end. No doubt the cord had been rendered abnormally brittle by chronic structural changes. The heart is in the museum of the college.

We have seen that serious organic disease may exist without abnormal sound. I must now tell you that *a murmur may exist without organic disease.* The most common and un-

[1] The explanation of such fibrinous deposits has been before given, Chapter XXXIII.

doubted form of inorganic murmur is a soft systolic blowing at the base of the heart, which may be heard not unfrequently in cases of anæmia. The thin and watery blood passes through the aortic and pulmonary orifices with an audible blowing sound. The sound is more constant and louder when the heart's action is excited and violent; and, indeed, violent action of the heart, whether the result of muscular exertion or of emotional excitement, is not unfrequently attended with a systolic blowing at the base, in persons who are not anæmic. I have frequently observed this in examining nervous candidates for life insurance; and I have found that, after waiting for a short time to allow the emotional excitement to pass away, the heart's action has become quiet, and the blowing sound has ceased.

In children with very flexible chest-walls, firm pressure by the stethoscope placed over the aorta, will sometimes cause a temporary systolic blowing.

When a systolic blowing sound at the base of the heart is the result simply of anæmia, a loud humming sound may often be heard, when the stethoscope is placed over the veins at the root of the neck. This venous murmur has received the name of *bruit du diable*. It is a continuous murmur, with periodical intensification of loudness; the intensified sound corresponding with the more rapid current of blood in the veins during the diastole of the auricle. A deep inspiration also increases the sound by sucking the venous blood more forcibly into the chest. An inorganic cardiac murmur is always systolic, and, according to my experience, always heard at the base of the heart. Some writers have suggested that an inorganic systolic murmur may originate at the mitral orifice. I doubt this. A systolic murmur at the mitral orifice would indicate regurgitation through that orifice; and this must result from insufficiency of the valve. Such a murmur, therefore, would not be an inorganic murmur, though it might be caused by a temporary dilatation of the ventricle. It is possible that roughening of the ventricular surface of the anterior flap of the mitral valve might cause a systolic murmur at the apex. In such a case, and in the absence of regurgitation, the pulse would be normal.

A murmur apparently originating in one of the heart's orifices may result from organic disease *outside the heart.* Thus, in one of my hospital patients, a cancerous growth in the left lung surrounded and compressed the pulmonary artery, and so caused a systolic blowing at the base of the heart.[1]

SECTION II.

A LECTURE ON VALVULAR DISEASE OF THE HEART.[2]

The General or Functional Symptoms of Valvular Disease—Prognosis—Treatment.

We have now to consider *the general or functional symptoms* of valvular disease. And, first, I may remark that disease of one or more valves, as indicated by physical signs, may exist for years, yet so long as the walls of the heart continue to be well nourished, there may be few or even no symptoms of functional disturbance. It has happened to me twice, in examining candidates for life insurance, to discover the unmistakable signs of aortic regurgitation in men of middle age who believed themselves to be in perfect health, and who declared that they had never been ill. Moreover, in one of these cases the medical attendant of the applicant certified that he was sound and in good health. In each of these cases I chanced to hear that death had occurred within a few months after the discovery of the disease, and the consequent rejection of the candidate by the insurance office. It is a matter of common observation that, when valvular disease has been caused by an attack of rheumatic fever, the physical signs may be unmistakably present; yet many years may elapse before any symptoms referable to the heart are complained of. In some rare cases of valvular disease of rheumatic origin, the murmur, in the course of years, finally ceases to be heard, the valve apparently having reverted to the normal condition; but as I told you in my last lecture, the cessation of a murmur may result from other conditions than the restoration of the valve to its structural integrity.

[1] Case John Campbell, aged 41; admitted April 5, died April 11, 1862, *Hospital Case Book*, xiv. p. 71.

[2] *British Medical Journal*, Jan. 20, 1872.

Amongst the earliest and most constant symptoms of valvular disease, when it begins to act injuriously upon the circulation, is an excited or irregular action of the heart on exertion, such as going up-stairs or running or walking fast. With this there is often hurried and difficult breathing, the result of an impeded circulation through the lungs. As the impediment increases, the lungs become congested and œdematous; moist sounds are heard over the lower lobes; and the patient becomes liable to attacks of catarrh and bronchitis.

In the advanced stages, *hæmoptysis* is not an unfrequent symptom, the result of passive engorgement and rupture of minute pulmonary or bronchial vessels.

When we were discussing the subject of *vesicular emphysema* of the lungs I explained to you the manner in which valvular disease of the left side of the heart tends to bring about that condition of the lungs.

In most cases of advanced valvular disease there is a sense of uneasiness, and in some there is actual pain in the region of the heart. The pain varies much in degree. In some cases of aortic regurgitation, with dilatation and weakness of the left ventricle, severe pain comes on in paroxysms, and extends down the left arm, constituting one form of what is called *angina pectoris*. These attacks often result in sudden and fatal syncope.

Most of the symptoms of valvular disease are the direct result of the interrupted circulation through various tissues and organs; this interruption of the circulation involving, as we have seen, a defective supply of arterial blood and an impeded return of venous blood. I have already described the result of this interrupted circulation upon the nutrition of the heart's walls, and I have just now spoken of its effect upon the lungs. The abdominal organs suffer from the same cause. The interrupted circulation through the walls of the *stomach* results in a defective or an abnormal secretion of gastric juice. The consequence is, that the food, not being fully acted upon by its natural solvent, decomposes and ferments. The patient is dyspeptic and flatulent, and the flatulent distension of the stomach increases the dyspnœa and the cardiac distress. A similarly impeded circulation through

the *kidneys* brings with it a scanty secretion of urine (which is sometimes albuminous), a consequent retention of urinary excreta, both solid and liquid, and then dropsical effusion into the areolar tissue and the serous cavities.[1] In cases of cardiac dropsy, the dropsical effusion is always preceded by a scanty secretion of urine, the result of an interrupted circulation through the kidneys. The impeded circulation through the *liver* has amongst its results a scanty secretion of bile, a jaundiced tinge in the eyes and skin, and an effusion of serum into the cavity of the peritoneum.

Extreme engorgement of the portal vessels sometimes results in hæmorrhage into the stomach or intestines, and blood is expelled either by vomiting or by stool.

The liver and the spleen are often visibly and palpably enlarged and indurated, in consequence of the continued passive engorgement of the portal venous system; and after death it happens not unfrequently that yellow fibrinous patches are seen in the liver, spleen, and kidneys, the result of arterial and capillary obstruction by particles of fibrine which have been detached from the valves of the heart.

The functions and even the structure of the *brain* are often seriously affected. There may be vertigo, dimness of sight, headache, confusion of thought, delirium, and even insanity, ending perhaps in paralysis and coma, with *post-mortem* evidence of cerebral softening or hæmorrhage. These symptoms and structural changes may occur as results of the general impediment of the circulation which valvular disease occasions, and quite apart from the accidental plugging of cerebral vessels by fibrinous concretions which have been detached from a diseased valve.

This plugging of cerebral vessels by masses of fibrine from a damaged valve is, however, a frequent cause of brain-disease. The symptoms, which vary according to the size and the situation of the vessels thus plugged, usually come on suddenly at the moment when the circulation is arrested. Sudden hemiplegia from a plug in the middle cerebral artery is not uncommon; and, when the obstruction is on the left side of the brain, aphasia may be associated with hemiplegia.

[1] See *ante*, p. 38.

Chorea is sometimes associated with, and apparently caused by, capillary embolism in some portion of the brain near the corpus striatum; and quite recently (January 1872) we have had in the hospital a woman with the physical signs of an incompetent mitral valve, who, in addition to plugging of the brachial artery by embolism, has had pain in the head, vertigo, mental excitement and illusions, the result, probably, of cerebral embolism.[1]

Prognosis.—The diagnosis of valvular disease is, for a trained ear and finger, comparatively easy, but to accurately estimate the probable effect of the disease upon the duration of life is a much more difficult matter. To tell a man who feels in perfect health that he has serious heart-disease because a murmur is heard over one of his valves, would be as unwise and as mischievous in its effects upon his mental and bodily condition, as it would be to say to another man, equally free from ailments, but whose urine has been found to be albuminous, that he has Bright's disease; which is always understood by the laity to be an incurable degeneration of the kidneys. To discuss fully all the questions involved in the prognosis of valvular disease would require more than one lecture, but I can, in a few sentences, give you some hints which may be of use to you.

There can, I think, be no question that, in the order of gravity, aortic incompetence stands first and aortic obstruction last, amongst the forms of valvular disease. There is some difference of opinion as to the relative gravity of mitral stenosis and mitral incompetence. I certainly have seen many more cases of mitral regurgitation than of mitral obstruction, with prolonged freedom from cardiac distress. My own conviction is that constriction of the mitral orifice is a more formidable condition than mitral incompetence, but much will depend in each case upon the amount of obstruction or of reflux; and this is to be estimated, not by the intensity of the murmur, but by the character of the pulse, which will be small in proportion to the impediment to the onward flow, or to the valvular defect which permits the reflux of blood, and so prevents the filling of the arteries. In cases of aortic

[1] See Chapter XXXVII. on *Thrombosis and Embolism.*

regurgitation, the extent to which the valvular defect has caused dilatation of the ventricle may be determined by the position of the heart's apex, and the area of cardiac dulness and impulse, together with the correlated amount of momentary arterial fulness and throb which precede the subsequent collapse of the pulse.

But the prognosis must not be based solely upon anatomical data. So long as the dilatation of the ventricular cavity is compensated by hypertrophy of the muscular walls, the circulation is carried on with apparently perfect regularity, in some cases even to old age. Some years since, one of our museum porters was known for a long time to have had a double aortic murmur, but he went on doing his work beyond the age of 70 before he finally broke down. Knowing the crippled condition of his heart, I have often, with fear and trembling, watched his apparently easy ascent, with a tray full of heavy preparations, from the anatomical theatre below, to the museum far above. You will understand that such an exertion, although possible for a man with an incompetent aortic valve, is not free from danger, and is calculated to increase the destructive dilatation of the ventricle.

One of my patients was 76 years of age when I first saw him in March 1878. He had then a double aortic murmur, with the heart's apex in the sixth interspace, and also a characteristic collapsing pulse. The valvular defect was first discovered three years before I saw him, and it had probably existed long before that. He lived five years after his first visit to me, and until within a few months of his death he was actively engaged as chairman of several public companies.

It is but rarely that a patient with serious valvular disease lives to so advanced an age as this. In the great majority of cases life is shortened by some of the results of an increasing impediment to the circulation through various organs. Amongst the most frequent indications that the end is approaching are the physical signs of congestion of the lungs, with the resulting œdema, bronchitis, hæmoptysis, pulmonary apoplexy and hydrothorax; congestion of the liver, with jaundice and hæmorrhage from the stomach and bowels; congestion of the kidneys,

with the resulting dropsy and its consequences; more especially inflammation and sloughing of the skin and cellular tissue; and, lastly, impeded circulation through the brain, with the associated nervous symptoms. Sudden death by syncope is an occasional result of aortic regurgitation, but it is rare in connection with any other form of valvular disease.

A preparation on the table from one of my hospital patients illustrates one cause of sudden death from valvular disease. The mitral valve is much thickened and its orifice narrowed; a decolourised fibrinous coagulum has formed in the dilated left auricle. This had become detached from the wall of the auricle and blocked the mitral orifice, thus causing an instantaneous arrest of the circulation.

In the *treatment* of valvular disease, the first lesson to be learnt and acted upon is, that no treatment can have the slightest beneficial influence upon a damaged valve. Mischief has often been done by a lowering and a so-called antiphlogistic treatment after the discovery of a bellows sound, which has been supposed to indicate recent inflammatory disease, but which in reality has been the result of a long past endocarditis or of chronic degeneration of a valve. The main object of treatment is to prevent the mischievous reaction of a damaged valve upon the muscular walls of the heart; in other words, to promote hypertrophy of the muscular tissue, as the most effectual means of preventing dilatation of the cavities. For it may safely be assumed, as a general rule, that the duration of life, in these cases, depends upon the degree in which hypertrophy of the muscular walls enables the heart to overcome the impediment to the circulation which results from a constricted orifice or an incompetent valve.

The diet should be nutritious, including a good proportion of animal food. Fatigue and all depressing influences should be as much as possible avoided, as well as everything that tends to excite and hurry the circulation, such as mental or emotional excitement, running, or quick walking, and violent muscular exertion of all kinds.

Tonics are often useful: quinine, nux vomica, and mineral acids to help the appetite and digestion; iron, to counteract anæmia.

It has sometimes been said that one object of treatment is to prevent excessive hypertrophy of the heart; but it is doubtful whether this hypertrophy is ever in excess of what is required: doubtful, therefore, whether attempts should ever be made to lessen it. It is true that relief from pain, palpitation, and dyspnœa is sometimes afforded by a moderate venesection or by local bleeding, by purging, and by temporary low diet. But the probable explanation of this is, that by these means the arterial tension, and with that the work of the heart, is lessened, and not that the nutrition and the strength of the heart's walls are beneficially diminished. Small doses of digitalis are often very useful in checking rapid and irregular action of the heart; and a combination of tincture of digitalis with tincture of the perchloride of iron is an efficacious sedative tonic and diuretic. The action of digitalis on the heart must always be carefully watched. It sometimes causes a distressing fluttering and depression, when, of course it must be immediately discontinued. Digitalis is more useful in cases of mitral incompetence and stenosis than in cases of aortic incompetence. In the latter class of cases, by prolonging the diastole, it may increase the regurgitation and the resulting risk of syncope. It is especially useful as a cardiac tonic when the muscular tissue of the heart is enfeebled by granular and fatty degeneration.

Of late years two vegetable drugs have been much used as substitutes for digitalis—namely, *convallaria* and *caffeine*. The convallaria, in doses of from ten to thirty minims of the tincture, is said to act as a cardiac tonic and diuretic, and to cause none of the deleterious effects which are sometimes caused by digitalis. Caffeine, as a cardiac tonic, appears in some respects to resemble digitalis. The citrate may be given in doses of from one to five grains. Extract of aconite appears to possess the power of relieving painful sensations and disquietude about the heart. One-eighth of a grain of the alcoholic extract may be given twice or thrice daily. The effect must be carefully watched. In some cases pain and distress are much relieved by the cautious use of morphine, either by hypodermic injection or by the stomach. A belladonna plaster worn over the heart often proves an efficient local anodyne. Belladonna,

remember, cannot be applied to an abraded surface, without risk of absorption and alarming symptoms of poisoning.

The state of the circulation through the lungs and other internal organs must be watched and regulated. Exposure to cold and wet should be carefully avoided. The result of such exposure might be an attack of pulmonary congestion and bronchitis. The occurrence of such symptoms would be best met by the prompt application of the treatment for catarrh and bronchitis which I described fully in Chapter XXXVIII. Congestion of the kidney, with a scanty secretion of urine, may be treated by the warm bath, or by the hot-air bath, dry cupping on the loins, mustard and linseed poultices, and an occasional purgative.

Diuretics are useful in cases of valvular disease, not only to lessen, but also to prevent, dropsy. Various diuretics may be tried in succession and in combination—broom, digitalis, juniper, squill, acetate of potassium, spirit of nitrous æther, compound tincture of iodine. I have often seen the happiest results from the following combination: ℞. Succi scoparii, ʒj.; spirit. ætheris nit., ʒss.; tinct. digitalis, ♏x.; potassii acetatis, gr. xx.; aquæ ad ℥j. M. Ter die sum. A pleasant and often efficient diuretic is the so-called 'imperial drink,' made with cream of tartar, lemon-juice and peel, to which a small quantity of gin may be added. Another useful diuretic is a combination of powdered squill, digitalis, and calomel, a grain of each in a pill, repeated night and morning until six doses have been taken. The action of diuretics is often promoted by an occasional hydragogue, as, for example, one-sixth or one-fourth of a grain of elaterium, or half a drachm of compound jalap powder or ten grains of compound gamboge pill. In some of these cases, as also in cases of renal dropsy, the best means of promoting a free secretion of urine, and so removing the dropsy, consist in keeping the patient for a time exclusively upon a diet of cold or tepid milk, of which six or eight pints may be taken in twenty-four hours. If there be a difficulty in digesting the cream, the milk should be skimmed. The diuretic action of the milk may be aided by giving twenty or thirty grains of acetate of potassium three times a day.

When other means fail to prevent the increase of dropsy

the anasarcous fluid may be allowed to escape through incisions or punctures in the legs. The drain of liquid usually affords great temporary relief, and is often followed by a copious secretion of urine. It is not to be denied—and the patient and his friends should be informed—that incisions or punctures are sometimes followed by inflammation and sloughing of the integuments; but then, on the other hand, distension of the skin by an excessive dropsical accumulation is not unfrequently attended with the same unpleasant results. In my experience, erysipelatous inflammation and sloughing have resulted more frequently from over distension of the integuments than from punctures made to remove distension. On the whole, then, puncturing anasarcous legs is an operation which, when done with proper precautions, is so advantageous to the patient, that we dare not shrink from the responsibility of advising it. Many instances have come under my observation in which this simple operation has prolonged life in comparative comfort for a very considerable period.

Messrs. Matthews have made for me a spring sacrifier, like a cupping sacrifier, with one blade. By this instrument the incision is made so rapidly as to be painless.

CHAPTER XXXV.

A CLINICAL LECTURE ON THE DIAGNOSIS OF COEXISTING AORTIC AND MITRAL INCOMPETENCE.[1]

Case of Combined Aortic and Mitral Incompetence—Explanation of the Modified Pulse—Aortic Incompetence without Dilatation of the Ventricle—Mitral and Aortic Incompetence combined without a Diastolic Aortic Murmur—Explanation of the Absence of Murmur.

In my lecture on valvular disease of the heart I have described and explained the diagnostic signs of aortic and mitral incompetency, as these valvular defects occur apart from each other, and I briefly referred to the fact that these signs may be much modified by the coexistence of mitral and aortic incompetence. I propose now to give some further illustrations of this interesting subject, by referring to cases in the hospital.

The two valves may be affected simultaneously, as a result of rheumatic endocarditis, for instance. But very frequently a primary incompetence of the aortic valves leads to a secondary incompetence of the mitral, through the resulting dilatation of the ventricle and of the mitral orifice.

In Craven ward we have one good example of this double valvular lesion.

William C——, æt. 50, was discharged from the army eleven years ago, after fifteen years' service. He has not had rheumatic fever. For the last six months he has suffered from shortness of breath and palpitation, and lately his legs have become œdematous.

The apex of the heart beats in the sixth intercostal space

[1] *The Medical Examiner*, January 6, 1876.

external to the mammary line. The area of cardiac dulness is increased. There is a systolic murmur at the apex, and a double murmur, systolic and diastolic, at the base. Here we have the undoubted physical signs of mitral regurgitation, with aortic obstruction and regurgitation. The pulse is indicative of this double valvular defect. It is distinctly collapsing, but it is only moderately full before it collapses. The collapse is the result of the reflux current through the aortic orifice during the diastole. The only moderate degree of fulness before the collapse is explained by the regurgitation through the defective mitral valve during the systole. The pulse is smaller than it would be if the mitral valve closed and prevented reflux into the auricle; it is larger and fuller than it is in a case of mitral regurgitation, with competent aortic valves and an undilated left ventricle, of which we have just now seen an example in Twining ward. In a case of aortic incompetence with a normal mitral valve, the fulness of the pulse which precedes the collapse bears a direct relation to the dilatation of the ventricle, and the volume of blood which it propels into the aorta. With aortic and mitral incompetence coexistent, the volume of the pulse depends on the relation between the dilatation of the ventricle and the amount of mitral reflux.

[Since this lecture was given, the patient, William C——, has died. There was found thickening and incompetence of the mitral and aortic valves, with dilatation and hypertrophy of the left ventricle.]

In the early stage of aortic incompetence, before the ventricle has become dilated, the pulse has the collapsing character, but the arterial fulness which precedes the collapse is normal. We have an example of this in the case of Arthur S——, in Craven ward.[1] He is 15 years of age, and in June last he was in St. Bartholomew's Hospital with rheumatic fever. He has a diastolic murmur, heard most distinctly over the base of the heart. The apex beats in the normal position, and the area of cardiac dulness is not increased. The pulse is sharp and collapsing, but the fulness which precedes the collapse is not abnormal. Here we have

[1] *Case Book*, vol. li. p. 448.

the physical signs of aortic incompetence which has not as yet resulted in dilatation of the ventricle, and the pulse has much the same character as in the man in the same ward (William C——), who, with a dilated ventricle, the result of aortic incompetence, has also mitral regurgitant disease.

Now I scarcely need impress upon you that these refinements, as some may think them, of cardiac diagnosis have a practical value, especially with reference to prognosis. Aortic valve incompetence and mitral incompetence, existing alone, are nearly equally grave; but the coexistence of both defects is far more formidable than either by itself. There are some cases in which a careful study of the pulse will assist you in the diognosis of this double valvular incompetence, when auscultation alone would be insufficient. We have such a case in Twining ward.

Louisa W——, æt. 22, was admitted November 17. She had rheumatic fever three years ago. Since the end of last year she has suffered from palpitation and shortness of breath, which of late have become very distressing. The apex of the heart is felt in the sixth interspace, about two inches outside the nipple. The area of cardiac dulness is much increased. There is a loud systolic murmur at the apex, and another systolic bruit is heard at the base. The second sound, to the right of the sternum, is less clear than over the pulmonary artery to the left, but no diastolic bellows sound is heard there. The pulse has the mixed character which I have before described. It is only moderately full, but distinctly collapsing. Now in this case, although there is no diastolic mumur, I confidently diagnose aortic incompetence in addition to mitral incompetence; and I base my diagnosis on the evidence of dilatation of the ventricle and the collapsing pulse, with a greater precollapsing volume than could occur, if, with mitral regurgitation, there did not coexist dilatation of the ventricle, consequent on aortic incompetence. The systolic murmur at the base and the dulness of the second sound over the aortic valves are probably explained by thickening of the margins of the aortic valves, and this same thickening may permit a diastolic reflux of blood without an audible diastolic murmur.

It is not very uncommon to meet, as in this case, with unquestionable evidence of aortic regurgitation unaccompanied by a diastolic murmur. I have seen several cases in which the diastolic murmur of aortic regurgitation has, in the course of years, gradually disappeared, while other signs of the valvular defect have continued and increased. The causes of this cessation of a diastolic aortic murmur are different in different cases. In some cases it is the result of thickening of the margins of the semilunar valves. The thinner the margins of the valves, the more sonorously they are made to vibrate by a regurgitant current of blood. The loudest diastolic aortic murmur is that which results from the rupture of a valve by mechanical violence: one segment of a valve being torn, the reflux of blood over the tense edges of the other two segments, causes a murmur which may sometimes be heard several inches away from the patient's chest. I have seen some remarkable instances of this. But when the margins of the valves are thickened by a deposit of fibrin, as may be seen in the specimen from our museum which I here show you, their vibrations are damped, as those of the vocal cords are impeded by a diphtheritic false membrane or a warty growth. The margins of the valve segments being thus thickened and rounded, aortic regurgitation may occur, unassociated with an audible diastolic basic murmur. It is not improbable that this condition of aortic valves exists in the case of the girl Louisa W——.

In other cases the diminished intensity, or entire cessation of a regurgitant aortic murmur, is a result of degeneration and impaired elasticity of the coats of the aorta, resulting from the continued forcible over-distension and consequent injury of that vessel by the dilated and hypertrophied left ventricle. One result of the diminished resiliency of the aorta is a defective blood-supply to the heart, through the coronary arteries. The overworked left ventricle receiving a diminished supply of blood, begins to degenerate, and becomes soft, pale, flabby, and fat. The enfeebled ventricle propels the blood less forcibly into the now comparatively inelastic aorta, and the consequence is that the regurgitant current

through the aortic orifice has not sufficient force and velocity to excite an audible murmur. It is obvious that, when matters have reached this pass, the prognosis is very unfavourable, and death soon occurs from a rapidly increasing failure of the circulation.

CHAPTER XXXVI.

A CLINICAL LECTURE ON TRIPLE PERICARDIAL FRICTION-SOUND, AND ON REDUPLICATION OF THE FIRST SOUND OF THE HEART.[1]

Dr. Salter's Cases of Triple Friction-sound and Presystolic Auricular Friction —Dr. Johnson's Case of Presystolic and Systolic Friction—Explanation of Triple Pericardial Friction Sound—Reduplication of Cardiac First Sound—Dr. Sibson's Theory of Asynchronous Ventricular Contraction—Objections to his Theory—Intimate Muscular Connection between the Ventricles compels their simultaneous Contraction—The first Element of the Double Sound caused by Contraction of the Auricle—Facts and Arguments in support of the Auricular Theory—Comparison of Double First Sound with Auricular Friction and Auriculo-systolic Murmur—Double First Sound with Emphysema of Lungs, and in cases of Dilated Heart without Obstruction of the Circulation—Practical Advantages of Exact Cardiac Diagnosis —M. Potain's independent Adoption of the Auricular Theory—Case of Double First Sound with a greatly Dilated Auricle.

Most of you here will remember that in my lectures on the principles and practice of medicine, I have been in the habit of teaching that the friction-sound of pericarditis is, in most instances, not merely to-and-fro—systolic and diastolic—but that a third sound often intervenes somewhere between the other two. I said 'somewhere,' because until quite recently I did not know at what period of the heart's revolution the third friction-sound occurred. During the last few months I have gradually worked out what I believe to be the true interpretation of the triple pericardial friction-sound, and I purpose now to explain to you the successive steps by which I have arrived at this result.

I got the first hint towards a solution of the problem from a very interesting and suggestive clinical lecture published by the late Dr. Hyde Salter.[2] In that lecture Dr. Salter describes, first, a case of rheumatic pericarditis, in which a friction-sound

[1] *Lancet*, May 13, 1876. [2] *Ib.* July 29, 1871, p. 151.

double over mid-sternum, became triple over the right third intercostal space, close to the sternum; and as this triple character of the friction-sound was most marked when the stethoscope was placed directly over the right auricle, Dr. Salter said: 'I feel no doubt that the third element of the sound, on passing from the surface of the ventricle to that of the auricle, is due to auricular pericardial friction.'

Dr. Salter next relates a case of renal pericarditis, in which a single friction-sound of distinctly presystolic rhythm, was heard over the third costal cartilage, about an inch to the left of the sternum; and, the patient dying a few days afterwards, the left auricle was found covered and roughened with lymph. 'The roughening was confined to the surface of the auricle, and, therefore,' Dr. Salter remarks, 'the friction-sound coincided with the movements of the auricle.'

In a third case, which was also one of uræmic pericarditis, Dr. Salter heard a to-and-fro friction-sound over a spot not larger than a shilling, in the intercostal space close to the right edge of the sternum. The first and louder sound was presystolic, the second systolic in rhythm; and, as the diastole of the auricle coincided in time with the systole of the ventricle, Dr. Salter inferred that this double sound, limited to the region of the right auricle, was caused by the systole and diastole of an auricle roughened by lymph. A day or two after the occurrence of the auricular friction, the sound spread down over the ventricles, and became general, and a day or two after that the patient died. At the *post-mortem* examination the pericardium was found to be the seat of recent general inflammation, and was everywhere roughened with soft lymph. Lastly, Dr. Salter refers briefly to a case in which a circumscribed friction-sound was heard over the third right interspace close to the sternum; the sound was to-and-fro, and the first and loudest of its two elements was sharply presystolic in time.

Not long after the publication of Dr. Salter's lecture, the following case came under my own observation.

John E——, aged 55, was admitted into Craven ward on October 27, 1873. He had been a wine porter at the London Docks; he had drunk freely, and at the time of

his admission he was suffering from the symptoms of contracted granular kidney in an advanced stage. At my visit, soon after his admission, I noted that a presystolic exocardial friction-sound was heard most distinctly between the left nipple and the sternum, and, as it was obviously synchronous with the auricular systole, I remarked that in all probability the sound was caused by the friction of one auricle, having its surface roughened by lymph. On October 30, in addition to the presystolic sound before noted, there was a systolic friction-sound heard most distinctly at the heart's apex slightly to the left of the mammary line. I then expressed my belief that, in addition to the roughening of the auricle by lymph, there was probably a patch of lymph near the apex of the ventricle. On November 6, the presystolic friction at the base had ceased to be audible, while the systolic sound at the apex was still heard. On December 1, the systolic friction was still heard at the apex, but on December 8 it had become less distinct, and an endocardial systolic blowing-sound was heard at the apex. At subsequent visits it was noted that the systolic sound at the apex had lost its friction character, and assumed the character of a regurgitant mitral murmur. This continued until the patient's death on January 3. We found, as I had anticipated, that the left auricle and the apices of the right and left ventricle were covered, each by a patch of lymph, the surface of which, having become smoothed down by friction, accounted for the cessation of the friction-sounds which had been present when the surfaces were roughened by recent exudation. The margins of the mitral valve were thickened by lymph, and thus the regurgitant mitral murmur was explained.

And now, having learnt, not only by the study of Dr. Salter's recorded cases, but by the observation of this one case under my own care, that an auricle roughened by lymph may cause a friction-sound of presystolic rhythm, I soon saw that in this sonorous influence of the contracting auricle, was to be found the interpretation of the triple friction-sound of pericarditis, with which I had long been familiar as a clinical fact, although I had not hitherto been able to explain it.

Dr. Salter, in his concluding remarks, says: 'Triple friction,

as far as I know, is a new thing, or rather, I should say, an undescribed thing, for no doubt it is as old as pericarditis itself.' This remark is not quite correct, for Dr. Stokes refers to a case of pericarditis in which he observed a triple friction-sound.[1] But although Dr. Salter was not the first to describe a triple pericardial friction-sound, he was, so far as I know, the first to explain it. He evidently, however, looked upon the triple sound as a rare and exceptional phenomenon, whereas it is, in fact, the usual result and indiction of pericarditis, affecting the general surface of the heart.

During the past winter session I have had under my care in the hospital seven cases of acute pericarditis, four rheumatic, two uræmic, and one the result of exposure to cold; and in four out of these seven cases most of you had the opportunity of observing that when the disease was at its height, a triple friction-sound was distinctly audible over the heart, the triple character of the sound being most distinct near the base of the heart, at the junction of the auricles and ventricles. In the two uræmic cases, there being advanced granular contraction of the kidney, the disease was fatal, and the whole surface of both auricles and ventricles was found roughened by recent lymph. In the three cases with a friction-sound not triple, but only to-and-fro, the disease assumed a milder character, having apparently been cut short by the early application of leeches. I have said that the friction-sound resulting from a severe and general pericarditis is triple—*rub-rub—rub*—reminding one, as Dr. Salter say, of the triple sound of a canter. The first two divisions of the triple sound occur in quick succession, the third after a rather longer interval; then follows a pause, and again occurs the *rub-rub—rub*. Now, if while you are listening to this triple sound, you place your finger over the heart's apex or over one carotid, and at the same time bear in mind what you may have seen of the rhythmical contractions of the exposed heart of a living or recently dead animal, you will readily perceive that the first element of the triple sound is auriculo-systolic, the second ventriculo-systolic, and the third ventriculo-diastolic, while the silent interval which follows coincides

[1] *Diseases of the Heart and Aorta*, pp. 29, 30.

in time with the post-diastolic pause. The relation of the triple sound with the heart's movements and rhythm may be represented as follows :—

Rub	Auricular systole.	Triple Friction-sound.
Rub	Ventricular systole.	
Rub	Ventricular diastole.	
Post-diastolic pause.		
Rub	Auricular systole.	Triple Friction-sound.
Rub	Ventricular systole.	
Rub	Ventricular diastole.	

A to-and-fro friction sound might be caused by lymph on the surface of one auricle alone, as happened probably in Dr. Salter's third and fourth cases. When both the auricle and the ventricle are roughened, the second auricular friction-sound (auriculo-diastolic) would of course be blended with the ventricular systolic, the diastole of the auricle and the systole of the ventricle being coincident. A friction-sound at the commencement of pericarditis is usually single and systolic, but it may quickly become double and triple. On the other hand, when the disease is subsiding, the first sound to cease is usually the auriculo-systolic, then the ventriculo-diastolic. A single systolic brush may remain for many days and even for weeks, after the active symptoms have passed away.

The cessation of the friction-sounds after an attack of acute pericarditis is due in a large proportion of cases, not to adhesion of the two layers of pericardium, but to the smoothing down and polishing of the previously roughened surface of the serous membrane. So long as the pericardial surface is roughened, the first sound may be associated with a sound of a superficial brushing character, which an inexperienced or careless auscultator may readily mistake for an endocardial mitral murmur. The diagnosis may be aided by observing that the exocardinal sound is often much increased, and sometimes made to-and-fro, by making firm pressure with the stethoscope over the spot where it is most distinctly heard. The exocardial sound, too, is often heard most distinctly, not at the apex, where a regurgitant mitral murmur is most

audible, but some distance above the apex, over the body of the ventricle, and it is rarely heard at the back of the chest.

I now proceed to the consideration of another interesting phenomenon of cardiac auscultation to which attention has been of late especially directed—I mean the so called *reduplication of the first sound*, which is often associated with Bright's disease, both acute and chronic, and which is also heard in connection with other morbid states to which I shall presently refer. The late Dr. Sibson devoted much time and labour to the investigation of the reduplication of the first sound in connection with Bright's disease. In many cases of Bright's disease it is admitted by all pathologists who have given their attention to the subject, that there is an increase of systemic arterial tension, consequent on the impeded passage of the impure blood through the terminal vessels. The contraction of the muscular arterioles throughout the systemic circulation is, without doubt, the cause of this impediment and of the resulting arterial tension, and the doubling of the first sound is, in some direct way, associated with the increased arterial tension. Dr. Sibson explained the reduplicate first sound by saying that the left ventricle, owing to the resistance offered by the tight arteries to the expulsion of its contents, continues its contraction later than the right, which has expelled its blood into the pulmonary artery with comparative ease. The shock of the first sound is heard at the end of the contraction of the ventricle. Hence, in consequence of the left ventricle contracting more tardily than the right, there is a doubling of the first sound.[1] Dr. Sibson admitted that there is a difficulty in reconciling this explanation of the double first sound with the absence of doubling of the second sound in the same cases, and he met the difficulty with the following statement: —'In these cases the systemic arteries are always in a state of great tension. When the blood ceases to be sent into the tight aorta, the instant contraction of the walls of the arteries sends the blood back upon the aortic walls and valve. The pulmonary arteries at the commencement of the systole are comparatively flaccid, but become tense at the end of it. The walls of the pulmonary artery begin to contract and send

[1] *Lumleian Lectures. Lancet*, March 28 and April 11, 1874.

back a return wave upon the trunk of the artery; but as these walls are not always in a state of tension, they take a longer time to contract than those of the aorta and its branches. Owing, therefore, to the slowness of the pulmonary, and the quickness of the aortic, contraction, the latter, which is already heavily handicapped, makes up in speed what it loses in time, and the two systems of arteries deliver their backstroke at the same instant.' Now, it appears to me that this explanation, while it apparently removes one difficulty, raises another of a very formidable character. If the greater tension of the aorta in the cases under consideration enables it to overtake the earlier but less forcibly contracting pulmonary artery, then it is obvious that in the normal condition, the aorta and the pulmonary artery, commencing their contractions at the same instant, the much greater tension of the aorta should react upon and close its valves before those of the more feebly contracting pulmonary artery are closed, and the result should be reduplication of the second sound as a constant and normal condition.

Again, I have, in numberless instances, demonstrated to you in the wards an analogous doubling of the first sound in cases of advanced general emphysema of the lung, with resulting fulness and hypertrophy of the right ventricle. Here the increased tension of the pulmonary artery consequent on the impeded circulation through the lungs can rarely, if ever, equal the normal tension of the aorta. If then, in accordance with Dr. Sibson's theory, the right ventricle completes its contraction later than the left, and so causes the reduplication of the first sound, the closure of the pulmonary valves must inevitably be effected later than that of the aortic valves, and the second sound would also be doubled. The reverse, however, is the case. The second sound, although accentuated over the pulmonary valves, remains single, while the first is distinctly reduplicated.

But the chief objection to the theory of an asynchronous contraction of the ventricles is an anatomical one. The beautiful dissections of Pettigrew[1] and others show that while the three

[1] *Philosophical Transactions*, 1864. A clear summary, with illustrations of Pettigrew's description, is given in the seventh edition of Quain's *Anatomy*, vol. i. p. 316.

internal layers of muscular fibre belong solely to each ventricle, the three outer layers and a part of the fourth are distributed round both ventricles (figs. 3 and 4). The septum, again,

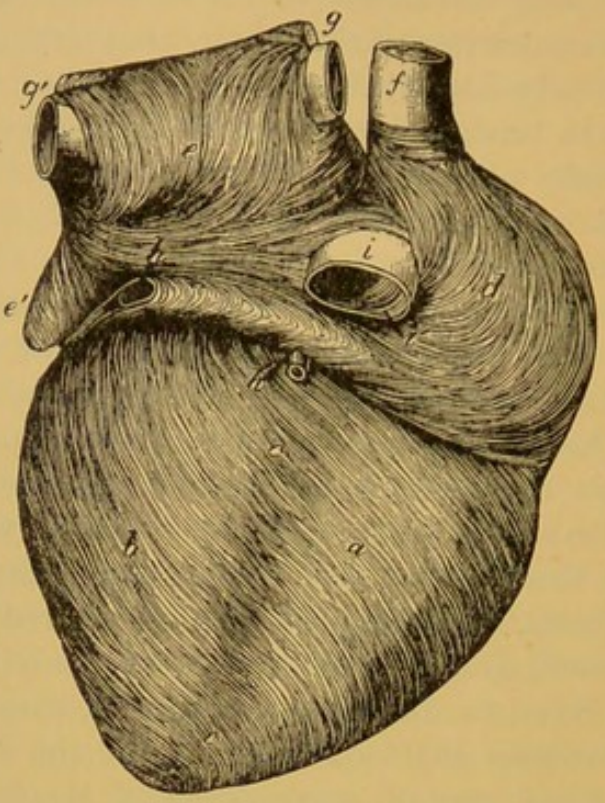

FIG. 3.—HEART OF A YOUNG SUBJECT DISSECTED AFTER BOILING TO SHOW THE SUPERFICIAL MUSCULAR FIBRES SEEN POSTERIORLY. $\frac{2}{3}$.

a. Posterior surface of the right ventricle, with its superficial muscular fibres dissected. *b*. the same of the left ventricle, the fibres passing across from one ventricle to the other. *c*. posterior interventricular groove, from which the coronary vessels have been removed. *d*. right auricle. *e*. left auricle, showing some tranverse fibres common to both auricles, and some belonging to each one. *f*. superior vena cava. *g g'*. pulmonary veins cut short. *h*. sinus of the great coronary vein covered by muscular fibres. *h'*. posterior coronary vein joining the principal one. *i*, inferior vena cava. *i'*. Eustachian valve as seen from behind.—From Quain's *Anatomy*, seventh edition.

consists of three sets of fibres—viz. (1) those belonging to the right ventricle; (2) those belonging to the left; and (3) those common to both. It is obvious from this construction of the muscular walls of the ventricles, that their simultaneous contraction is a physical necessity. Again, the muscular fibres of the auricles consist of a superficial set common to both cavities and of deeper fibres proper to each (fig. 3). This intimate blending together of the muscular fibres of the two simultaneously contracting right and left cavities, both auricular and ventricular, forms a marked contrast with the

manner in which the alternately contracting auricle and ventricle are connected with each other.

The muscular fibres of the auricles are not continuous

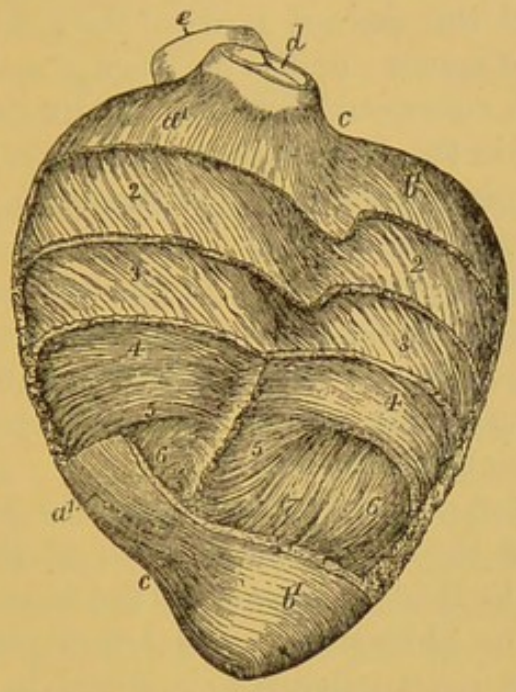

FIG. 4.—VIEW OF A PARTIAL DISSECTION OF THE LAYERS OF FIBRES OF THE VENTRICLES OF A SHEEP'S HEART IN FRONT (AFTER THE MANNER OF PETTIGREW).—$\frac{2}{3}$.

At the base and apex, the pericardium and connective tissue and fat alone have been removed, and the superficial layer of fibres is displayed on these parts of both ventricles. In the intervening space, layer after layer of the fibres has been removed from above downwards, reaching to a greater depth on the left than on the right side. a^1 a^1. the superficial layer of the right ventricle. b^1 b^1. the same of the left ventricle. 2. the second layer of both ventricles. 3. the third. 4. the fourth or central, with fibres nearly transverse. 5. and 6. two of the deeper layers coming next. 7. a small part of the fibres of the deepest layer on the front of the left ventricle, passing into one of the larger papillary muscles, and derived from the posterior superficial fibres, which have entered the whorl of the apex anteriorly. The different degree of obliquity and other changes of direction of the fibres is shown in these several layers. *c c*. between these letters and the numbers is the anterior coronary or interventricular groove, in which, superiorly, the greater part of the fibres of the superficial layer is seen to cross from right to left; in the remaining part of the groove, which is dissected, part of the fibres from both ventricles is seen to turn backward towards the septum. *d*. the pulmonary artery cut short. *e*. the first part of the aorta.—From Quain's *Anatomy*, seventh edition.

with those of the ventricles, the two sets being connected only by the fibrous rings round the auriculo-ventricular orifices; so that when these rings are dissolved by boiling a heart for a few hours, the auricles may be easily separated from the ventricles, while the intimate muscular connection between the two auricles and the two ventricles, respectively, remains indissoluble.

I am at a loss to understand how anyone with a know-

ledge of these anatomical facts can maintain the theory of a want of synchronism in the ventricular contraction, so considerable as to cause an audible double sound.

During the experiments of Dr. Rutherford before described (p. 26) I watched the exposed heart of the living dog in the different stages of apnœa ; first in the stage of systemic arterial obstruction, with enormous distension of the left cavities of the heart, then in the final stage of pulmonary obstruction, with great distension of the right cavities, while the left were collapsed and nearly empty ; and I have been struck by the uninterrupted exact synchronism of the contractions on the two sides ; and this notwithstanding a difference in the blood-pressure and the resistance opposed to the contraction of the two ventricles, very much greater than can occur in the most extreme case of Bright's disease.

A consideration of these difficulties in the way of accepting Dr. Sibson's theory of reduplication of the first sound in association with Bright's disease led me to seek for another explanation of the phenomena ; and I now venture to suggest that the true explanation is to be found in the fact that *the contraction of a dilated, and especially of a hypertrophied, auricle becomes sonorous, and that the first division of the double first sound, in the cases under consideration, is the result of the auricular systole.*

I believe that this explanation of reduplication of the first sound will be found consistent with all the ascertained facts. In the first place, it will be observed that the rhythm of the sound, in cases of well-marked reduplication, is precisely the same as that of the triple friction-sound before referred to, as a result of pericarditis. The triple friction-sound being expressed by *rub-rub—rub*, the triple sounds in a case of reduplication may be represented by *rat-tat—tat*. The cantering character of the sounds may be imitated by bringing down sharply upon the table in quick succession the ends of three flexed fingers, making the two first taps nearer together than the second and third. The triple friction-sounds are longer and more nearly continuous with each other, but the rhythm is precisely the same in the two classes of cases. The relation of the triple sound to the heart's movements may be represented as follows : —

Rat Tat	Auricular systole Ventricular systole	Double first sound.
Tat	Ventricular diastole.	Normal second sound.
	Post-diastolic pause.	
Rat Tat	Auricular systole Ventricular systole	Double first sound.
Tat	Ventricular diastole.	Normal second sound.

The reduplication of the first sound in cases of Bright's disease is usually most distinctly heard between the mamma and the sternum in the third left interspace—that is, about the line of junction between the auricle and ventricle. The sound may be single or indistinctly double at the apex, while it is decidedly double at the third interspace, and again single over the aorta. The position in which I have stated that the reduplication is best heard accords with Dr. Sibson's account, but our explanations differ essentially. He states that, in this position, the supposed asynchronous contraction of the two ventricles is heard, while I believe that the contraction of the dilated, tense, and often hypertrophied auricle is there heard immediately before that of the ventricular systole. That the reduplicate first sound is often inaudible at the apex of the heart, where the normal first sound is best heard, is in itself suggestive that some other agency than ventricular contraction is concerned in the causation of the phenomenon. The assumed asynchronism of the ventricular contraction is a theory unsupported by evidence, but the rapidly successive contraction of the auricle and ventricle is an undoubted fact. The question then arises, Does the contraction of the auricle afford a satisfactory explanation of the first element in the reduplicate first sound? It is of course admitted that in the normal state the contraction of the auricle, contrary to Laennec's original theory of the heart's sounds, is inaudible; but we have positive evidence of sound resulting from the auricular systole in two distinct morbid conditions. First, as a result of constriction of the mitral orifice, we have the now well-known presystolic, or, as Dr. Gairdner happily designates it, the auriculo-systolic murmur. In these cases, the impediment resulting from the mitral constriction causes a slow auricular contraction, with a

prolonged presystolic murmur, followed by a short first sound, the result of rapid contraction of the partially filled left ventricle. Second, when the surface of the auricle is roughened by lymph, there occurs the presystolic, or, better, the auriculo-systolic friction-sound. Then, as a result of obstruction in the systemic arteries, and consequent distension of the left auricle, either with or without hypertrophy of its walls, we have, as I believe, an audible auricular systole, constituting the first division of the reduplicate first sound in cases of Bright's disease. The rhythm of this auriculo-systolic sound is precisely the same as that of the auriculo-systolic mitral murmur and of the auriculo-systolic pericardial friction-sound; and this identity of rhythm in the three classes of cases affords, to my mind, one of the strongest proofs that the first sound, in each case, is excited by the auricular systole. The triple friction-sound of pericarditis and the triple sound resulting from the doubling of the first sound are alike suggestive of a canter.

In one of my cases of rheumatic pericarditis before mentioned, the friction-sound, which had been distinctly triple, assumed for a time the character of a reduplicate first sound. At the stage to which I refer there was a single systolic brush over the ventricle near the mamma, while over the mid-sternum there was a short presystolic brush, which, being quickly followed by the first sound, bore a close resemblance to a reduplicate first sound. The presystolic brush was probably caused by the systole of the right auricle, still slightly roughened with lymph. In a few days more the presystolic sound had ceased, and there remained only the systolic brush over the body of the ventricle.

As I have before remarked, it is sometimes difficult to distinguish the sort of prolongation of the first sound, which is caused by a patch of lymph near the apex of the heart, from a regurgitant mitral murmur. I believe, too, that the forcible friction of a hypertrophied left ventricle in cases of chronic Bright's disease, frequently causes an uneven thickening of the pericardium over a circumscribed space (a white spot on the pericardium—a pericardial 'corn'), which gives a prolonged and brushing character to the first sound, sometimes not easily distinguishable from a systolic mitral murmur. I

have several times diagnosed such a white patch during life, and have found it after death, as some of you here can testify.

I have before stated that the reduplication of the first sound occurs not only in connection with Bright's disease, but it is almost constantly associated with advanced general emphysema of the lungs. I have observed it frequently in elderly persons with degeneration and rigidity of the arterial walls; also very distinctly, above and to the right of the left nipple, in some cases of mitral regurgitation. There is a good example of this in the case of a girl now in Twining ward. There is one feature which is common to all these cases, and that is an impeded circulation, either pulmonary or systemic. The result of this obstruction, acting backwards, is to cause distension and, by degrees, hypertrophy of the walls of one or both auricles. It is obvious that an impediment commencing in the systemic arteries, or at the left side of the heart, may, by a retrograde action, extend through the lungs to the right cavities of the heart.

In some cases of coexisting emphysema of the lungs and chronic Bright's disease, both sides of the heart become simultaneously hypertrophied, and the reduplication of the first sound is distinctly heard over an extensive surface. I have quite recently, in private practice, seen several examples of this complication. It is obvious that the theory of asynchronous ventricular contraction is inadequate to the explanation of the reduplication which results from a simultaneous equal, or nearly equal, impediment in the pulmonary and systemic vessels; while the auricular theory explains the phenomena completely.

I have observed distinct reduplication of the first sound over the base of the heart in some cases of simple dilatation of the heart's cavities, without valvular disease or evidence of arterial obstruction, either pulmonary or systemic. Here, again, the facts are inconsistent with the theory of asynchronous ventricular contraction.

That the systole of a distended and hypertrophied auricle may generate sound is in the highest degree probable. There are two influences which, either separately or combined, may cause an audible sound during the auricular systole—first,

the sudden muscular tension of the walls of the auricle; and, secondly, the impulse against the chest wall.

Dr. Charcelay, of Tours, has published a case [1] in which a presystolic knocking (*claquement*), heard beneath the right edge of the sternum on a level with the second rib, was found after death to have been caused by an aneurysmal dilatation, with thickening of the right auricle; and Dr. Walshe states that 'in a case of hypertrophy of the left auricle, a loud presystolic sound, of maximum force at the third left cartilage, differed absolutely from a reduplicate sound by its special *knocking* character.' [2] Since, then, an aneurysmal, and a much hypertrophied auricle have been known to cause a knocking sound of presystolic rhythm, it is in the highest degree probable that an auricle, in an intermediate state of dilatation and hypertrophy, would generate exactly that amount and kind of sound which we recognise as the first division of the duplex first sound.

I have endeavoured to ascertain whether, by the position in which the double sound is best heard, it is possible to determine which of the two auricles is the cause of the first division of the sound in any given case.

Without doubt, if the auricle is roughened by lymph, the resulting friction-sound will be best heard over the third interspace, to the right or left of the sternum as the right or left auricle is the one affected. I find, too, that in cases of emphysema, with impeded pulmonary circulation, the reduplication is more distinctly heard at the right margin and the lower end of the sternum than to the left; while in cases of renal disease, and cases of senile degeneration of the systemic arterial walls, the sound is best heard between the mamma and the left margin of the sternum—that is, over the space where a distended left auricle would overlap the ventricle. I believe, however, that in consequence of the oblique position of the heart, and the overlapping of the left cavities by the right, the sound which results from contraction of the left auricle will be more or less diffused over the overlapping right ventricle. The left auricular sound, therefore, is with

[1] *Archives Générales de Médecine*, December, 1838, pp. 406-8.
[2] *Diseases of the Heart*, fourth ed. p. 75.

more difficulty localised and distinguished from the right than the aortic from the pulmonary share of the normal second sound, each of which, as we know, is conducted along the course of the artery in which it originates. It is probable, too, that in consequence of the closer approximation of the right cavities to the chest wall, the amount of obstruction in the pulmonary vessels, which will render the right auricular contraction audible, with resulting reduplication of the first sound, is less than the degree of systemic obstruction required to develop the same phenomena on the left side.

In addition to the evidence already adduced in favour of the auriculo-ventricular theory of the double first sound, the following facts appear to me to be of importance :—

1. The doubling of the sound is always more distinctly audible over the body of the heart than at the apex. The reduplication is either lost or indistinctly heard at the apex. Now, since the normal first sound is always best heard at the apex, we should expect to find that, if both elements of the double sound were the result of ventricular contraction, the double sound would be most distinct also at the apex.

2. The part of the double sound which is lost as we pass from base to apex is always the first or presystolic part. Now, in cases of Bright's disease, with obstruction in the systemic arteries, the advocates of the theory of asynchronous contraction of the ventricles might argue that when the stethoscope is placed over the apex of the heart which is formed by the left ventricle, the comparatively feeble sound which results from the earlier contraction of the right ventricle becomes inaudible, and only the single sound of the left ventricle reaches the ear. But, unfortunately for this argument, when, in consequence of emphysema with pulmonary obstruction, the first sound is double near the base of the heart and single at the left apex, it is still the *first* division of the double sound which disappears at the apex ; yet if the want of synchronism of the ventricular systole were the cause of the phenomena, the absent sound would be that of the earlier contracting strong left ventricle, over whose apex the stethoscope is applied. The facts, then, are irreconcilable with the theory which assumes that the doubling of the first sound is due to a retarded contraction

of one or other ventricle. You will readily understand that the distinctness of the reduplication is much influenced by the rapidity of the heart's action. The first sound, which is distinctly double when the heart's action is slow, may lose the reduplication when, in consequence of increased rapidity of the heart's contraction, the sounds of the auricular and the ventricular systole become blended together into one sound, which may then have a somewhat prolonged and brushing character, such as an inexperienced and unskilful auscultator might mistake for the murmur of valvular disease.

Some of you may, perhaps, be inclined to ask whether any practical gain is to be expected from the minute observation and analysis of the heart's sounds upon which I have been dwelling in this lecture. In reply to such an inquiry I would remark, first, that the careful study of physical signs is a very wholesome educational discipline for the student and practitioner of medicine. All observations of natural phenomena, to be of value either for the extension of scientific knowledge or for practical guidance, must be precise and accurate. No one familiar with the subject would question the proposition that the study of physical signs has served not only to correct many erroneous notions which had long prevailed with regard to the general or functional symptoms of pulmonary and cardiac diseases, but that it has also much facilitated and improved the treatment of many of those maladies. Then, just in proportion to the confidence with which you have learnt to rely upon physical signs in aid of diagnosis and prognosis, is the necessity for accuracy of observation and interpretation. If you mistake the soft systolic murmur of anæmia for organic obstruction at the aortic orifice, you not only are wrong in your diagnosis, but you base upon that error an unfavourable prognosis, and perhaps neglect to adopt the chalybeate and restorative treatment, which would at once remove the anæmia and the murmur to which it gave rise. Again, if you mistake a presystolic friction-sound for the presystolic murmur of mitral stenosis, or a systolic friction-sound for the murmur of mitral incompetence, you confound what may be only a transient and harmless roughening of the surface of the pericardium with a serious organic valvular lesion ; and you cause an in-

calculable amount of alarm and anxiety by a prognosis deduced from an erroneous observation. I scarcely need remind you that the only way to avoid these errors of observation and interpretation is, by constant and careful clinical study, to increase and perfect your diagnostic skill.

The reduplication of the first sound, upon the interpretation of which I have dwelt so long, is not without its practical prognostic significance and value, in so far as it affords undoubted evidence that the impediment to the circulation, whether in the pulmonary or in the systemic vessels, is acting backwards through the ventricle upon its associated auricle, and is causing some degree of auricular dilatation and hypertrophy; and, on the other hand, the cessation of the reduplication, as, for instance, in some cases of acute and transient Bright's disease, is evidence of the returning freedom of the circulation, and so far it is of favourable omen. That the contraction of the terminal muscular arterioles excited by blood-contamination, the result of renal disease, should act backwards through the systemic arteries upon the left ventricle and auricle so as to cause an appreciable modification of the heart's sounds, and ultimately hypertrophy of the muscular tissue of the propelling heart and of the resisting and regulating arterioles, is an interesting illustration of the correlation of physiological forces. And you will find hereafter, in the daily practice of your profession, that your ability to observe and to interpret some of the more obscure and doubtful phenomena of disease is much favoured by the study which you have bestowed upon minute physical signs such as those which we have now been discussing.

Postscript.

After the preceding lecture had been written, and a few days before it was delivered, my friend and colleague Dr. Curnow directed my attention to a recently-published thesis, entitled *D'un Phénomène Stéthoscopique propre à certaines formes d'Hypertrophie simple du Cœur*, by Dr. Exchaquet, Interne des Hôpitaux de Paris, 1875. In this publication the author gives the results of observations made in numerous cases by his teacher, M. Potain. These observations have

reference to the modification of the heart's sound which Dr. Sibson and others call reduplication of the first sound, but which the French observers designate *bruit de galop*. It should be borne in mind that *petit galop* is the French equivalent for the English word 'canter.' Dr. Exchaquet raises various forcible objections to Dr. Sibson's theory of reduplication, and maintains that the presystolic element of the double first sound is caused by *an abnormally energetic and sudden contraction of the left auricle*. I have been much interested to find that my explanation of the phenomena of reduplication, arrived at quite independently, has been anticipated and confirmed by M. Potain, who points out that when the chest is not thickly covered by fat the presystolic contraction of the auricle may be seen and felt in the third left intercostal space, where in the same cases the *bruit de galop* is also most distinctly heard. I find, however, that M. Potain looks upon this modification of the heart's sounds as being almost invariably associated with certain forms of albuminuria. As an exaggeration of a normal phenomenon he has observed it to a very slight degree, and as a transient condition, in persons free from organic disease and from functional disorder of any kind; but when the *bruit de galop* is pronounced and permanent, he believes it to be invariably associated with albuminuria and resulting distension of the left auricle, and he looks upon this acoustic sign as diagnostic of certain forms of albuminuria. One of Dr. Exchaquet's 'conclusions' is: 'La présence de l'albumine dans l'urine est constamment observée chez les malades qui présentent ce signe'—i.e. the *bruit de galop*. The author makes no reference to the very frequent association of the *bruit de galop*, *alias* doubling of the first sound, with emphysema and other conditions resulting in an impeded circulation through the lungs, and consequent distension of the right auricle, but unassociated with albuminuria—a class of cases of very common occurrence, to which I have directed attention in my lecture. Dr. Exchaquet discusses the theoretical explanation of the impeded circulation, and the resulting cardiac hypertrophy in cases of Bright's disease, and I have been as much amused as amazed to find him asserting that I adopt and defend Traube's theory, which

attributes the cardiac hypertrophy solely to the impeded circulation through the *kidney*. I had supposed myself to have been amongst the first to point out the insufficiency of Traube's theory, and to suggest an explanation of the phenomena more in accordance with anatomical facts and with physiological principles. If Dr. Exchaquet will do me the favour to refer to my little volume of *Lectures on Bright's Disease*, he will find that my explanation of the relation between cardiac hypertrophy and Bright's disease, whether true or not, is, at any rate, essentially different from that given by Traube.

EPILOGUE.

Since the preceding lecture was published, while I have observed many clinical facts confirmatory of the doctrine therein propounded, I have met with none apparently inconsistent with it. Not long since, the phenomena of a case under the care of my friend and colleague Dr. Beale appeared to afford a remarkable confirmation of the auricular theory of reduplication of the first sound. The following note was made shortly before the patient's death :—

'A. P——, a woman æt. 29, was admitted into Twining ward for disease of the spinal cord. She is thin and anæmic, the front of the chest is flattened. In the third left interspace there is a visible and palpable pulsation, which is immediately followed by the ventricular pulsation at the apex in the fifth interspace, directly below the nipple. It is obvious that the pulsation in the third interspace is auricular ; over that space there is a double first sound, the first division of the double sound being unusually loud and distinct, while at the apex the first sound is single, the auricular contraction being there inaudible. There is no apparent cause for the dilatation of the auricle in this patient. There is no evidence of valvular or renal disease.'

At the *post-mortem* examination, the condition of the heart was noted as follows :—The right auricle and ventricle were extremely dilated and full of decolourised clot. The orifice of the pulmonary artery was enlarged, and, together with the auricle and auricular appendix, occupied the position at which

the abnormal pulsation was felt during life. The tricuspid orifice easily admitted four fingers. The curtains of the valve were healthy. The apex of the heart was formed by the dilated right ventricle. The left ventricle and the aortic valve were normal. The mitral orifice was dilated, and easily admitted three fingers. The left auricle was slightly dilated. Close above the mitral orifice the wall of the auricle was irregularly thickened. The pulmonary and costal pleuræ on both sides were universally adherent. The lungs were emphysematous, the lower lobe of the left being bound down by old false membrane, shrunken, and nearly airless.

The microscopic appearance of the muscular tissue of the heart would have been interesting. In the absence of such information, it may be supposed that the great dilatation of the right cavities was a result of the impeded circulation through the emphysematous lungs.

There can be no question that in this case the first division of the double first sound was due to the contraction of the dilated right auricle. As the dilatation of the auricle was very great, the first element of the reduplicate sound was unusually loud, and approached in character to the 'knocking' sound which was observed in the cases of Drs. Charcelay and Walshe before referred to.

CHAPTER XXXVII.

ON THROMBOSIS AND EMBOLISM: CASES, WITH COMMENTS.[1]

The chief Causes of Coagula within the Circulatory System—Case I. A Thrombus in the Right Ventricle and Auricle—Sudden Death from Obstruction of the Tricuspid Orifice—Case II. Coagula in Dilated Right Cavities—Pulmonary Embolism and Apoplexy—Urgent and Fatal Dyspnœa—Case III. Coagula in Right Cavities and in the Pulmonary Artery—Dyspnœa and Collapse—Case IV. Gouty Thrombosis in the Femoral Vein—Pulmonary Embolism, Pleuro-pneumonia—Recovery—Case V. Clot in Right Side of the Heart—Pulmonary Embolism and Hæmoptysis—Thrombus in Veins of both Legs—Case VI. Hæmoptysis and Pulmonary Apoplexy—Thrombus in Veins of Leg—Pulmonary Embolism—Dyspnœa and Death—Case VII. Vegetations of the Aortic Valves—Embolism in Right Brachial Artery, then in Arteries of both Legs—Phlegmasia Dolens in Left Arm—Case VIII. Transient Pulmonary Embolism (Puerperal) followed by Phlegmasia Dolens—The Pathology of Phlegmasia Dolens—Swelled Leg after Typhoid Fever—Lobular Pneumonia and Ulcer in the Duodenum after Burns—Cases of Cerebral Embolism and Thrombosis—General Remarks on Embolism.

Within a comparatively short period I have met with a considerable number of cases of disease in which fibrinous coagula, large or small, either fixed or movable, and floating within the circulatory system, have played a conspicuous part; and I trust that a brief narrative of some of these cases, together with some reflections suggested by them, may not be without interest for this society. I will preface the recital of the cases by a very few general remarks.

The chief among the conditions which are known to determine the formation of coagula within the circulatory system are the following:—

1. Stagnation of blood or retardation of the blood-stream—as, for example, in an aneurysm after ligature or compression of the artery; in the right side of the heart, when there

[1] Read before the Medical Society of King's College. *British Medical Journal*, Nov. 30, 1872.

is an impeded escape of blood through the lungs or through the left side of the heart. Amongst the causes of an impeded flow of blood through the lungs, may be mentioned the state of apnœa which results from pneumonic consolidation, from capillary bronchitis, from the pressure of liquid on the pleura, or from obstruction in the larynx or trachea. The operation of tracheotomy, too long deferred, may fail to save life in consequence of coagula within the pulmonary artery, the right cavities of the heart, and the systemic veins. A coagulum may form in a dilated left auricle consequent on constriction of the mitral orifice. Of this there is a good example in one of the specimens on the table. Again, coagula may form in the right side of the heart when, in consequence of weakness of the walls or dilatation of the cavities by an obstacle in front, there is an incomplete emptying of these cavities and a partial blood stasis.

2. Coagula will form and adhere upon any part of the interior of the heart or of the vessels which may have been roughened or abraded by disease or accidental injury. Thus, fibrinous concretions form upon the surface of a cardiac valve which has been roughened by inflammation, upon the rough ends of a ruptured tendinous cord, upon the edges of a crack in the endocardium or in the inner coat of an artery; and the blood may coagulate and form a thrombus within an artery which has been roughened by atheromatous or calcareous degeneration, or by syphilitic or other forms of inflammation.

3. A small coagulum often forms the nucleus of a larger concretion. Thus, as layer upon layer of laminated fibrine forms within an aneurysm or on the warty concretions covering an inflamed cardiac valve, so a nucleus of coagulated fibrine within one of the cavities of the heart may rapidly increase by determining a further deposition upon its surface; and minute floating particles of fibrine—capillary embola—may lead to the formation of larger coagula in the systemic veins, in the right side of the heart, or in the pulmonary vessels.

4. There is reason to believe that in certain states of system, such as are found, for instance, in rheumatic subjects, the blood contains either an excess of coagulable material or material which is more than ordinarily prone to coagulate,

possibly in consequence of the presence of some morbid material in the blood.

My first three cases are examples of thrombosis in the right side of the heart—a result, as I suppose, of weakness of the heart's walls, dilatation of the cavities, and consequent partial stasis of blood in holes and corners of those cavities.

CASE I.—Mr. H——, aged 64, had not been in his usual good state of health for about four months before his death, which occurred on October 25, 1871. In June he had an attack of what was called 'gastric disorder,' after which he suffered from shortness of breath on walking uphill. On one occasion in particular, while walking up a steep hill at Dover, his breathing became very difficult. This was at the end of September. Immediately after this he returned to his home in London, where I saw him occasionally with my friend, Dr. Lavies, who was in daily attendance upon him. Mr. H—— was then confined to his room, with a feeling of general weakness, a quick and feeble pulse, hurried breathing on any exertion, some œdema of the ankles, and crepitation over the bases of both lungs. The heart's impulse was feeble, but there were no abnormal valvular sounds. The urine was normal. One of the most distressing symptoms was a sensation of alarm and impending suffocation on first awaking. There was a considerable amount of subcutaneous and abdominal fat, and we concluded that we had to deal with a fat and flabby heart. His condition varied from time to time; but, on the whole, he appeared to be improving under the influence of rest and judicious nursing. On the morning of October 25 he awoke, told the nurse, who was sitting by him, that he felt comfortable, and asked the time. She said, 'A quarter to three.' He began to reply, 'Oh, a quarter,' but suddenly he stopped in the middle of the sentence: the nurse turned and saw that he was black in the face; his tongue protruded between the teeth, both arms and legs were convulsed, and in a few seconds he was dead.

At the *post-mortem* examination we had the assistance of Dr. Kelly. There were small spots of ecchymosis beneath the pericardium. The heart was large, with much fatty growth on its surface. The right side, but especially the auricle,

was much distended with blood. A firm decolourised coagulum filled the apex of the right ventricle, adhering closely to its inner surface, and extended about three or four inches along the pulmonary artery. Its diameter there did not appear sufficient to obstruct seriously the current of blood; but it was also continued backwards through the tricuspid orifice into the auricle, where it formed a band two inches long, an inch wide, and a quarter of an inch thick. The outer surface of this firm clot had been moulded on the wall of the auricle; but it was now free in the cavity, and it had evidently been driven by the current of blood over the tricuspid orifice; the sudden and complete obstruction of which had caused general venous distension with lividity of the surface and arterial emptiness, cerebral anæmia, and consequent convulsions. The lungs were gorged and œdematous at their bases, the bronchi were deeply congested, and contained much frothy mucus.

There can, I think, be no doubt that the abrupt arrest of the circulation in this case was caused by the separation of the fibrinous coagulum from the wall of the auricle, and its consequent valve-like flapping over the tricuspid orifice.

It is probable that this fatal clot had been gradually forming for some weeks before death; and that the immediate cause of its formation was the imperfect contraction of the fat and flabby right ventricle, and the consequent incomplete emptying of its cavity.

Case II.—On November 7, 1871, I saw Miss R——, aged 19. She was breathing rapidly and deeply. She said, 'I feel as if I could not get a sufficiently deep breath.' Her lips were livid, and her skin pallid; the pulse small, rapid, and feeble. The hands felt cold; the temperature in the armpit was 98°. The chest in front was everywhere resonant, except over the middle lobe of the right lung, where it was quite dull. The respiratory sound was exaggerated (puerile), except over the dull space before mentioned, where the respiration was inaudible. The dyspnœa rendered auscultation at the back difficult, but moist râles were heard over both lower lobes. With these sounds, however, there was a free entrance of air. I expressed my belief that she was dying from obstruction at

the right side of the heart or in the pulmonary artery. As she had spat some blood a few hours before, it was probable that the dulness over the middle lobe was the result of pulmonary apoplexy. My first visit was at 3 P.M., and at 7 P.M. her condition remained the same. She died at 11 the same evening.

Dr. Kelly made an inspection with me, thirty-four hours after death. The middle lobe of the right lung was solidified by impacted blood. In other parts of the lungs there were a few smaller hæmorrhagic patches, and also some ecchymoses beneath the pleuræ. The bronchi were congested, and contained much bloody tenacious mucus, down to their smaller branches. This condition of the bronchi was general over both lungs. The cardiac area was rather larger than usual; there was a little clear fluid in the pericardial cavity. The right side of the heart was full of black coagulum; the left side moderately full. Each ventricle formed an apex, so that the heart's apex appeared double. The valves were all healthy. The right ventricle was dilated; its columnæ carneæ and musculi papillares were thicker than usual. A large loose *post-mortem* coagulum lay in the right auricle, ventricle, and pulmonary artery. In the appendix of the right auricle was an adherent, pretty firm, decolourised clot of fibrine, entangled in the musculi pectinati. Similar but smaller clots were found in the apex of the right ventricle, where the deep fossæ between the prominent musculi papillares form a complicated network, most favourable for the formation of a thrombus. On opening the pulmonary artery, a clot was found at the commencement of each main branch; and firm decolourised clots were met with in the smaller branches as far as they could be dissected; so that these vessels were actually distended by coagula which had been driven into them by the right ventricle. The pulmonary veins were healthy, and contained a little fluid blood.

In the early part of the year Miss R—— had been much frightened by a domestic occurrence. From that time she suffered from occasional palpitation, cough, and difficult breathing. She was a zealous dancer; but always, after waltzing, she looked pale and suffered from palpitation. About three weeks before her death she was much distressed by the loss of a locket, the gift of her deceased mother. She became

feeble, and her breathing was oppressed. On October 31 she had great difficulty in attending a wedding as bridesmaid; and after the ceremony was over she went to bed thoroughly prostrated. She suffered from increasing difficulty of breathing and palpitation, occasional sickness and cough, with frothy mucous expectoration, which had an occasional rusty tinge. On the morning of the day on which she died she spat up about a table-spoonful of pure blood. This was followed by an increase of dyspnœa; and at 3 P.M. I found her in the condition which I have already described, the symptoms continuing unchanged until her death at 11 the same night.

In this case, dilatation of the right cavities of the heart—a result, probably, of the combination of feebleness of the muscular walls, emotional excitement, and over-exertion in dancing—led to the formation of coagula in those portions of the auricle and ventricle where, in consequence of incomplete contraction of the walls, the blood is liable to stagnate. Then branches of the pulmonary artery were gradually and successively blocked by embola detached from clots within the right cavities. At length, the obstruction to the pulmonary circulation was so great as to excite urgent dyspnœa, which was still further increased by the hæmorrhage and the consequent impaction of blood in the terminal bronchi and air-cells. It is probable that the blood escaped from ruptured bronchial capillaries, the result of a retrograde passive engorgement of the bronchial veins consequent on the block in the pulmonary vessels,[1] though it is possible that some blood may have escaped from ruptured pulmonary vessels in the neighbourhood of an embolic infarction. The ecchymoses beneath the pleuræ were explained by the same backward engorgement of the bronchial veins and capillaries.

CASE III.—Mr. B——, aged 33, a merchant of somewhat intemperate habits, had hæmoptysis about the middle of March 1872. For this he was seen by Dr. Mumford, who found that he was passing, copiously, urine having a specific gravity of 1035, and containing much sugar. I saw the patient with Dr. Mumford on March 28. In addition to the diabetic symptoms, there was crepitation at the apices of both

[1] See Chapter IV. p. 45.

lungs. On April 17 the symptoms were somewhat mitigated. The patient was dressed and down-stairs. On April 23 I was called to see him at 8.30 in the evening, in consequence of a serious aggravation of his symptoms. I found him in bed, breathing hurriedly and deeply. The hands were very cold and clammy; the pulse small, feeble, and rapid; the lips blue; the face pale and anxious. The respiration was puerile everywhere except at both apices, where loose crepitation was audible. The sounds of the heart were normal, except that the first sound was shorter than usual. I was told that difficulty of breathing had been gradually coming on for two days, and that the dyspnœa had been urgent for about twelve hours. I expressed my opinion that he was dying from a coagulum at the right side of the heart. He died at 11.30 A.M. the following day.

Thirty hours after death, Dr. Kelly examined the body with Dr. Mumford and me. A firm, colourless clot was found adherent to the apex of the right ventricle; this extended along the pulmonary artery, becoming small and black beyond the bifurcation. At the pulmonary orifice, the clot was equal to about one-third the diameter of the vessel. It extended also backwards from the ventricle through the tricuspid orifice into the auricle, where, on the auricular side of the tricuspid orifice, it was enlarged to the diameter of nearly an inch; so that the tricuspid orifice was much obstructed and the auricle was distended with dark liquid blood; while the ventricle was little, if at all, distended. The walls of both the auricle and the ventricle were thin, soft, and covered by fatty growth. The left cavities were empty. There was a moderate quantity of serous fluid in the pericardium, and some spots of ecchymosis beneath the serous layer. There were cheesy deposits in the apices of both lungs, and two small cavities in the left apex. The lungs were gorged with serum; the mucous membrane of the bronchi was congested, and covered by frothy mucus. This engorgement with mucous exudation must have come on after my visit—i.e. during the last fourteen hours of life—a result of passive congestion of the bronchial veins and capillaries, in common with the whole systemic venous system, consequent on the obstruction at the right

side of the heart. The serous effusion into the pericardium and the ecchymoses beneath the membrane were results of a similar passive engorgement of the coronary veins and capillaries.[1]

The formation of the clot in this case probably began in the apex of the right ventricle, a result of partial stasis of blood there, consequent on degeneration and impaired contractile power of the muscular walls. The patient, for some weeks before his death, had been subjected to frequent and violent emotional excitement, which may probably have increased the distension of the right cavities, while it tended to lessen the contractile power of their walls.

It will be seen that the condition of this patient, a few hours before his death, was very similar to that of one in the collapse stage of cholera.[2]

An embolon passing into the pulmonary artery and not causing a fatal obstruction there may excite inflammation of the tissue of the lung. Of this the following case is an example.

Case IV.—I am indebted to my friend, the late Dr. I——, for the outline sketch of an illness of which he was himself the subject. He was in his fifty-second year, and had had repeated attacks of gout. While travelling in Switzerland, he sprained his left knee severely on August 3, 1872. On the 7th he had gout in that joint and in the ankle on the same side. Next day, the left foot was inflamed; and on the following day, the right foot. On August 12, the gout was gone, and the knee better. On the 15th he went down-stairs. On the 16th, 17th, and 18th, the calf of the left leg was increasingly painful. On the 16th, while sitting quietly, he felt a curious sensation in the left calf and along the thigh, and felt sure that a clot had started on its travels. In about two minutes he felt it settle in the right lung, two inches below the nipple. He was not at first conscious of any dyspnœa; but a friend called, and in conversation he found that he was short-breathed. A short irritative cough came on. Pulse 76. On the 21st, the pain in the left calf was so severe as to compel him to lie in bed. He then found that the leg below the knee was much swollen and hard. The

[1] See Chapter IV. p. 46. [2] See Chapter VI. sec. vi.

dyspnœa was so great that he could neither read aloud nor talk. On the 24th there was pain along the course of the femoral vein, and the vein felt hard and distended. On the 28th the whole left limb was from two to two and a half inches larger in diameter than the right; dyspnœa more considerable. The temperature of the left leg was 104, that of the right 98°. He had extreme debility, and complete loss of appetite. On August 29 he left Zermatt in a litter at 7.30 A.M., and reached Visp at 6.30 P.M. August 30 he was in bed all day, with pain in the right side. On August 31 he was travelling by carriage and rail from 7 A.M. to 1 P.M. The dyspnœa was so severe that he could with great difficulty take half a table-spoonful of soup at a time. In the afternoon there was intense pain below the right mamma, and the physical signs of pleurisy were present. On September 1 the cough was more severe; he expectorated one large solid mass of blood and mucus, followed by three smaller masses, and the cough ceased for the day. September 2 cough and expectoration returned from 4 A.M. to 6 A.M., then ceased; pleuritic pain continued. On September 3 he slept soundly, for the first time since the sprain, thirteen hours; sweated profusely; the morning cough and bloody expectoration were as before. He went to Geneva in an invalid carriage; on arrival, the dyspnœa was extreme, so that he could not talk in his usual tone of voice. From this period all the pulmonary symptoms gradually abated, and his appetite and strength returned; but the limb remained enlarged and painful. I saw Dr. I—— in London on September 30. There was then no trace of mischief within the chest, and, with the exception of the remaining swelling and weakness of the left lower extremity, he was in his usual good health and spirits.

In this case, the original source of the mischief was a clot which had formed in a vein in the leg, consequent on a partial blood-stasis, connected with the gouty inflammation.

Case V.—On October 26, 1871, I saw, with Mr. Erichsen and Mr. Jones, of Epsom, a gentleman aged 64, who, having had much mental anxiety for several months, suffered from sciatica on the left side. This came on in May, and con-

tinued for several weeks. One day in July he was seized with sudden faintness at the water-closet, and nearly died. A second faint occurred the following morning. Then there followed the signs of pulmonary engorgement, wheezing over the chest, hæmoptysis, and signs of consolidation of a portion of one lung. The lungs gradually became clear; then followed a painful swelling of the left leg and thigh; and afterwards the right limb was similarly affected. When I saw him, both lower limbs were still swollen, and the left was discharging freely from punctures in the skin. The heart's sounds were normal, but the impulse was feeble. The pulmonary sounds were normal. There was a moderate quantity of albumen in the urine. I saw this gentleman again with Mr. Jones on February 16, 1872. I then learnt that the swelling of the legs had gradually passed away; and the urine had ceased to be albuminous. About a fortnight before he had a sudden faintness, continuing for about twenty minutes; since then he had a conviction that, if he swallowed food, it would not pass through him. He consequently refused nourishment, and became very feeble. The pulse was 120, weak, sometimes irregular; the heart's impulse feeble, the first sound sharp and clear. He gradually sank and died from exhaustion.

In this case it is probable that a thrombus, originally formed on the right side of a feeble and perhaps dilated heart, passed as an embolon into the pulmonary artery, where it caused pulmonary congestion and hæmoptysis; then coagula, passing into the systemic arteries and capillaries, led to the formation of clots, first in the veins of one leg, and subsequently in those of the other. It is probable too that the albuminuria may have been a result of embola in the renal vessels.

There is, I think, good reason to believe that coagula, which have originally formed within the pulmonary capillaries, may thence pass through the systemic arteries and cause capillary and venous obstruction in the lower extremities.

CASE VI.—In the autumn of 1869 I saw the following case. A married lady, aged about 40, the mother of a large family, healthy, but of a somewhat delicate frame, during the early part of September had a troublesome dry cough; and

one day she began to cough up a large quantity of florid blood. So rapid was the hæmorrhage, that at one time she was nearly suffocated; and the active bleeding continued for several hours. I first saw her at Eastbourne on September 17, a week after the onset of the bleeding. She was then expectorating small quantities of semi-solid dark blood, which had evidently been for a considerable time out of the vessels. Percussion and auscultation over the front of the chest gave quite normal results, and, as the chest-walls were thin, the air could be heard entering the lung with great clearness. Over the right lower lobe at the back there was marked dulness on percussion, and, on auscultation, rather fine crepitation, with diffused blowing expiration. The inference was, that the right lower lobe had been partially consolidated by blood driven into its tubes and air-cells (pulmonary apoplexy). Percussion and respiratory sounds over the left back were quite normal. The pulse and breathing were somewhat quickened, but there was no febrile excitement. A very few days afterwards, I heard that the chest symptoms were better, but that the left leg below the knee had become painful and swollen, though not inflamed; and I suggested that probably some coagula from within the pulmonary capillaries, having entered the circulation, had led to the formation of clots within the capillaries and veins of the leg. On October 4 I again saw the patient. I found that, during the last few days, the painful swelling of the leg had entirely subsided; but this had been succeeded by urgent dyspnœa. The breathing was still upwards of forty in a minute, the pulse rapid and feeble, the countenance anxious, the face pale, the lips livid.[1] There was now normal resonance over the lower lobe of the right lung; the air entered freely, and there was scarcely a trace of crepitation left. Over every other part of the chest, percussion and respiration were quite normal. The blood which, for a time, had blocked a portion of the lung-tissue had been expectorated, and evidently there was nothing in the state of the lung to explain the alarming dyspnœa. What, then, was its cause? Probably a fibrinous clot, from the

[1] In this case, again, the condition was very similar to that of choleraic collapse. See Chapter VI. Section VI.

temporarily obstructed vein in the leg, had made its way to the right side of the heart, and there was embolic obstruction of the pulmonary artery. The dyspnœa was due, not to want of air in the lungs, but to want of moving blood. After my visit, the dyspnœa continued and increased, and she died early on the morning of October 6.

There was no inspection of the body, but there can be scarcely a doubt as to the sequence of events. Pulmonary hæmorrhage led to impaction of blood in the lower lobe of the right lung. This was soon expelled by coughing; but meanwhile probably, coagula formed within the pulmonary capillaries while the blood was rendered stagnant by pressure from without, passed into the circulation and led to the formation of clots within the capillaries and veins of the leg; thence embola found their way back to the right side of the heart, where they increased and caused a fatal obstruction to the circulation.

Case VII.—In confirmation of the explanation which I have given of this and the previous case, I may refer to a case related by Sir James Simpson.[1] A woman died of phlegmasia dolens of the left arm and side of the face some weeks after an exhausting labour. About a year before she became pregnant she had an attack of rheumatic endocarditis. After her delivery she had signs of embolism, first in the right brachial artery, and then in both legs; then came on phlegmasia dolens in the left arm. After death, the aortic valves were covered over with wart-like excrescences, and it is suggested as probable that fibrinous detritus, passing from the arteries into the capillaries and veins of the left arm, caused phlebitic thrombosis and the other phenomena of phlegmasia dolens.

It is notorious that coagula in the right side of the heart and in the pulmonary artery have frequently caused the death of parturient women. This fatal occurrence appears sometimes to have been unassociated with symptoms of blood-poisoning or of constitutional disturbance; and it seems not improbable that coagula from the uterine veins may pass into the returning current of venous blood, and so form the nuclei of further concretions at the right side of the heart.

[1] *Clinical Lectures on the Diseases of Women*, p. 354.

CASE VIII.—Five years ago (in 1867) I saw, with Mr. Charles Matthews, a woman who, a few days after her confinement, had been suddenly seized with the most alarming symptoms of collapse. Mr. Matthews having been called to her at this time of alarm, heard a bellows sound over the situation of the pulmonary artery, and he concluded that a clot was partially obstructing the vessel. On the following day, when I saw her with Mr. Matthews, the alarming symptoms had passed away and the cardiac and pulmonary sounds were quite normal. A few days afterwards, she had phlegmasia dolens in one leg, and it is probable that fibrinous *débris* from the clot in the pulmonary artery may have passed through the capillaries of the lung into the systemic arteries, and thence to the capillaries and minute veins of the limb which was the seat of phlegmasia dolens. The passage of fibrinous coagula from the pulmonary capillaries into the systemic arteries, and the resulting phlegmasia dolens in one leg, would render this case analogous to Cases V. and VI., which I have before narrated.

The late Dr. F. W. Mackenzie, in his elaborate paper on phlegmasia dolens,[1] has shown that obstruction of the femoral or iliac vein causes soft œdema of the legs; but that, in order to excite the hard painful swelling which constitutes phlegmasia dolens, the smaller venous radicles must also be obstructed; and he shows it to be probable that this obstruction is often the result of some morbid element in the blood which has gone the round of the circulation. Thus in one experiment,[2] lactic acid having been injected into the right femoral vein of a dog towards the heart, not only were the right iliac veins and some of their branches obstructed by a firm adherent decolourised coagulum, but the right femoral vein, down to the popliteal and several of its branches, were similarly obstructed, and a firm coagulum also occupied the left femoral vein down to the popliteal. The coagula in the left femoral vein and in the right femoral below the place of injection are attributed to the action of the lactic acid after it had gone the round of the circulation.

Phlegmasia dolens, although in the majority of cases

[1] *Medico-Chirurgical Transactions*, vol. xxxvi. [2] P. 210.

puerperal, is of by no means rare occurrence in non-puerperal conditions. Thus, out of 100 cases referred to and analysed by Dr. Mackenzie, no fewer than forty were non-puerperal, and of these forty, more than one-third occurred in males. Even in parturient women, it is doubtful whether it is ever a result of a direct extension of disease from the uterine to the pelvic and iliac veins. It seems, on the whole, more probable that some morbid materials—possibly small fibrinous coagula or minute particles of fibrine in a state of decomposition—passing from the uterine veins into the general current of the circulation and returning through the systemic arteries, determine the coagulation of the blood in the capillaries and veins.

The greater frequency of the disease in the lower than in the upper extremities is probably accounted for by the retardation of the blood-stream in the more distant parts, where, too, the venous current has to return against gravity. It has also been suggested that the greater frequency of the disease in the left than in the right leg, in the proportion of three to two, may be, in part at least, explained by the fact that the left common iliac vein has to pass behind the right iliac artery ; and when the blood-moving forces are defective, it is thought that the pressure of the artery upon the vein may somewhat retard the venous current, and so favour coagulation of the blood. It is well known that swelling of one or other leg, sometimes of both—a form of phlegmasia dolens—is an occasional sequela of typhus and of typhoid fever. This complication has occurred in several cases under my own observation. What is the explanation of this phenomenon? While thinking over this subject in connection with the history of the cases which I have here narrated, it has occurred to me that probably the swollen white leg after fever may be thus explained. Pulmonary engorgement, more or less, is a common result of fever, both typhus and typhoid; during this state of engorgement, coagula may form in the pulmonary capillaries, and, subsequently passing into the systemic circulation, may cause capillary and venous obstruction in a manner analogous to that in which I suppose it to have occurred in Cases V., VI., and VII.

In a fatal case of typhoid fever,[1] in which there was much pulmonary engorgement, there was found after death a recent fibrinous patch in one kidney, a result, as I suppose, of the impaction there of fibrinous coagula from the pulmonary capillaries. The valves of the heart were quite normal, but the fibrinous patch was exactly similar to those which are often associated with warty concretions on the cardiac valves.[2] The probability of this being the true explanation of the swelled leg after fever is increased by the fact that a similar complication is occasionally observed in cases of phthisis, pleurisy, and pneumonia; in which cases it is likely that coagula may have passed from pulmonary capillaries into the systemic vessels. I have met with this complication in several cases of phthisis. The late Dr. Cursham has published four cases of phthisis complicated with phlegmasia dolens.[3] In two of these cases there was ulceration of the intestines, but in the other two no such ulceration was found, and Dr. Cursham attributes the obstruction of the veins to 'the presence of pus or some foreign matter in the blood.'

Dr. Warburton Begbie, in a paper on the swelled leg of fevers,[4] refers to four cases of pleurisy in which swelling of the lower limb occurred on the same side as the pleurisy. It is likely that coagula, formed with pulmonary capillaries where the circulation has been retarded by the pressure of liquid in the pleura, might pass on into the systemic vessels, and there become nuclei of new clots and consequent capillary and venous obstructions. If the occurrence of the swelling in the limb on the same side as the pleurisy be not a mere coincidence, it may possibly be due to the fact that, as a rule, a pleuritic patient lies on the affected side, and this position may involve greater pressure on the undermost leg, and some additional retardation of the venous current in the compressed limb.

The lobular pneumonia which so often follows an extensive

[1] Sarah Gaily, *Hospital Case Book*, xxvii. p. 69.

[2] In some cases of typhoid fever it is not improbable that coagula or some other form of blood-contamination may start from the vessels at the seat of the intestinal lesion.

[3] *Medico-Chirurgical Transactions*, vol. xliii.

[4] *Edinburgh Medical Journal*, September, 1872.

burn of the skin has for its probable exciting cause the passage of more or less completely coagulated blood from the seat of the burn into the minute pulmonary vessels; while the ulceration, which in these cases has sometimes been found at the commencement of the duodenum, may be explained by coagula passing through the pulmonary capillaries, forming embola in the intestinal vessels, and thus rendering circumscribed portions of the mucous membrane liable to be softened and dissolved by the acid chyme before it is mixed with the alkaline biliary secretion. These duodenal ulcers appear to be analogous to the so-called simple gastric ulcer, the most probable explanation of which is that plugging of vessels in the mucous membrane by thrombosis or embolism, so interferes with the nutrition of a circumscribed patch of the membrane as to render it soluble by the gastric fluid.

Time permits only a passing allusion to the various forms of brain-disturbance which are direct results of embolism or thrombosis. We have had in the hospital some most interesting typical cases.

In the case of a man named Edward Smith, in October 1867, with a diastolic bellows sound at the base, there was sudden left hemiplegia; and after death a plug was found in the right middle cerebral artery, with softening of the corresponding corpus striatum, and fibrinous vegetations on the aortic valves.

Until Professor Virchow directed the attention of pathologists and practising physicians to the phenomena of embolism, the association of sudden hemiplegia with the *post-mortem* appearance of a circumscribed patch of white or atrophic softening could not fail to be misinterpreted. Dr. Todd often directed attention to the clinical facts. He believed that the softening resulted from 'disease of the artery or arteries leading to the part,' and his explanation of the sudden onset of paralysis I give in his own words.[1] 'A portion of the brain may be undergoing a gradual process of softening for some time, and yet the continuity of its fibres may be preserved, and their constitution may not be so much altered as to prevent them from carrying on their function as conductors—in an

[1] *Clinical Lectures on Paralysis, &c.*, 1855, p. 95.

impaired way, perhaps—but not sufficiently so to call attention to it. Suddenly from some temporary excitement, perhaps, the fibres give way, and all power of voluntary motion is lost, as suddenly as the galvanic current ceases on breaking the circuit.' I doubt whether this hypothesis was entirely satisfactory even to the mind of the great clinical teacher who propounded it; and the subsequent discovery of embolism at once threw an entirely new light on this, heretofore, obscure subject. The closure of an arterial channel by an embolic plug, immediately suspends the functions of the part of the brain whose blood-supply is thus cut off; the softening of the brain-tissue, with or without hæmorrhage into the softened part, is an after-result of the arrested circulation.

In the case of Sarah Elizabeth Keen, December 1869, there was sudden coma, with first rigidity, then palsy of the right arm and leg; after death a thrombus was found in the atheromatous left middle cerebral artery, with softening of the whole corpus striatum and the outer part of the optic thalamus. In the twenty-third volume of the *Pathological Transactions*,[1] Dr. Kelly has published a case of acute fatal chorea in a girl aged 9. Amongst other points of interest there was softening of both corpora striata. Some minute arteries in the softened parts were filled with granular fibrine. There were recent soft vegetations on the mitral and tricuspid valves. It is pretty certain that acute chorea is sometimes a direct result of capillary embolism of the corpus striatum and the neighbouring parts.

Another most interesting case was that of a young woman named Mary Ann Hennessey.[2] She had rheumatism twelve years before admission and palpitation since; a systolic bellows sound at the apex was sometimes high pitched and musical, then soft and blowing. The changing character of the murmur was probably a result of the alternate deposit and dislodgment of fibrinous concretions on the mitral valve. In addition, there was a high and variable temperature; then plugging of the brachial artery; hæmaturia, probably from renal embolism; lastly, cerebral excitement, delirium, and illusions, consequent, as we supposed, on plugging of some cerebral vessels.

[1] P. 94.

[2] *Hospital Case Book*, vol. xliii. p. 269.

In this state she insisted on going home, where we heard that the excitement soon passed off, and she was able again to attend to her shop.

I venture to make one generalisation which appears to me to be warranted by clinical observation. The symptoms to which a stationary clot in the right side of the heart or in a systemic vein gives rise, by engorgement of vessels extending in a backward direction, are usually of a passive character, and are developed slowly; on the contrary, when coagula, large or small, pass onwards with the blood stream, they often excite active symptoms with great rapidity; the character of the symptoms being almost infinitely various, according to the nature of the part which is implicated—whether lung, brain, kidney, intestine, &c.; and, again, whether arterial trunk, minute arterial branches, capillaries, or veins. Minute particles of fibrine mingling with the blood may probably go the whole round of the circulation, with, of course, a liability to be arrested in any set of capillaries, and thus to become the centre and source of fresh mischief. There is reason to believe, too, that, apart from the mere mechanical results of floating masses of fibrine, fibrinous *débris* in the blood may have something of a toxic influence, and thus induce febrile excitement, with a high temperature.

I cannot conclude without an expression of admiration for the clinical acumen of the late Dr. Kirkes, who was the first in this country to follow up Professor Virchow's original observations and to indicate what, in his paper in the *Medico-Chirurgical Transactions* (vol. xxxv.) he calls 'some of the principal effects resulting from the detachment of fibrinous deposits from the interior of the heart, and their mixture with the circulating blood.' That paper opened out an entirely new field of pathological and clinical research, the importance of which is yearly becoming more apparent.

CHAPTER XXXVIII.

ON THORACIC AND ABDOMINAL ANEURYSM.[1]

SECTION I.

A CLINICAL LECTURE ON ANEURYSM OF THE ARCH OF THE AORTA.

Case of Aneurysm of the Transverse Aorta pressing on the Trachea—Two similar Cases in which a loud Cough was a Prominent Symptom—Aneurysm of the Arch of the Aorta causing Loud Stridor and ultimately Suffocation by Pressure on the Trachea—Cases in which Tracheotomy may be useful—General Rules for Treatment of Internal Aneurysms.

GENTLEMEN,—I wish to direct your attention to a case of aneurysm of the arch of the aorta in Craven ward. The case I refer to is that of C. H——, aged 40, who was admitted January 29. I will give you a brief abstract of his history from the notes of Mr. (now Dr.) Tirard. He was formerly a labourer in Woolwich Dockyard, but during the last five years he has been a storekeeper there. He states that he has not been intemperate; and so far as one can see there is no evidence of intemperate habits. He believes that he had syphilis about twenty years ago, but he appears not to have had secondary symptoms. He had rheumatic fever when a lad, and had suffered from occasional rheumatic pains since. The history of his present attack is this:—About four months ago he caught cold, and felt ill. He had a cough, with scanty expectoration, and the cough has continued up to the present time. For the last three months he has had difficulty of breathing, especially when lying on his back. When admitted his skin was somewhat cyanosed; he had an anxious expression of countenance and considerable difficulty of breathing. There was a frequent loud ringing cough with scanty mucous

[1] *Medical Examiner*, March 16, 1876.

expectoration. The physical signs are these:—The chest is somewhat barrel-shaped, with very limited expansion, these signs indicating general emphysema; no abnormal dulness on percussion; loud mucous râles in the large bronchi; on deep inspiration there is tracheal stridor; the heart-sounds are normal. The loud ringing cough first excited our suspicion that he had aneurysm; and on further examination we found evidence of that disease. At the top of the sternum there is a rather rough systolic murmur and a very distinct audible but not palpable impulse. I mean that we can *hear* the impulse against the sternum, but we cannot *feel* it by the hand. On auscultation over the same spot we hear tracheal breathing, and loud vocal resonance; also over the spinous processes of the upper dorsal vertebræ we hear the same sound of tracheal breathing, and increased vocal resonance. On pressing firmly the ends of the fingers into the second intercostal space at the right edge of the sternum, we fancy sometimes that we feel an impulse there; this, however, is a little doubtful. The pulse at the wrist is equal on the two sides. There is no difficulty of swallowing.

The inference from these physical signs is that the man has an aneurysm of the transverse portion of the arch of the aorta which is pulsating against the sternum in front. We hear the pulsation distinctly on applying the stethoscope there, and we hear the tracheal breathing in front over the top of the sternum and also over the upper dorsal spines at the back. There is also very marked increased vocal resonance at those spots. This increased vocal resonance we may call *tracheophony*, just as we speak of bronchophony. The explanation of the signs is obvious enough. As the aorta is dilated, it touches the sternum in front and presses back upon the trachea behind, so that the sound of the respiration and of the voice is conducted from the trachea through the aneurysm to the sternum in front, and through the bodies of the vertebræ at the back.

Some of you will remember a case of aneurysm which was under my care in Craven ward about two months ago. In that case the symptoms and physical signs were very similar to those in the case which I have just described, and

it will be interesting to compare the two cases. James H——, aged 40, was admitted on November 5. The notes of his case were taken by Mr. Tirard. I will briefly recapitulate the chief points of the man's history, and you will see how strikingly similar the two cases are in their main features. This man was twenty years in the army, of which nineteen years were spent in India. He drank rather freely. He left the army at the end of 1871, and since then he has worked as a labourer in Woolwich Arsenal. Three months before admission he had been troubled with cough and difficulty of breathing. A few days before admission he came as an out-patient, and the house-physician, Dr. Hebb, hearing his loud resonant cough, as he stood amongst a crowd of patients, suspected that an aneurysm was pressing on the trachea, and calling him forward discovered the disease, and persuaded him to come in. This was a diagnosis made instantaneously by attention to that loud resonant cough, of which I shall have more to say presently. In that case the physical signs were even more distinct than in the man who is now in the hospital. There was a very distinct pulsation in the second intercostal space just to the right of the sternum. On auscultation over the top of the sternum a distinct impulse was heard with each systole of the heart, also harsh tracheal breathing and loud vocal resonance over the upper dorsal spinous processes. In that case there was an inequality of pulse at the two wrists. The right radial pulse was weaker than the left. There was also a harsh, resonant, barking cough, which was always worse at night when lying down or when he bent his head forward. On laryngoscopic examination we found the structure of the larynx quite normal. A cough mixture, containing morphine and hydrate of chloral, was given, and the cough, which at first was very troublesome, under the influence of the sedative and rest in bed soon became easier. At his own request he was discharged on December 11, having been relieved of his more distressing symptoms, though the physical signs of the aneurysm remained unchanged. The physical signs and the symptoms, you see, were much the same in these two cases, and, in particular, both patients had the same loud ringing cough.

Now to this cough, as a sign of aneurysm, I wish to call your particular attenion. It is very peculiar and characteristic, and it may sometimes lead you, as it has led me, to search for and find an aneurysm which might otherwise have remained undiscovered.

The late Dr. E. J—— consulted me at the beginning of July on account of a cough, apparently caused by a long uvula, which, by my advice, he had removed. The removal of the uvula greatly relieved the cough for a time, but when I saw him about a fortnight later he said he was no better, and he had determined to go to Switzerland for three weeks' holiday. It so happened that on July 31 I met him on board the steamer at Dover, and I travelled from Ostend to Strasburg in the same railway carriage with him and the medical friend who was to be his travelling companion through Switzerland. In the course of that journey of about fifteen hours my attention was arrested by his loud ringing cough, and I expressed to his companion my belief that this cough was caused by an aneurysm of the aorta pressing on the trachea. In consequence of that conversation Dr. J——'s friend and companion did everything that was possible to prevent him from taking long walks or exerting himself during his Swiss tour. I left the train at Strasburg, and did not see Dr. J—— again. I afterwards heard that he called on me after his return from Switzerland, in the last week of August, and, hearing that I was not expected home for some days, he sought other advice; and about six weeks later he died, after the operation of tracheotomy had been performed, without relieving the dyspnœa. I heard from a friend, who was present at the *post-mortem* examination, that a large aneurysm of the arch of the aorta was found pressing against the sternum in front, and on the trachea behind. The pressure on the trachea caused death by apnœa. I did not examine Dr. J——'s chest before I heard the characteristic cough, and I had no opportunity of doing so afterwards; but the loud ringing cough left no doubt in my mind that the pressure of an aneurysm on the trachea was the cause of that symptom.

You see that this loud cough is a really valuable diagnostic symptom. The question then arises, what is the explanation

of its peculiar resonant character? I believe that the cough is more tracheal than laryngeal; I mean that the sound originates mainly in the trachea, and not in the larynx. In ordinary coughing the patient, taking a deep inspiration, fills the chest and closes the glottis, then he forcibly drives the air through the partially closed glottis, and so the sound of the cough originates in the larynx. But a cough which results from the pressure of an aneurysm on the front of the trachea is, I believe, much modified by the forcible driving of the current of air through a constricted and misshaped trachea; as a consequence of which the walls of the trachea are thrown into strongly resonant vibrations. With a forced inspiration there is usually in these cases tracheal respiratory stridor; the inspiratory current of air is interrupted by the narrowing of the canal, and this causes stridulous breathing, which originates at the constricted portion of the trachea.

There are two modes in which an aneurysm pressing on the trachea may destroy life. First, it may burst into the trachea, and cause death either by hæmorrhage or by suffocation; secondly, it may so narrow the canal of the trachea as to cause death simply by suffocation. This happened in the case of an aneurysm which is represented in this photograph. Many of you may have heard me refer to it before. I published the particulars of the case in the *British Medical Journal*, Dec. 23, 1871. It was the case of a gentleman who was brought to me by my friend Dr. Richards, of Winchester.

A gentleman 33 years of age had suffered from shortness of breath, with noise in the throat, on any unusual exertion, for nearly a year. When I saw him on October 30, 1871, there was loud stridor on a deep inspiration; but with this the voice was clear and distinct, indicating, as I at once said, that the obstruction which caused the stridor was below the larynx. The laryngoscope showed nothing more than slight redness of the mucous membrane, without swelling. The vocal cords moved freely; and, during a deep inspiration with a widely open glottis, there was loud stridor, evidently originating below the larynx. Suspecting aneurysm, I carefully examined the chest. There was no abnormal impulse or dulness on percussion. On auscultation behind, over the upper

dorsal spinous processes, there were loud tracheal stridor and vocal resonance. The respiratory murmur was decidedly more feeble over the lower lobe of the right lung than over the corresponding part of the left side. There had been occasional difficulty in swallowing. The pulse was equal at the two wrists. The right pupil was smaller than the left—a result, probably, of iritis some years before. The diagnosis was, aneurysm of the transverse aorta pressing on the trachea. I did not see the patient again; but I learnt from Dr. Richards that the symptoms became steadily worse, until he died from apnœa on November 20, three weeks after his visit to me. An aneurysm of the size of a walnut projected from the posterior wall of the transverse aorta, and bulged into the trachea just above its bifurcation (fig. 5). The opening of the right

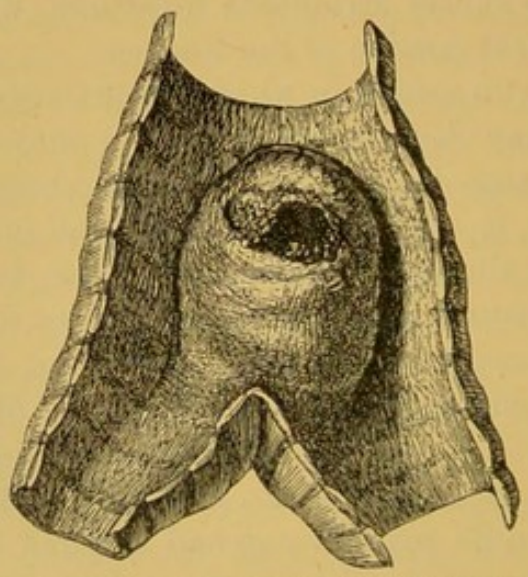

FIG. 5.—THE TRACHEA OPENED FROM BEHIND, SHOWING AN ANEURYSM BULGING THROUGH ITS ANTERIOR WALL.
From a Photograph kindly sent to me by Dr. Richards.

bronchus was somewhat more obstructed than that of the left, thus explaining the comparative feebleness of the respiratory murmur on the right side of the chest. One advantage of an exact diagnosis in this case was, that the patient was not subjected to the operation of tracheotomy, which, on a superficial view of the symptoms, might seem to have been indicated. The operation was not resorted to, because it was known that the obstruction in the trachea was below the point at which an artificial opening could be made, and therefore no relief could have been given by an operation.

I scarcely need tell you that, with reference to the question of tracheotomy, it is of primary importance to distinguish between tracheal obstruction by direct pressure of an aneurysm, and laryngeal obstruction, the result of disease in the larynx, or spasm, or paralysis of the laryngeal muscles, which may be caused by the pressure of an aneurysm or other tumour on the vagus and recurrent nerves. In most cases the diagnosis by the aid of the laryngoscope is not difficult. If you see, on laryngoscopic examination, a perfectly healthy larynx and a widely opened glottis during inspiration, you may be quite sure that the stridulous and difficult breathing is the result of an obstruction below the larynx. If, unfortunately, you cannot use the laryngoscope, and so are unable to get a view of the larynx, auscultation will help you to distinguish between stridor which is the result of obstruction in the larynx, and stridor the result of obstruction in the trachea. Laryngeal stridor is louder over the larynx than over the trachea, just above the sternum. If you apply the stethoscope over the larynx, and then over the trachea, you may often satisfy yourself that the noise is much louder over the larynx than it is just above the sternal notch. Auscultation over the spinous processes of the vertebræ is often of great assistance. I have repeatedly in the wards pointed out to you how marked is the difference in the two classes of cases. In the case of laryngeal obstruction you hear the stridor most distinctly over the spinous process of the middle cervical region. If, on the other hand, the stridor is the result of obstruction of the trachea just above its bifurcation, you will hear it much more distinctly on auscultation over the upper dorsal spinous processes than over the middle cervical region.

When an aneurysm, by pressing on the vagus and recurrent nerves, has caused paralysis of the laryngeal muscles, and consequent difficulty of breathing, tracheotomy may afford great relief, as it did in the case recorded in Chapter XXV. In that case there was bilateral paralysis of the abductors of the larynx, almost complete closure of the glottis, and great dyspnœa in consequence. We then discovered that there was aneurysm of the transverse aorta. Now, although the aneurysm was in the chest, the obstruction was really in the

larynx, the result of paralysis of the laryngeal muscles; and Sir William Fergusson, at my request, performed tracheotomy which give great temporary relief; whereas tracheotomy in the case represented in the photograph would have been utterly useless. The opening of the trachea would have been above the obstruction, and therefore no relief would have been obtained by the operation.

The main point of treatment in all cases of internal aneurysm, which are beyond the reach of surgical aid, is to keep the patient absolutely at rest in bed on a nutritious but restricted diet, the object being to keep the circulation as quiet as possible. When there is much cough a combination of chloral and morphine, in small doses, is very useful. We are giving our patient that mixture at the present time, and already the cough has been relieved, partly by the rest and partly by the sedative medicine. We shall keep him in bed as long as he will submit to that wholesome restraint. In addition to this we are also giving him iodide of potassium. Probably you know that several cases have been recorded, in which rather large doses of iodide of potassium, beginning with five or ten grains, and going on to doses of fifteen or twenty grains three times a day, have appeared to do considerable good in cases of internal aneurysm. I confess that I am somewhat sceptical as to the influence of iodide of potassium in cases of aneurysm of the aorta. I do not see very clearly how the iodide can be beneficial. We know, however, that some of our most efficacious remedies act in a way which it is not easy to explain. Therefore, I have thought it right in this case, and in some others, to put the patient upon iodide of potassium; we are now giving ten grains, and, by-and-by, we shall double the dose. We know that cases of aneurysm treated with rest in bed, and a suitable diet, almost invariably improve for a time. On the other hand, if a patient finds himself much benefited by treatment in hospital, and, getting impatient of restraint, returns to his work and takes active exercise, there is almost always an aggravation of the symptoms, and the patient comes back in a short time, much worse than before. There can be no doubt that the beneficial influence of rest in these cases is very marked

indeed; and I believe that some of the good results which have been attributed to iodide of potassium, and other drugs, in the treatment of these cases have really been due more to rest and suitable diet than to the medicines which have been simultaneously given.

SECTION II.

ABSTRACT OF A CLINICAL LECTURE ON THORACIC ANEURYSM.[1]

The subject of the lecture was Aneurysm of the Thoracic Aorta, two patients being then in the wards, one suffering with aneurysm, and the other presenting some symptoms of aneurysm, but not so definitely as to render the diagnosis quite certain.

CASE I. *Signs of Aneurysm of the Arch of the Aorta—Heaving Impulse against the Left Clavicle—Bellows Murmur—Inequality of Pulse at the two Wrists—Enlargement of Superficial Veins of Chest.*

The first case that the lecturer spoke of was one in which there was no doubt as to the diagnosis. The patient was a labourer, aged 45, admitted January 4. His general health had usually been good, and, except an attack which he calls inflammation of the chest, sixteen years before, he had had no illness until the present. He had been intemperate until twelve months ago, when, after a drinking bout, he first began to suffer from his present symptoms. He first had sharp pains in the region of the left nipple, over a space of about three inches, occasionally extending lower, to the epigastrium. Fourteen days later he had pain down the left arm, sometimes associated with the pain in the chest and sometimes alone. The pain was not made worse by breathing deeply, but was ncreased by exertion.

When admitted he had the pain as above mentioned, both

[1] The above lecture was published in the *Medical Times and Gazette* (March 15, 1852) from the notes of an able and accurate reporter, now a very eminent physician.

in the region of the heart and down the arm. He had lost flesh, and his appetite was bad. Another striking peculiarity was the change in the voice. It was feeble and cracked, more like the voice at puberty. The change in the voice he said had lasted ten months. He had no cough, no difficulty in breathing, and none in swallowing. Therefore, it was probable that there was no pressure either on the œsophagus or on the trachea.

On examining the chest it was found to be of the barrel shape, characteristic of extensive emphysema of the lung. There was comparatively little expansion, which was no doubt due to the emphysema. There was a remarkable difference in the pulse at the two wrists; the right was full and throbbing, and almost of the character of the pulse in cases of aortic regurgitation. The left pulse was small and feeble. This was an important sign to note, and probably showed some obstruction to the flow of blood into the left subclavian artery. Another circumstance pointing to interference with the circulation of this limb, was that the veins of the arm and of the left side of the chest also were much fuller than on the right side. There was at first no abnormal pulsation detected, but after a week or so, pulsation was easily made out at the sternal end of the left clavicle—a heaving impulse occurring at each systole of the heart. This was not observed on his admission. There was dulness on percussion in the same position. Without any great pressure a thrill was felt by the fingers placed over the subclavian artery, and a distinct systolic (rough blowing) sound was heard at the base of the heart, and upwards to the top of the sternum. Near the sternal end of the left clavicle the roughness of the sound was greater than over the base of the heart. There was also some indistinctness of the second sound of the heart, and sometimes the lecturer believed he could detect a soft, diastolic sound. Dr. Johnson then spoke of the character of the pulse in the right wrist; it was like, very like, the pulse of aortic regurgitation.[1] The respiratory sounds were more feeble over the right than over the left

[1] See clinical lecture by Dr. Johnson, *Medical Times and Gazette*, February 8, 1862, p. 133. Also Chapter XXXIV., p. 499.

lung; but no great importance was to be attached to this, as in most cases the respiratory murmur is normally less audible in the right than in the left lung; and as regards the apices, this was important to bear in mind in the diagnosis of incipient phthisis; a feeble respiratory murmur at the *right* apex is much less significant of deposit in the lung than a comparative feebleness at the *left* apex.[1]

The peculiarity of the voice was probably due to pressure on the recurrent nerve. It is not uncommon in such cases to get some alteration of voice, and the breathing is often stridulous. In this case, however, the breathing was easy and noiseless.[2]

Dr. Johnson said there could be little doubt that there was an aneurysm of the aorta, near the giving off of the subclavian artery. The position of the pulsation; the rough bruit in the same place; the obstruction to the circulation in the veins on the left side of the chest; the small pulse in the left radial artery; the symptoms of pressure on the recurrent laryngeal nerve, all pointed to the aorta near the origin of the subclavian artery, as the seat of disease. This was certain; but there was one doubtful point—viz. whether the semilunar valves of the aorta were incompetent or not.

The patient was placed on good diet; was kept quiet in bed; and took fifteen minims of the tincture of digitalis, and fifteen minims of the tincture of the perchloride of iron, three times a day. A belladonna plaster was applied over the region of the heart.

CASE II. *Signs of Insufficiency of Aortic Valves and of Disease of Coats of Aorta—Double Bellows Sound at Base of Heart—Rough Systolic Sound at Top of Sternum—Thrill in Left Subclavian—Pulse at Right Wrist Full and Collapsing—On Left Side comparatively Small and Feeble—Sudden Death—Post-mortem Examination.*

In this case there was clear evidence that the coats of the aorta were diseased, but whether there was an aneurysm or

[1] See Chapter XXXII.

[2] This case occurred in the dark age, before the laryngoscope had come into use, and we could only guess at the condition of the larynx.

not was doubtful. The patient was a baker, a pale, but generally healthy man, aged 53. He had been intemperate, but he had never had gout or rheumatism, nor in fact any serious illness, until two months ago, when his present ailment began. His first symptoms were cough, shortness of breath, pains in the back, and palpitation, all worse on exertion. He kept at work, however, until January 11, when being worse and his legs swelling, he sought and obtained admission into the hospital.

When admitted he was suffering a good deal. He had much dyspnœa and cough, and his legs were swollen from the ankles to the knees. He looked pale and feeble, and complained of pain over the region of the heart. There was no albumen in the urine. On examining the chest it was found that the heart's impulse and the extent of dulness were much increased, showing that the heart was enlarged. A distinct double bellows sound was heard over the base of the heart. The diastolic sound was best heard at the base, but the systolic sound was louder at the top of the sternum. On the least touch there was felt a strong thrill in the left subclavian artery. There was also in this (as in the other case) a remarkable difference in the pulse at each wrist; that on the right side being twice as strong and full, and indicating unquestionably aortic regurgitation. There was no abnormal pulsation against the walls of the chest.

In this case, the lecturer said, we have evidence, first, of disease of the aortic valves as indicated by the double murmur at the base of the heart and the collapsing pulse; and, secondly, of disease of the aorta and roughening of its inner coat, at the giving off of the left subclavian artery, as shown by the strong thrill in the left subclavian, the small and feeble pulse in the left radial artery, and the rough systolic sound at the top of the sternum. There was no positive evidence of aneurysm. An atheromatous condition of the aorta would be enough to impede the flow of blood into the subclavian artery, and a similar condition of the aortic valves, or perhaps dilatation of the aorta at its beginning, would suffice to produce incompetence of the valves. There was no abnormal pulsation

against the walls of the chest, but this might, as in the other case, occur after a time.

The treatment, so far as success in such a case could be hoped for, was successful. All his urgent symptoms were relieved. He was, when admitted, in much distress from dyspnœa, the heart being unequal to its work; the lungs were congested; and for the same reason (impaired action of the heart) there was œdema of the legs. There was no albumen in the urine from congestion of the kidney, a frequent result of retarded circulation, as pointed out in a previous lecture.[1] He took, as a hydragogue cathartic, drachm doses of compound jalap powder, and under the use of this, and rest in bed, the swelling of his legs subsided, and his breathing became easier. The compound spirit of æther was also given occasionally as an anti-spasmodic. Brandy and good diet were allowed, and on the third day the tincture of the perchloride of iron was commenced. The man was much better. The dyspnœa and the dropsy had entirely passed away. Of course the organic conditions giving rise to these symptoms remained pretty much the same.

The above comments on this case were made by Dr. Johnson in his lecture delivered on February 11. Since then the patient has died and the body has been examined. On February 27 he was dressed and sitting by the fire with some other patients, when he rose from his chair, and without saying a word suddenly fell; his face became dark-coloured; he made a few gasps, and was dead before the house-physician could reach him. On examination of the body twenty-four hours after death, the heart was found much enlarged, and weighed twenty-two and a half ounces, the enlargement being chiefly due to dilatation of the left ventricle and hypertrophy of its walls. The aortic valves were thickened, quite to their margins. The arch of the aorta and a great portion of the thoracic aorta were much dilated; the inner circumference of the vessel just above the valves was five inches; the dilatation gradually diminished towards the termination of the thoracic aorta. The whole inner surface of the dilated vessel was

[1] See Chapter XLV., section viii.

roughened by atheromatous, and in some parts by calcareous, deposit. The innominate artery was dilated and its walls diseased; the orifice of the left carotid was narrowed by atheroma, but its walls beyond were smooth and healthy.

The left subclavian was slightly dilated at its origin, and its inner surface was very uneven, with atheromatous deposit for about three inches from its commencement. The diseased condition of this vessel probably caused the thrill which was felt over it, and the comparative feebleness of the pulse at the left wrist. There had been no rupture of the aorta or of its branches. The left pleura contained about two pints of liquid. The brain was healthy. It was remarked at the *post-mortem* examination that in most cases of sudden death from incompetence of the aortic valves the left ventricle is found distended. In the absence of any discoverable obstruction in the pulmonary vessels the explanation of the empty ventricle in this case was not obvious.

In reference to the treatment of aneurysm, Dr. Johnson said the object was to place the patient under such circumstances as to favour the filling of the sac with coagula. To attain this object the surgeon arrests or retards the flow of blood by ligaturing the artery, or by pressure. In cases of thoracic aneurysm there was no way of moderating the flow of blood through the artery, except by reducing that of the whole circulation. We attempt to moderate the circulation, and to induce consolidation, by prescribing that the patient should avoid excitement, both mental and physical, and by ordering a nutritious diet, to improve the health and the condition of the blood. The lecturer said he believed it was desirable that the patients should have a nutritious but a somewhat restricted diet, the object being to improve the condition of the blood, but at the same time not to excite the circulation. Another indication was to relieve pain, and to do this we are sometimes justified in abstracting blood. The treatment of aneurysm by starvation and depletion, known as Valsalva's treatment, had for its object, not merely to relieve pain, but also to cure the aneurysm, by retarding the flow of blood, and thus increasing its tendency to coagulation. There is, however,

one great objection to this, even as a theory—viz. that, the weaker the circulation, the more rapid it becomes; and then the blood being impoverished, and especially the quantity of solids being diminished, the tendency to coagulation is lessened. Further, Valsalva's treatment, by rendering the patient weaker, reduces the power of resistance in the tissues, and therefore the already diseased artery is likely to become more rapidly dilated. In spite of this, however, there were, the lecturer said, some cases which appeared to show that at least this treatment did no harm. The following case was a most remarkable one, on account of the long duration of the symptoms, and the very frequent venesections which were practised during its progress.

CASE III. *Aneurysm of the Aorta, treated by very frequent Venesections—After the Symptoms had continued Fifteen Years, Death at the advanced Age of Sixty-nine, without Rupture of the Aneurysm.*

Dr. Johnson then read the notes of the case, taken in the year 1840. The patient died four years afterwards. A cast of the chest, and also a preparation of the aneurysm (removed after death) were shown to the class, and they are now in the King's College Museum.

The patient was a labourer, 65 years of age, when Dr. Johnson first saw him (in 1840). He had then been ill ten years. In 1830, after violent exertion, the patient felt a sharp pain in the chest. This, the occurrence of a sudden sharp pain in the chest, was, the lecturer said, a common symptom at the onset of such cases. He suffered pain in the chest, attended by pulsation, for two years, and then he observed a swelling on the right side of the sternum, which continued to increase. In 1840 this swelling had become so large that it extended from the sternum to the axilla, and from the second to the sixth rib, and projected forwards in a conical form to the extent of about four inches. The right side of the sternum appeared thinned, and extended some way over the tumour. The ribs could not be felt over the aneurysm; they had evidently been absorbed by pressure. The walls of the tumour were firm and but slightly yielding on pressure; on grasping

it with the hand it was felt to expand in all directions during the systole of the heart. The body inclined towards the side of the tumour, and the patient walked in a bent position. There was no blowing sound over the aneurysm, but two sounds similar to the normal heart-sounds were heard. The apex of the heart beat between the fifth and sixth ribs, and about two inches external to the mamma.

Three or four years before, he had expectoration of blood. He never had much dyspnœa, except on walking quickly. He had occasional headaches, which were relieved by bending his head and body forwards. Respiration was heard all over the chest, except over the position of the tumour.

In February, 1833, the patient was bled from the arm to twelve ounces, and this was repeated every four days for nine months; then ten ounces were taken every five days. When Dr. Johnson saw the man in 1840 he had been bled one hundred and fifty-seven times. After this the bleedings were repeated only nine or ten times. The object of the bleedings at first was to cure, but subsequently merely to relieve pain. Except that he always looked pale, the patient had good general health.

He died in January, 1845, at the age of 69 years, not from the bursting of the aneurysm, but apparently from sudden syncope while lying in bed. Dr. Johnson made a cast of the chest, which gives a good idea of the external appearance of the tumour, and the aneurysm itself was removed and preserved as a dried specimen. The aneurysm arose from the right side of the ascending aorta; it was of a size sufficient to contain about two pints of liquid. When recent it was pretty thickly lined by laminated coagula. It communicated with the aorta by an oval opening about an inch long and half an inch wide. There had been no rupture of the sac, nor any escape of blood. The coats of the aorta were in a state of atheromatous and calcareous degeneration, the whole arch was more or less dilated, and there was a small aneurysmal dilatation at the origin of the left carotid.

The lecturer stated that this case occurred in the practice of his friend, the late Mr. Newington, of Goudhurst, in Kent, to whom he was indebted for the opportunity of seeing the

patient from time to time, and also of obtaining the specimen after death. He had already stated some reasons for not adopting, as a rule, a lowering plan of treatment in cases of internal aneurysm. He believed, however, that in this case the repeated bleedings had prolonged the patient's life. It was a most remarkable circumstance that a patient should have had unequivocal symptoms of aneurysm of the aorta for a period of fifteen years, and that he should have died at the advanced age of 69, and then not from the direct consequence of the aneurysm. The number of bleedings, and the great duration of life with so formidable a disease, were both most unusual, and it seemed highly probable that they were related to each other as cause and effect.

SECTION III.

The two following cases of aneurysm of the thoracic aorta present some features of special clinical interest.

CASE I. *Aneurysm of the Aorta encroaching upon the Pulmonary Artery and so obstructing the Circulation as to cause Tricuspid Incompetence and Engorgement of the entire systemic Venous System.*

E. N——, æt. 38, a waiter, was admitted under my care first on April 18, 1864 (Case Book, vol. xx. 29). Habits described as temperate. For the last four years has suffered from winter cough, and during the last three months he has been troubled by palpitation. The lung sounds are normal. Cardiac dulness extended; the apex beat is two inches below the nipple. A loud systolic apex murmur, which is faintly audible at the back. Pulse small. The liver extends below its normal position, and is painful and tender on pressure. Urine normal. Considerable dyspnœa and palpitation, with occasional cough and vomiting. He gradually improved with rest and a quinine and iron tonic, and was discharged on May 11.

On the following September 25 he was readmitted, suffer-

ing from palpitation and dyspnœa, with a considerable amount of general œdema. The urine was high-coloured, scanty, and albuminous to the extent of one-eighth. There were no abnormal lung sounds. The heart's apex was in the same position as before, with a loud systolic apex murmur. Pulse 150 small and feeble.

He was ordered an ammonia and æther mixture, and an occasional dose of pulv. jalapæ co. He complained of pain at the scrobiculus cordis, which was reduced by dry cupping. The general œdema continued; the face and lips became very livid. He had much headache. The lung sounds were, as a rule, quite normal. On one occasion only was there a note of rather fine crepitation at the base of the right lung. The pain at the pit of the stomach, the palpitation, and dyspnœa increased, with intense blueness of the lips, while the lungs remained clear. He died on October 12.

On inspection there was found an aneurysm of the ascending aorta, which had pressed upon and greatly narrowed the canal of the pulmonary artery. The mitral valve and orifice were normal, but the right ventricle was so much dilated and hypertrophied that it formed the apex of the heart. The murmur at the apex was now seen to have been the result of tricuspid regurgitation, the valve having been rendered incompetent by dilatation of the orifice. The condition of the right ventricle was explained by the compression and narrowing of the pulmonary artery. There was no engorgement of the lungs. The absence of pulmonary engorgement, which, with the signs of systemic venous fulness, albuminuria, and general œdema, it was difficult to explain on the supposition that mitral incompetence was the starting-point of the malady, was intelligible now that the obstruction was seen to be at the entrance and not at the exit of blood from the lungs. The crepitation at the base of the right lung, which was noted on one occasion, was no doubt a result of exudation from the gorged systemic bronchial veins and capillaries (see Chapter IV., on *Retrograde Engorgement of the Blood-vessels*). All the abdominal viscera, the liver, spleen, and kidneys, were much congested. Many of the results of systemic venous engorgement in this case were similar to those observed in the case of tricuspid incompetence

recorded at p. 51. In that case, however, 'the lungs were much engorged.' The difference in the condition of the lungs in the two cases is probably accounted for by the fact that, in consequence of the extreme constriction of the pulmonary artery, the supply of blood to the lungs was much diminished. So that, although there was some evidence of bronchial venous and capillary engorgement, there was none of the secondary pulmonary congestion which occurred in the other case.

CASE II. *Aneurysm of the Aorta with Paralysis of the Abductor of the Right Vocal Cord—Sudden Death.*

Major-General S——, æt. 59, was sent to me by Dr. Philson, of Cheltenham, on May 19, 1886. The patient said that, during the last three weeks, he had suffered from cough and some shortness of breath on exertion. These symptoms had come on suddenly, after a ride of two miles on a tricycle; a kind of exertion to which he had not been accustomed. The area of cardiac dulness extended upwards as high as the third rib. The sounds of the heart were heard over the dull space, but there was no abnormal pulsation or bruit. The voice was clear, but on inspection of the larynx, the right vocal cord was seen to be motionless in the middle line—that is, in the phonatory position; so that, the other cord moving freely, the voice was unchanged, notwithstanding the paralysis of the right abductor muscle. The radial arteries felt hard, and were probably, therefore, atheromatous. The pulse was equal in the two wrists.

In writing to Dr. Philson I said that, although in the absence of direct physical signs of a pulsating tumour, the evidence was not conclusive, the paralysis of the right abductor muscle excited my suspicion that an aneurysmal dilatation of the aorta was pressing on the right recurrent nerve. This opinion naturally alarmed the patient and his friends, and on June 11 he was taken to another physician, who, not using the laryngoscope, assumed that the reported laryngeal palsy had passed away, because the voice was natural. I have before stated that the voice was not affected by paralysis of the abductor, as it would have been if the adductors had been paralysed. This consultant, however, believing that the

laryngeal palsy had been got over, and in the absence of any trouble in swallowing, thought that the symptoms might be due to other causes than aneurysm.

Dr. Philson wrote to me on October 30, that the patient had remained under his care until the month of August, during which time the dyspnœa on exertion got gradually worse. Since then he had been staying with friends in Ireland.

Dr. Philson adds: 'I have just heard that he died suddenly about ten days ago. He was on the sofa and had taken some breakfast, when he was seized with faintness and so passed away. Doubtless, rupture of the sac occurred, with internal hæmorrhage. No *post-mortem* was permitted. Your opinion was evidently correct, though the case was obscure and puzzling.'

The chief interest of the case is in the illustration which it affords of the value of the laryngoscope in the diagnosis of some cases of aneurysm. The laryngeal palsy afforded conclusive evidence of pressure on one recurrent nerve, and the other symptoms rendered it at least highly probable that a dilated aorta was the cause of the pressure. Without the aid of the laryngeal mirror I could have formed no definite opinion as to the nature of the disease.

SECTION IV.

In the present section I propose to give the history of some cases of aneurysm of the abdominal aorta, which, with reference to diagnosis, were of special interest. I shall then briefly refer to the treatment of abdominal aneurysm.

CASE I. *Aneurysm of the Abdominal Aorta bursting behind the Peritoneum and causing enormous Swelling of the Abdomen.*

On September 17, 1872, I saw at my own house, Mr. J. T——, aged 35, who, for fifteen months, had suffered from severe pain in the back, and latterly in the left testicle. I found a pulsating tumour in the epigastrium, over which a loud bellows sound was heard. The impulse of the aorta was distinctly heard over the lower dorsal spinous processes. Two

days afterwards, I saw him at his own house, and was told that the night before, while lying on the sofa, he had a sudden increase of pain in the left side of the abdomen, and appeared to be dying. He now complained of tenderness in the left loin, where I found a pulsating fulness, over which a bellows sound was audible. I concluded that the aneurysmal sac had given way, and a false aneurysm had formed in the connective tissues behind the peritoneum. On September 26 I again saw him, and found that the pulsating tumour extended from the margin of the ribs to the crest of the ilium, and as far as the median line of the abdomen. He died a few days afterwards, having been seen in the interval by Sir James Paget and Sir William Gull, who, I was told, confirmed my diagnosis.

The enlargement of the abdomen in this case was greater than I have ever seen in any similar case. In the absence of a *post-mortem* examination, it is impossible to say whether the effused blood was entirely behind the peritoneum or whether, as is probable, some had escaped into the cavity of the abdomen.

The following case, it will be seen, was of the same nature as the preceding.

CASE II. *Aneurysm of the Abdominal Aorta bursting behind the Peritoneum.*

Mr. H. J. T——, æt. 28, barrister's clerk, was first seen by me on July 23, 1866. About fifteen months before, he got wet while riding on an omnibus, and since then he had had more or less constant pain in what he supposed to be his kidneys. In December he got temporary relief from cupping on the loins. Placing him on his back, I felt a pulsating tumour in the epigastrium, over which there was a loud and rough murmur. No pulsation or murmur was audible over the back. I saw him again on July 30, when I found the condition unchanged. After this second visit I wrote to the patient's employer, the late Dr. A. J. Stephens, Q.C., the eminent ecclesiastical lawyer, and said that if he would call on me I would explain to him the serious nature of his clerk's illness. Dr. Stephens soon came, and I told him that the case was one

of abdominal aneurysm, and that, although the patient might live for an indefinite period, his sudden death might occur at any moment. From that time I saw no more of the patient, nor did I hear of him until December 28, when I received from Dr. Stephens a letter from which the following is an extract :—

'As *five physicians* expressed a positive opinion that you had given an erroneous opinion respecting the illness of my late clerk, I deem it but an act of justice to forward you the enclosed, which I only received this morning from Mr. John H. South, Ex-President of the College of Surgeons.' Mr. South's letter, which was dated December 25, 1866, was in substance as follows :—

He had gone the day before to St. Bartholomew's Hospital to inquire for the patient, and learnt that he had died on December 14. He then saw the case-book and the gentleman who had made the *post-mortem* examination. The case had been thought very obscure. About eight o'clock on Sunday evening the patient felt a sudden tear internally and became very faint. The pain was relieved by opium. On the Monday evening he had a second tearing sensation, and on that or the following day a large swelling was observed running down the left flank, and he became very pallid, or, as the ward sister said, 'like marble,' and on the Thursday he died.

On inspection, two aneurysmal sacs were found at the back of the aorta, just below the diaphragm and close together; the upper sac about the size of a walnut, the lower that of an orange. The lower sac had burst, probably, on the Sunday night *behind* the peritoneum, and the blood had found its way in front of the kidney through the flank into the pelvis ; and low down there was a little crack in the peritoneum through which a little blood had escaped into its cavity. The mass of blood behind was about two or three pounds. It was thought probable that the second tear he felt might have been the rent in the peritoneum from the distension caused by the blood behind. Mr. South adds : 'You will thus see that the doctor who diagnosed aneurysm was right.'

In the following case I did not detect the nature of the disease until after the patient's death.

CASE III. *False Aneurysm of the Abdominal Aorta pressing on the Lumbar Plexus and causing Violent Pain.*

George V——, aged 39, was admitted into the hospital under my care in September 1877. For several months he had suffered from very severe pain in the left loin and thigh. Beneath the false ribs on the left side there was a soft pulsating tumour, over which a bellows sound was distinctly audible. I took it to be a case of malignant tumour, so vascular as to give rise to the pulsation and the murmur; the pain being caused by the pressure of the tumour upon the lumbar plexus of nerves. In spite of the free hypodermic use of morphine, the pain continued unrelieved, and the poor man died, worn out by the intense suffering, on November 4. The tumour was found to be a false aneurysm. There was a small opening at the back of the aorta just below the diaphragm; and the blood, having forced its way behind the peritoneum, had quite surrounded the left kidney. There had been no escape of blood into the cavity of the peritoneum.

CASE IV. *Abdominal Aneurysm quickly fatal after bursting behind the Peritoneum.*

On December 9, 1881, I saw, in consultation with Dr. Poulain, Mrs. S——, æt. 50. Five days before, she had been suddenly seized with severe pain in the left loin, during the night. The pain extended to the thigh, and was attended with vomiting and irritation of the bladder. The urine was reported to have been high-coloured, and to have contained blood and pus. The specimen which I saw was pale, and contained some pus-cells and squamous epithelium. There was a trace of albumen, but no blood. During the last twelve hours the pain in the loin had subsided.

The history and the symptoms seemed consistent with the diagnosis of renal calculus. There was no palpable tumour in the loin.

I did not see the patient again, but a few days afterwards I heard of her sudden death. Her daughter wrote to me the following particulars:—

On the evening of the 9th, the day of my visit, and the

following day, she appeared much better. She took plenty of nourishment and slept a good deal. At 2 A.M. on the 11th 'she had a kind of fit,' but it was over in a few moments. She was quieter afterwards and in no pain; towards morning she got very restless, but was quite conscious and able to take food. This went on until about 6 A.M., when she became slightly convulsed again, and in a moment she was gone.

There was no inspection of the body, and I received no further particulars; but the facts, now stated, clearly show that the case was one of abdominal aneurysm. The sudden onset of pain was caused by the aneurysm giving way behind the peritoneum. The convulsive attacks between 2 and 6 A.M. of the 11th and the sudden death were the result of hæmorrhage, behind or into the cavity of the peritoneum. The hæmaturia was probably caused by the pressure of the effused blood on the renal vein. In the analogous case of George V—— (p. 581), 'the blood had completely surrounded the left kidney;' and in the case of H. J. T——(p. 579), the blood had 'passed in front of the kidney,' and so would probably compress the renal vein.

CASE V. *Abdominal Aneurysm bursting behind the Peritoneum, and soon fatal.*

Mr. H. J——, æt. 38, hair-dresser, was seen by me, in consultation with Mr. J. G. Clark, on January 8, 1882. Two years before, he had a bad fall; since which he has suffered more or less from backache. Five weeks ago he was suddenly seized with pain in the left loin, but unattended by vomiting or irritation of the bladder. The pain during the last five weeks has sometimes been absent, and he has been able to go out occasionally in a Bath-chair. At the time of my visit he was moaning and tossing about the bed in great agony. The pain extended from the left loin to the groin. No tumour could be felt, but a rough blowing murmur was heard on placing the stethoscope just below the left false ribs and behind, to the left of the upper lumbar spinous processes. The urine is now normal, but Mr. Clark reports it to have been albuminous at the onset of the pain.

I expressed a very decided opinion that the aorta had

given way behind the peritoneum, and of course the prognosis was most unfavourable. On January 13 I received from Mr. Clark the following statement :—

'The patient whom you saw with me on the 8th instant died suddenly on the morning of the 10th. On turning suddenly in bed on the morning of the 9th, he felt something give way in his abdomen, followed by an intense burning heat in the left lumbar region. When I saw him about ten minutes afterwards he was blanched and almost pulseless, and there was a distinct swelling to be felt in the left loin. He said he felt a sensation in his belly like a small fountain playing. Later in the day he vomited several times, each time giving him violent pain in his belly, and rendering him more blanched and his pulse weaker. The tumour in the lumbar region continued to get larger during the day, until it became quite visible on looking at the abdomen. The tumour did not pulsate visibly or palpably, and no murmur could be heard over it. The family being Jewish, no autopsy was permitted.'

In this case the sudden onset of pain five weeks before death was, no doubt, caused by the giving way of the aorta behind the peritoneum and the formation of a false aneurysm, while the sensation of something giving way, followed by burning pain and the appearance of swelling in the loin, on the morning after my visit were probably the result of a still further breaking down of the tissues, and the escape of a larger quantity of blood, perhaps into the cavity of the peritoneum.

An inspection of the body would pretty certainly have revealed much the same appearances as were found in the case of H. J. T—— (p. 579).

The preceding cases will serve to illustrate most of the symptoms and results of abdominal aneurysm.

Amongst the symptoms of abdominal aneurysm, pain in the back, more or less severe, is one of the most constant. When persistent backache is complained of, the question of aneurysm should always be considered. Palpation and auscultation of the abdomen will, in most cases, enable us to form a diagnosis ; care being taken not to mistake a merely throbbing aorta, with a soft blowing produced by the pressure of

the stethoscope, for the expansive pulsation and the usually rough sound of an aneurysm.

The giving way of the aorta and the formation of a false aneurysm behind the peritoneum, are usually attended by sudden severe pain in the left loin and more or less collapse, resulting from the conjoint influence of the pain and the escape of blood from the circulation. The pain may be mistaken for an attack of renal colic. In most cases a continued escape of blood is fatal within a few hours or days. It can rarely happen that a patient survives the giving way of the aorta for so many weeks, as did the man George V—— (p. 581).

The pulsation and the murmur which are almost constantly observed in the epigastrium or in the left loin when a false aneurysm has formed behind the peritoneum, may sometimes, but not constantly, be heard at the back, either over the lumbar spinous processes or somewhat to the left of the median line.

CASES VI. and VII. *Two Cases of Abdominal Aneurysm in which the Liver lay in front of the Tumour and rendered the Diagnosis difficult.*

I have met with two cases in which the diagnosis of abdominal aneurysm was rendered somewhat doubtful by the liver being placed in front of the aneurysm. The first case occurred in the hospital, under the care of my esteemed teacher and colleague, the late Dr. George Budd, when I was house-surgeon, in 1843. A man, D. M——, aged 38, had suffered from pain in the back for three years, and for a year there had been pain in the right shoulder. A tumour, having the form and position of the liver, extended as low as the umbilicus. The hepatic dulness extended as high as the right mamma, and the lower ribs bulged outwards. Over the whole abdominal portion of the tumour a strong heaving impulse was felt, and a loud, rather rough murmur was heard. The murmur was also heard at the back over the lower dorsal spinous processes. After remaining some days in the hospital he died quite suddenly. I saw him, with my friend the late Dr. Russell, of Birmingham, who was then house-physician, immediately after his death; when we found that the hepatic swelling had disappeared, and

had been replaced by a feeling of fluctuation over the whole abdomen. The abdominal cavity was filled with blood, which had escaped from a ruptured aneurysm of the aorta, large enough to hold at least a gallon. The liver had become flattened and expanded over the surface of the aneurysm, so that along the longitudinal fissure the hepatic substance was not more than half an inch thick.

The other case was one the history of which I published in the *Pathological Transactions*, vol. x. p. 99. A carpenter, aged 37, had suffered from pain in the back for a year before his admission into the hospital, December 31, 1858. About seven weeks before admission he first detected a swelling at the pit of the stomach. There was a pulsating tumour at the epigastrium, rather to the left of the median line; above, it seemed to pass beneath the ribs, but below it had a well-defined margin about an inch above the umbilicus. Its anterior surface was rounded and convex, and it distinctly descended during a deep inspiration. No murmur was at first heard over the tumour or over the spinous processes at the back, but on January 13 it was first noticed that a bellows sound was audible, not over the most prominent part of the tumour, but at its upper margin, just below the ensiform cartilage. The sound continued to be heard from that date. The tumour appeared to increase in size, until it could be just covered by the half-closed hand. On February 6, while he was sitting up in bed, he suddenly vomited a large quantity of fluid blood, his face became blanched, and he quickly died. After death, the tumour in the abdomen could not be felt. On opening the abdomen, it was found that the tumour which had been felt during life was formed by the lower margin of the right lobe of the liver. This portion of the liver was indurated by a deposit which was probably a syphilitic gumma, though he denied having had syphilis. The morbid mass was separated from the bulk of the liver by a deep fissure, evidently the result of atrophy of a portion of hepatic tissue. The liver weighed 3lb. 14ozs. The semi-detached portion of liver before mentioned lay in front of an aneurysm, which sprang from about the middle of the abdominal aorta. It was about the size of the double fist; its pressure had caused erosion of the

bodies of the second and third lumbar vertebræ, and its cavity communicated by a round opening, about two or three lines in diameter, with the lower portion of the duodenum. The stomach and the small intestines contained much clotted blood.

The phenomena in this case were very perplexing. The persistent backache, the pulsation, and the bellows sound led me to the diagnosis of an aneurysm, but until the abdomen was opened I could not explain the movable tumour which descended at each deep inspiration.

Treatment.—The spontaneous cure of an internal aneurysm is sometimes effected, the sac becoming consolidated by fibrinous coagula. The best method of favouring this desirable result consists in securing absolute rest in bed for a lengthened period of from two to three months, while a nutritious but restricted diet is allowed, in accordance with the method which was successfully practised by the late Mr. Jolliffe Tufnell. The rest must be absolute; the patient neither leaving his bed nor even sitting up for any purpose. The diet is restricted to about ten ounces of solid food, of which one-half is meat or fish, and eight ounces of liquid *per diem*. The bowels to be carefully regulated so as to avoid constipation and straining. In carrying out this treatment drugs are given only to meet particular symptoms: opiates to relieve pain, laxatives to obviate constipation, &c.

Surgical.—Some cases of abdominal aneurysm have been successfully treated by pressure either on the proximal or the distal side of the sac, according to the position of the aneurysm. In other cases death has resulted from pressure on a portion of bowel causing inflammation and sloughing.

Galvanopuncture, with the object of promoting coagulation within the sac, is reported to have been successful in some cases.

The introduction of foreign bodies, such as iron-wire, catgut, and horsehair, for the purpose of promoting a deposit of fibrine upon the foreign body, has been practised in a few cases of thoracic aneurysm, but hitherto without success; and in some cases the death of the patient has been hastened by

the operation. The chief dangers to be apprehended from the puncture of an aneurysm and the introduction of a foreign body are the occurrence of inflammation and sloughing of the sac, and the detachment of some recently deposited coagula which, passing into the circulating current, may form embolic plugs in the cerebral or other arteries and capillaries.

CHAPTER XXXIX.

A LECTURE ON BACKACHE, AND THE DIAGNOSIS OF ITS VARIOUS CAUSES, WITH HINTS ON TREATMENT.

The Various Causes of Backache—Overstrain of Dorsal Muscles from Excessive Weight in the Abdomen—Dietary for Excess of Fat—Cases of Muscular Dyspepsia—Treatment—Growing Pains—Sudden Lumbago from a Strain or Rupture or Cramp of Muscles—High-heeled Boots—Fatigue of Weak Muscles—Cold and Damp—Treatment—Aneurysm of the Aorta—Cancerous Glands in the Abdomen—Cases—Diagnosis—Kidney Disease and Displacement—Gastric Ulcer—Uterine Disease and Displacement—Disease of the Vertebræ and of the Spinal Cord.

GENTLEMEN,—The careful study of a particular prominent symptom, with a view to ascertain the various causes from which it may arise, is often of great practical utility. I propose now to make this inquiry with regard to the very common symptom *backache*. A patient comes to us complaining of pain in the back; and, as an essential preliminary to the suggestion of a remedy, we endeavour to ascertain the precise seat, the nature, and the cause of the pain.

I will first refer to the more common causes of bachache, and afterwards to those which are of less frequent occurrence.

In the great majority of cases, the pain of backache has its seat in the muscles, and is a simple result of strain or over-fatigue of the lumbar and erectores spinæ muscles and tendons. Everyone must have had personal experience of the pain and soreness resulting from muscular fatigue consequent on any unaccustomed or unusually prolonged or violent exertion or strain. One remarkable feature of the pain which results from excessive muscular exercise is that, while it may continue more or less during rest in bed, it is usually much increased by the first movements after rest, but gradually diminishes after moderate exercise.

While investigating the various causes of muscular lumbago, it is well to bear in mind that standing still is more fatiguing for the legs and the back than walking, and that leaning forward puts a greater strain on the muscles of the back than standing erect. The backache resulting from fatigue of the dorsal muscles is usually bilateral, and equally severe on the two sides; but not unfrequently it is confined to one side, or is much more severe on one side. This is accounted for by the common practice of throwing the weight of the trunk, while standing, more upon one leg; the spine being more or less laterally curved, and the muscles on one side, therefore, having a greater strain upon them than those of the other.

A common cause of painful overstrain of the dorsal muscles is an excessive weight in the abdomen, such as results from the advanced stage of pregnancy; an accumulation of dropsical liquid, whether ascitic or ovarian; or an excessive development of fat. It is obvious that the continued effort required to maintain the erect posture with an exceedingly ponderous abdomen must often cause a painful strain of the dorsal muscles.

I do not propose now to discuss the treatment of abdominal dropsy. Pregnancy in due course brings its own natural cure; and an excessive weight of fat may be lessened and regulated by a system of diet which reduces to a minimum the amount of oily, saccharine, and farinaceous articles of food. It happens to me very frequently to be consulted by fat men and women suffering from pain in the back, who come to me under the impression that their pain has its seat in the kidneys. For the permanent relief of these cases, I rely mainly upon the following dietary; and I have received many grateful acknowledgments of the relief thereby afforded.

Diet for Excess of Fat.—May eat: Lean mutton and beef; veal; lamb; tongue; sweetbread; soups, not thickened; beef-tea and broths; poultry; game; fish of all kinds; cheese; eggs; bread *in moderation*; greens; spinach; water-cress; mustard and cress; lettuce; asparagus; celery; radishes; French beans; green peas; Brussels sprouts; cabbage; cauliflower; onions; broccoli; seakale; jellies, flavoured, but not sweetened; fresh fruit in moderation, without sugar or cream; pickles. *May not eat:* Fat bacon and

ham; fat of meat; butter; cream; sugar; potatoes; carrots; parsnips; beetroot; rice; arrowroot; sago; tapioca; macaroni; vermicelli; semolina; custard; pastry and puddings of all kinds; sweet cakes. *May drink*: Tea, coffee, cocoa from nibs, with milk; but without cream and sugar; dry wines of any kind in moderation; brandy, whisky, or gin, in moderation, without sugar; light bitter beer; Apollinaris water; soda water; seltzer water; salutaris water.[1] *May not drink*: Milk, except sparingly; cream; porter and stout; sweet ales; sweet wines. As a rule, alcoholic liquors in any form should be taken only very sparingly, and never without food.

Some varieties of backache having their seat in the muscles yet remain for special mention. Amongst the patients who consult me under the erroneous impression that they are suffering from disease of the kidney, are a considerable number who, in addition to pain in the back, are alarmed by the turbidity of the urine. The urine is excessively acid, often of high specific gravity, and, on cooling, deposits an abundant sediment of urates. Neither albumen nor sugar is present; but an excess of nitric acid often causes a copious crystallisation of nitrate of urea. Most of this class of patients are dyspeptics, and not a few eat and drink to excess. Now there is good reason to believe that the backache and the muscular pains in the limbs of which these patients complain are the result of malnutrition and irritation of the muscles, consequent on some defect in the processes of digestion and assimilation. Muscular rheumatism may be a result of *muscular dyspepsia*. It is highly probable that, for instance, a mutton-chop—the muscle of a sheep—which should be so thoroughly digested and assimilated as to nourish and strengthen our human muscles, may, through some defect of the primary digestive or secondary assimilative processes, reach the muscles in a state unfit to nourish them, and rather calculated to fret and annoy them. Thus, in this form of dyspeptic myalgia, while the muscles are starved and tortured, their unassimilated nutriment is ejected by the kidneys in the form of urea and urates.

[1] If any of the above permitted articles of food or drink should be found to disagree they are of course to be avoided.

The successful treatment of this class of cases obviously depends upon our ability first to discover and then to remove the causes of the imperfect digestion, which is the primary source of the muscular pains. With this object in view, each case requires a separate and careful study, in order to discover which, amongst the numerous causes of imperfect digestion, is operative in any given instance. Amongst the most common removable causes of indigestion is imperfect mastication of the food, either from the loss of the molar teeth or the common habit of rapid eating without thorough mastication; the result being, first, that the food is not sufficiently mixed with the saliva which is poured into the mouth during the process of mastication; and, secondly, that the food is swallowed in large masses which are difficult of digestion. In a large proportion of cases, the diet requires to be carefully regulated as regards both quantity and quality, especial care being taken to avoid excess of any kind, whether in food or drink; and most dyspeptics should be warned to avoid soups, sauces, nuts, pickles, spices, salted, smoked, dried, potted, or otherwise preserved meats, on the principle that most *antiseptics* are *antipeptics*, the process of digestion being, in fact, a form of regulated physiological fermentation. The following articles of food and drink are also more or less indigestible, and therefore to be avoided by dyspeptics: veal, pork, dried fish, lobster, salmon, pastry, new bread, cheese, jams, raw vegetables, dried fruits, malt liquors, effervescing wines, liqueurs, cyder, and all stimulants without food.

There is a form of muscular pains from which young people often suffer, and which are commonly called 'growing pains.' These pains are, in fact, the result of over-fatigue of young and growing muscles. I have a vivid recollection of having suffered severely from these pains when I was a rapidly growing youth. The chief remedy or preventive is to be found in rest and the avoidance of over-exertion and fatigue, while the nutrition of the muscles is promoted by fresh air and wholesome food in sufficient quantity.

There is a severe form of lumbago which often comes on suddenly during the act of stooping to pick up something from the floor, or perhaps to pull on a boot. The patient, in the

act of stooping or rising, is suddenly seized with a severe pain in the back, which is aggravated by every attempt to assume the erect posture. This sudden pain is probably caused by cramp or the rupture of some fibres of a muscle during the act of contraction. It is a well-known fact, that a muscle may be torn across by its own active contraction. The late Dr. Sibson, who was a remarkably fine muscular man, and much given to athletic exercises, was one day amusing himself by swinging a heavy wooden club, when, on stepping backwards, he had a sudden sensation of having been sharply struck by a stick on the calf of the leg. The muscles of the calf were found to have been extensively and deeply torn across; and the result was, that for several weeks he had to walk with crutches, while the suicidal muscles were undergoing repair. It is manifest that, short of such extensive injury as this, severe and prolonged myalgia may result from strain and mechanical tearing of over-exercised muscles and tendons. There are few persons who have not had personal experience of the muscular soreness which remains often for days after a severe attack of *cramp*. The common exciting causes of cramp are fatigue and cold and disorders of digestion. An indigestible article of food, which in one individual will cause pain and cramp in the stomach, may in another excite cramp in one or more muscles, thus affording another illustration of 'muscular dyspepsia.'

Dr. Hill Drury[1] has directed attention to the wearing of high-heeled boots as a common cause of backache in women. These absurd boots necessitate the continuous action of the muscles of the lower part of the spine in order to maintain the proper balance and the erect position. Dr. Drury knows of five cases cured by discontinuing the use of high-heeled boots.

When the nutrition of the muscles has been impaired by long inaction, the result of confinement to bed by illness or a mechanical injury, such as a broken leg, pains in the back and limbs often follow the first attempts at exercise during convalescence; and these pains usually continue with more or less severity until, by degrees, the muscles regain their normal state of nutrition and vigour. It is often necessary to warn

[1] *British Medical Journal*, March 26, 1881, p. 467.

those who are attempting to strengthen their muscles by exercise, that over-exertion and fatigue tend rather to weaken than to invigorate the muscles.

Amongst the causes of muscular pains, whether in the back or elsewhere, exposure to cold and damp is, no doubt, common and influential. A so-called stiff neck is often excited by a draught of cold air, and a severe attack of lumbago has frequently been caused by sitting or standing in damp clothes. For the relief of this form of myalgia or muscular rheumatism, the diaphoretic influence of a hot-air or Turkish bath, or of a warm-water bath followed by a vigorous rubbing or shampooing, is often very efficacious. Another useful local remedy is an embrocation composed of equal parts of linimentum belladonnæ and linimentum opii, either rubbed into the skin over the seat of pain or sprinkled on the rough surface of piline, which is then bandaged over the painful part.

Aneurysm of the Aorta.—Amongst the less frequent, but more formidable causes of severe and persistent backache, is pressure on the bodies of the vertebræ and the adjacent nerves by an aneurysm of the abdominal or of the thoracic aorta. In all cases of severe pain in the back, the possibility of aneurysm should be constantly borne in mind, and the signs carefully investigated. In case of suspected abdominal aneurysm, place the patient on his back, with the thighs flexed towards the abdomen, and the abdominal muscles relaxed; then press the ends of the fingers backwards towards the aorta; taking care not to mistake the merely nervous pulsation which occurs in excited states of the circulation, especially in anæmic women, for the expansive pulsation of an aneurysm. A soft blowing sound may always be excited by the pressure of the stethoscope on any large artery, but in most cases of abdominal aneurysm a more or less rough murmur is heard on applying the stethoscope even lightly over the pulsating tumour; and this sound, being conducted through the bones, is often distinctly audible at the back over the spinous processes of the corresponding vertebræ.

It not unfrequently happens that an abdominal aneurysm bursts, and forms a false aneurysm behind the peritoneum; and such a tumour, taking a lateral course towards the loin,

may seem to be so remote from the aorta as to be mistaken for a tumour of a different kind. One such case, that of George V——, will be found recorded at p. 581.[1]

Aneurysm of the *thoracic aorta below the arch* is, according to my experience, of less frequent occurrence than aneurysm of the abdominal aorta. It is usually associated with severe pain in the back, resulting from the pressure of the aneurysm on the vertebræ and the ribs. In one case which I saw, with the late Mr. Lavies, at the Westminster Prison, the physical signs were dulness on percussion in the left interscapular region, with an audible impulse and bellows sound. In another case, seen many years ago, with a since deceased relative, in the Cranbrook Union House, the aneurysm had caused absorption of the ribs, and formed a prominent pulsating tumour below the angle of the left scapula.

Cancerous glands in the abdomen are amongst the comparatively rare causes of pain in the back. Within a period of three years I have seen, in consultation, four cases of malignant disease, in which, there having been a pulsating tumour in the abdomen, the diagnosis between cancerous glands and abdominal aneurysm was, in the earlier stages, by no means easy.

Case I.—Mrs. H——, aged 65, was seen with the late Dr. Butler, of Winchester, in July 1878. There had been constant pain in the back and in the left loin and thigh since April. The pain *was worse after exercise.* A pulsating tumour was felt at the epigastrium, without audible murmur, but the pulsation was heard over the lumbar spinous processes. There was loss of flesh, which went on increasing. In September, a hard swelling was felt at the umbilicus, and another in the right groin, which, as Dr. Butler in writing to me said, confirmed the diagnosis of malignant disease. She died at the end of December. In the meantime, the liver had become much enlarged; there was some jaundice, ascites, and swelling of the legs. There was no *post-mortem* examination, but the disease was evidently malignant.

Case II.—Mr. G. B——, aged 38, seen, with Dr. Hearnden, of Sutton, and Mr. Drake, of Brixton, first in September 1878.

[1] For the history of other cases of abdominal aneurysm attended with more or less severe pain in the back, see Chapter XXXVIII., section iv.

In this case there was constant pain in the back, *worse at night*. A pulsation was felt, and a bellows sound heard at the epigastrium, and my first diagnosis was aneurysm; but in the course of a few weeks he rapidly lost flesh; and when I last saw him, in March 1879, he was much emaciated. He had been jaundiced for some weeks; there was ascites and some swelling of the legs. He died in April. There was no inspection, but it was undoubtedly a case of malignant disease.

Case III.—Mrs. H——, aged 67, seen with Mr. Holberton, of Hampton, in September 1878. There had been pain in the epigastrium and back for three months, uninfluenced by food; also loss of flesh and strength. There was a diffuse pulsation in the epigastrium without audible murmur. She continued to lose flesh and strength, the pain in the back continued, and towards the end, there was vomiting. There was no inspection, but Mr. Holberton wrote to me that latterly he believed that both the stomach and the liver became involved in the malignant disease. She died in March 1879.

Case IV.—Mr. J. T. A——, aged 31, seen with Mr. James Connor, of Battersea, on April 4 and 30, 1879. Had pain in the back—*worse at night*—since January. The pain was not made worse by food, or by exercise. He was pale, and had lost flesh considerably. A pulsating tumour was felt in the epigastrium, and a blowing murmur was heard. He continued to lose flesh and strength, and died on October 5. A large mass of cancerous glands was found 'behind and below the stomach, and in front of the aorta,' as I learn from my friend Mr. Connor, who made the *post-mortem* examination.

In all these cases, it will be seen that pain in the back was a constant symptom. In all there was a pulsating tumour at the epigastrium; in two there was an audible murmur, while in the others no murmur was heard, though in one of these the sound of pulsation was conducted through the vertebræ to the back. It is remarkable that in two cases the pain was more severe at night, and in only one was it spoken of as made worse by exercise. The pain of abdominal aneurysm is usually relieved by rest in the recumbent posture, and aggravated by exercise. In all the cases there was a more or less

rapid loss of flesh and strength, and death within a period of nine months from the commencement of the symptoms.

The persistent backache and epigastric pulsation are common to cases of abdominal cancer and aneurysm. In the former a blowing murmur is less common than in the latter. One main difference between the two diseases consists in the evidence of serious constitutional disorder, with progressive emaciation, and death within a year from the onset of malignant disease.

Disease of the kidneys, although a less frequent cause of pain in the back than is generally supposed, is a not uncommon cause of more or less severe pain in one or both loins. The forms of kidney disease which are the most frequent causes of lumbar pain are calculus in the kidney, but especially in the ureter; malignant or scrofulous disease with enlargement of the gland; acute congestive forms of Bright's disease; temporary blocking of the ureter by a blood-clot in cases of renal hæmorrhage; distension and dilatation of the pelvis of the kidney resulting from retention of urine, whether caused by stricture of the urethra, enlargement of the prostate, or paralysis or other morbid condition of the bladder. In some gouty and dyspeptic subjects, nephralgia appears to result from the highly acid and irritating quality of the urine. Lastly, I have seen a considerable number of cases in which the dragging of a misplaced, movable kidney upon its attachments has caused severe pain in the back. I do not propose now to discuss the diagnosis of the various forms of kidney disease to which I have here referred. (See Chapters XLV., XLVII., and XLVIII.)

Gastric Ulcer.—It may be well to mention in passing that the pain of simple ulcer of the stomach is usually felt not only at the seat of the ulcer, which is generally at the lesser curvature or in the posterior wall of the stomach, but it is also referred to the corresponding spot at the back. This is an example of a referred or reflected pain.

Uterine Disease and Displacement.—Amongst the common causes of backache in females are certain morbid conditions and displacements of the uterus, the diagnosis of which is important, inasmuch as many of them admit of much relief by suitable treatment. As this is a special subject of which

I have little practical knowledge, I dismiss it with this brief reference.

Disease of the Bones of the Spine, and of the Spinal Cord.—Amongst the most serious forms of pain in the back are those which result from disease of the bony column, or of the spinal cord, or its membranes. The most common form of disease of the vertebræ is the strumous, with angular curvature, and often with lumbar or psoas abscess. The bones of the spine, too, are sometimes the seat of malignant disease. The spinal cord and its membranes may become secondarily implicated in cases of primary disease or injury of their bony sheath, but more frequently disease of the cord and its coverings is unconnected with disease of the vertebræ. In cases of *spinal meningitis*, there is, as a rule, more pain in the back than when the substance of the cord is alone affected. The pain is usually increased by movement, by percussion on the spine over the seat of disease, and by hot applications to the surface. The pain is often referred to the extremities of the nerves which pass out from the part affected, and is commonly associated with numbness, a sense of tingling, and other perverted sensations. A sense of constriction across the chest is a frequent symptom. There is often spasmodic twitching of the muscles; and in proportion to the affection of the substance of the cord is the frequency of paralysis (paraplegia) affecting the parts below the seat of disease, and implicating not only the voluntary muscles, but also those of the bladder and rectum, with resulting retention or incontinence of urine and fæces.

Disease of the spinal cord itself is usually attended with less pain than when the membranes are the seat of disease. Inflammation of the substance of the cord (myelitis) may quickly result in paraplegia, with little or no local pain in the back. In some cases of spinal *hæmorrhage*, too, paralysis below the seat of pressure has been associated with little or no pain; while, in others, more or less severe dorsal pain has marked the onset of the hæmorrhage.

Pressure on the spinal cord by a *tumour*, fibrous, strumous, or cancerous, has often been associated with severe pain in the back. In a case recorded by Mr. Shaw,[1] paraplegia was

[1] *Pathological Transactions*, vol. ii. p. 24.

caused by two scrofulous tumours occupying the interior and lower part of the spinal cord, and invested on all sides by a thin layer of medullary matter. In that case, the pain in the lumbar region was so severe that the symptoms were supposed to result from caries of the vertebræ; and the more so, as there was a slight projection of one of the lumbar spinous processes.

The pain resulting from a tumour pressing on the spinal cord is very variable. In one case quoted by Dr. Abercrombie, the first symptom was neuralgic pain in the arm, which diminished as paralysis came on; in another case the patient had sciatic pain extending to the toes. Mostly the pain is referred to the back, and indicates the seat of the disease; thence it radiates in the direction of the nerves whose roots are invaded. When there is no actual pain, there may be modifications of sensation, such as a sense of coldness or heat, or sudden alternations of these; numbness and pricking, or formication. Next in frequency to pain are muscular contractions in the affected limbs, followed in some cases by rigid flexion of a limb, and attended by a great susceptibility to the excito-motor stimulus, and, in a yet further stage, by complete paralysis. In short, the effect of moderate pressure on the cord is to cause spasm and neuralgia, passing on, with increase of the lesion, to paralysis and complete anæsthesia.

I scarcely need warn you to be vigilant and careful not to mistake any of these formidable diseases of the spinal cord and its membranes for the muscular and rheumatic affections to which I referred in the earlier part of this lecture.

There is an acute and transient form of backache which probably has its seat in the spinal cord. I allude to the pain in the back which occurs at the commencement of many acute diseases, especially the febrile exanthemata, and which is usually much complained of during the initiatory fever of small-pox. In some cases which have proved to be nothing more serious than febrile catarrh, the initiatory backache has been so severe as to excite a suspicion of incipient small-pox.[1]

[1] See Chapter X. for the suggested cause of this pain.

CHAPTER XL.

CLINICAL LECTURE ON HÆMATEMESIS AND PERFORATING ULCER OF THE STOMACH.[1]

A Case of Hæmatemesis from Gastric Ulcer—Treatment of Hæmorrhage—Treatment of Perforation—One Case of Perforation successfully treated—Question of Surgical Treatment in such Cases—Chief Diagnostic Signs of Ulcer and of Perforation—The Perforating Ulcers are generally in the Anterior Wall or on the Lesser Curvature—The Symptoms of Duodenal Ulcer—Case of Perforating Duodenal Ulcer.

I WISH to direct your attention to a case of hæmatemesis recently admitted into the hospital, and I will give you a history of the case, condensed from the more detailed report which has been drawn up by my clinical clerk, Mr. Mayo.

Sarah D——, aged 28, a maid-servant, was admitted into Twining ward on February 3, 1870. She said that, for three months before her admission, she had suffered from pain in the right side, loss of appetite, and lowness of spirits. The catamenia had been quite regular. On January 30 she twice fainted, without apparent cause; and on each occasion, just as she was recovering consciousness, she vomited a large quantity of black blood—she says, as much as a pint each time. She went to bed for the remainder of the day. The next day she got up; and, having taken some gruel and beef-tea, in the course of the morning she again fainted, and a third time she vomited a large quantity of blood—'about half a basinful.' On February 1 she again fainted twice; and each time the faintness was succeeded by vomiting of blood.

On admission she had a very anæmic appearance, the lips and the tongue being quite pallid. She complained of a constant thumping in her head; a feeling of sickness; a bitter

[1] *British Medical Journal*, March 26, 1870.

taste in the mouth; pain and tenderness in the right hypochondriac, but not in the epigastric region. The tongue was covered with a yellowish fur. Pulse 100, very small and feeble. A soft systolic blowing was heard over the base of the heart. The urine was normal.

She was placed upon milk diet. On the 4th—the day after her admission—she vomited once, but brought up no blood. On the 5th she twice vomited about a table-spoonful of blood. She was then ordered to take ten grains of tannic acid in water every four hours; to have no food by the mouth, but, in place of it, an injection of beef-tea, egg, and brandy, every two hours. On the 7th there had been no return of sickness. A stool was passed in the morning containing no blood. After six doses of the medicine had been taken it was discontinued, on account of its exciting nausea. She had no food by the mouth. The nutritive enemata were continued. On the 9th there had been no return of the sickness, but she was thirsty, and wanted food. She was ordered to take some iced water and milk, and cold beef-tea; three grains of sulphate of iron in two pills, with extract of gentian and ginger, three times a day; the enemata to be discontinued. On the 10th she vomited after eating an orange which had been surreptitiously obtained, but no blood appeared. On the 15th she had fish and potatoes for dinner; on the 16th, a chop. Since then she has gradually returned to the ordinary meat diet. There has been no return of the bleeding. She continues the iron pills, and she is rapidly recovering her lost strength and colour.

Now, in this case there can be no doubt that the stomach was the source of the bleeding. In cases of hæmorrhage from the stomach it not unfrequently happens, as in this case, that, while a copious bleeding is going on internally, the patient faints and loses consciousness, and may actually appear to be dying before any blood is expelled by vomiting. In some cases no vomiting occurs; the blood passes into the bowels; and a patient who has become rapidly exhausted and anæmic is found to be passing blood by stool. If, in the history of an attack of hæmorrhage, you learn that the patient fainted before any blood appeared externally, and then that black and

perhaps clotted blood was vomited, you may conclude that the blood came from the stomach. In this respect there is a marked contrast between gastric and pulmonary hæmorrhage. Bleeding into the air-passages immediately excites coughing. The blood begins to be expectorated as soon as it escapes from the vessels. It is generally florid and frothy; and it sometimes happens that the first appearance of the blood so alarms a nervous patient as to cause faintness, when the mere loss of blood has been quite inconsiderable.

That the cause of the hæmorrhage in our patient is a simple gastric ulcer is highly probable, although some of the symptoms of that disease were wanting. In particular, it is to be observed that the pain, which was referred to the right side, had not been increased by taking food. She had not been troubled with water-brash, or with vomiting, before the occurrence of the hæmatemesis. There is no evidence of impeded circulation through the liver or the heart; the soft systolic blowing over the base is doubtless anæmic; the catamenia have appeared regularly; and there can be little doubt that a gastric ulcer is the source of the bleeding.

Now I wish to direct your attention particularly to the treatment of the two most serious and alarming accidents of gastric ulcer—I mean hæmorrhage and perforation.

In the treatment of hæmorrhage absolute rest in bed is essential. Then, while the tendency to bleeding continues, no food should be introduced into the stomach; but the patient should be sustained by nutritive enemata. The introduction of food into the stomach excites an increased afflux of blood to the organ. This alone may cause a return of the bleeding; and with the increased supply of blood there is a copious secretion of gastric juice, which, coming in contact with the ulcer, may dissolve the recently formed coagula that had plugged the bleeding vessels. The bleeding patient, therefore, should lie still, sip iced water, and be fed by the rectum. The most useful styptics in these cases are—tannic acid in ten-grain doses, tincture of perchloride of iron in twenty-minim doses, or oil of turpentine in twenty-minim doses. Each of these medicines I have seen speedily arrest the bleeding in different cases. In the present case the tannic acid excited nausea, and

it was discontinued. In any case, I believe that the exclusion of food from the bleeding stomach is of more importance than the administration of medicine; yet I should always give one or other of the styptics that I have mentioned, if the bleeding continue in spite of abstinence and iced water. When the bleeding has ceased, liquid food may gradually and cautiously be given by the stomach; then solids; and, lastly, iron is a most valuable restorative tonic.

In the treatment of *perforation* of the stomach, the necessity for keeping the stomach free, not only from food, but from medicine, is absolute. This accident is generally fatal, and more rapidly so when, as very frequently happens, the perforation occurs soon after a full meal; but there are on record a few cases in which a patient has recovered after symptoms of perforation had occurred. One such case has been published by the late Dr. Hughes in the *Guy's Hospital Reports* (1846), and one case occurred in my own practice.

On Christmas Eve, 1863, I saw, in consultation with Mr. Henry Lee, Mrs. T——, aged 40. About two hours before she had been seized suddenly in the street with severe pain, rapidly extending over the whole abdomen. She felt faint and vomited, and had to be taken home in a cab. When I saw her she had excessive pain and tenderness over the whole abdomen, the pain being much increased by pressure, and by any movement of the body. The pulse was rapid and feeble and the skin cold. I learnt that for several months past she had suffered from pain in the region of the stomach, increased by taking food. She had frequent acid eructations, for which she was in the habit of taking calcined magnesia; and, about three months before, she had on one occasion vomited a large quantity of blood. Here there was a distinct history of chronic gastric ulcer; and now the symptoms pointed clearly to perforation and consequent peritonitis. Acting upon that view of the case, we prescribed every two hours an enema consisting of half a pint of beef-tea, half an ounce of brandy, and fifteen minims of laudanum; by the mouth, only an occasional dessert-spoonful of water or barley water to allay thirst; perfect quietude in bed; hot fomentations to the abdomen. Under this treatment the symptoms gradually subsided. As the

pain abated, the laudanum was discontinued; but she was fed entirely by the rectum for nearly three weeks. She then began to take by the stomach small quantities of bread and milk, beef-tea, and arrowroot. For the first two or three days of feeding by the stomach, the food was rejected by vomiting; but this soon subsided, and she steadily regained strength. She called at my house on February 19, 1864. She was then quite convalescent; but she had an anæmic appearance, and still complained of her old dyspeptic symptoms. I have not since seen or heard of her.

In this case, recovery having taken place, it is, of course, not certain that there had been perforation of the stomach The history, however, leaves very little room for doubt. This, at any rate, is certain: that the only chance of recovery from perforation of the stomach is afforded by giving both food and medicine by the rectum, and excluding both from the stomach until the aperture has been firmly closed.

Many years ago, on *post-mortem* examination of a case of perforating ulcer of the stomach, I found that a large pill had passed through the perforation into the cavity of the peritoneum. There can be little doubt that much food and irritating physic has often taken the same course in similar cases. Let us be careful to avoid a practice that of necessity must aggravate the patient's pain and peril.

Postscript.

Since the preceding lecture was published abdominal surgery has made great progress, and there is now reason to hope that, in some cases of perforation of the stomach, life may be saved by prompt surgical interference.[1] Two years ago, at a meeting of the Royal Medical and Chirurgical Society, over which I had the honour to preside, papers were read by Mr. Treves and Mr. Howard Marsh[2] on the successful treatment of acute peritonitis by abdominal section. In the course of the discussion I asked if surgeons could not come

[1] See *ante*, p. 139.

[2] *Proceedings of the Royal Medical and Chirurgical Society*, March 10, 1885 and *Med.-Chir. Transactions*, vol. lxviii.

to the rescue, in cases of perforation of the stomach. After the publication of the discussion, I received from Mr. Nelson C. Dobson, of Clifton, a paper entitled 'Abdominal Section in Perforating Ulcer of the Stomach.' This paper had been published the previous year in the *Bristol Med.-Chir. Journal*, and Mr. Dobson therein gives good reasons for his belief that surgical interference may, sometimes at least, be successful in cases of perforating ulcer of the stomach. Now it is obvious that since, in cases of this kind, death usually occurs in from eighteen to thirty hours from the onset of the symptoms of perforation, surgical treatment, to be successful, must be resorted to with the least possible delay. The question of diagnosis, then, is one of primary importance. The previous history is an important aid in the formation of a diagnosis. The symptoms which are commonly present in cases of simple ulcer of the stomach are pain and soreness of the stomach, almost always brought on or increased by food, continuing for many weeks or months, with occasional sour eructations and occasional vomiting, but without much constitutional disturbance. If to these symptoms be added profuse vomiting of blood, the existence of gastric ulcer may be inferred with almost absolute certainty. Some of these symptoms, however, the hæmatemesis especially, may not have occurred before the ulcer has perforated the coats of the stomach.

The symptoms which directly result from perforation are sudden agonising pain at the epigastrium, coming on usually soon after a meal, and rapidly spreading over the whole abdomen, a quick and feeble pulse, and a cold and clammy skin. The state of collapse hourly increases, and speedily ends in death.

Now, there is one anatomical fact in the history of perforating ulcer, mentioned by Dr. George Budd,[1] which is favourable for surgical treatment—namely, that perforation is most common when the ulcer is situated either on the anterior wall of the stomach or along its lesser curvature, where, of course, the aperture is more accessible through the abdominal wall. It fortunately happens that an ulcer on the posterior wall very commonly leads to the formation of adhesions between the

[1] *Diseases of the Stomach*, p. 127.

stomach and the neighbouring viscera, by which means perforation is prevented.

When abdominal section is resorted to in these cases, it should be borne in mind that the perforation may have occurred, not in the stomach, but in the upper part of the duodenum.

Dr. Budd refers to three cases of perforating duodenal ulcer, one of which occurred in my own practice.

The symptoms of duodenal ulcer are usually much less severe than those of gastric ulcer, and perforation may occur with little or no previous warning. The urgency of the symptoms after the occurrence of duodenal perforation is probably much influenced, as in the gastric cases, by the amount of food in the stomach, which, passing on into the bowel, would escape into the cavity of the abdomen. In one of the three cases recorded by Dr. Budd, death occurred eighteen hours, and in another twenty-four hours, after the occurrence of perforation. In the third case, the particulars of which I communicated to Dr. Budd, a healthy carman, 35 years of age, was suddenly seized with the pain which indicated the occurrence of perforation about noon, when the stomach was probably empty, and he survived the attack four days. I found a single perforating ulcer on the anterior surface of the first part of the duodenum. An ulcer in that position might readily be discovered and treated by abdominal section.

CHAPTER XLI.

ON CASES OF EFFUSION INTO THE PERITONEUM, ANALOGOUS TO CASES OF LATENT PLEURISY.[1]

Effusion into the Peritoneum cured by a Single Tapping—Ascites cured by Diuretics—Effusion into the Peritoneum and both Pleuræ with Albuminuria—These Cases analogous to Latent Pleurisy—Two Cases recorded by Mr. Meade—Sir Thomas Watson's Description.

THERE is a class of cases of serous effusion into the cavity of the peritoneum which are but little noticed by writers on the practice of medicine. During the last nine months the following three cases have come under my observation.[2]

CASE I.—Mary D——, aged 51, was admitted on October 28, 1875. She was married, had had three children and five miscarriages. She menstruated regularly up to last July; she then caught cold whilst menstruating. This was followed by pain in the lower part of the abdomen, and soon by swelling, which rather rapidly increased. Her habits had been active and strictly temperate. She was in good health before the commencement of the present illness.

On admission the abdomen was much distended, resonant on percussion at the upper and anterior part, dull in the flanks; fluctuation was very distinct. The breathing was impeded and difficult, especially in the recumbent posture. The cardiac and pulmonary sounds were normal; the liver, so far as could be ascertained, of normal size. She had lost flesh since her illness, but she had not a jaundiced or a cachectic appearance. The urine was normal; pulse, 104; tempera-

[1] *British Medical Journal*, September 16, 1876.

[2] A fourth case, of which I gave some particulars in my original paper, proved to be one of tubercular peritonitis, and is therefore not included in the present series.

ture, 98°. She was ordered, as a diuretic, half a drachm of potassium acetate and ten minims of tincture of digitalis three times a day. A fortnight after her admission her condition remained unchanged; and, at my request, my colleague the late Mr. Royes Bell tapped her, and drew off 204 ounces of dark straw-coloured serum. She expressed herself much relieved. There was no pain or tenderness after the operation. There was no return of the swelling, and she was discharged cured on November 30.

Case II.—Mr. G——, aged about 50, a verger of St. Paul's Cathedral, was seen by me twice, on November 11 and 19, 1875, in consultation with my friend Mr. Holding. The patient's habits had always been regular and strictly temperate. About six weeks before I saw him he began to have some tenderness of the abdomen; and this was soon followed by an enlargement which had steadily increased. At the time of my first visit the abdomen was much distended and fluctuating, but not painful or tender. There was no evidence of cardiac, pulmonary, or hepatic disease. The urine was normal. We agreed to give the following diuretic:—℞ Potassii iodidi, ʒj.; potassii acetatis, ℥ss.; tinct. scillæ, ʒij.; syrupi zingiberis, ℥j.; aquæ ad ℥vj. M. A tablespoonful in water three times a day.

I afterwards learnt from Mr. Holding that, under the influence of the diuretic, which the patient continued to take until January 6, the urine had increased in quantity, while the abdominal swelling had steadily decreased; and the recovery had been complete. I have since seen him in the active discharge of his duties in the cathedral, and apparently in good health, as, indeed, he assured me that he was.

Case III.—Edmund F——, aged 20, was admitted into Craven ward on March 25. He is a wood-turner, with a good family history. About March 1 he began to feel weak, so that he could scarcely walk home after his day's work. Soon he had a sensation as of something rolling about in his belly, which was beginning to enlarge, but was not painful. He was not aware of any exposure to cold.

On his admission he had an anæmic appearance. The abdomen was enlarged, the measurement round the umbilicus

being thirty inches. The anterior part of the abdomen was resonant; the flanks were dull, with distinct fluctuation. There was the normal extent of percussion dulness over the liver; no tenderness on pressure over the abdomen. There was dulness on percussion, with feeble respiratory sound, as high as the angle of the left scapula. The cardiac sounds were normal. The urine was acid, specific gravity 1020, of normal colour, and free from albumen. Temperature, 101°. He was ordered: Potassii iodidi, gr. iij.; infusi quassiæ, ℥j.; three times a day.

March 30 —There were dulness on percussion and feeble respiratory sounds over the right base, but not to the same extent as on the left side.

April 3.—The measurement of the abdomen had increased to thirty-one inches, and there was slight tenderness on pressure. He was ordered a wet pack daily, and to add to each dose of the mixture half a drachm of potassium acetate.

April 6.—He sweated profusely at night. The temperature had ranged from 98°·2 in the morning to 104 ·2 in the evening. He was ordered to discontinue the packing.

April 10.—The signs of effusion into the chest and abdomen remained the same. He was ordered two drachms of cod-liver oil three times a day, and the following draught twice daily:—℞ Quininæ sulphatis, gr. ij.; acidi sulphurici diluti, ♏v.; aquæ ℥j.

April 27.—The urine now contained a small amount of albumen (one in twenty).

May 1.—The albumen had increased to one in four, and contained small hyaline casts, some of which contained leucocytes. He was ordered to have a diet of milk exclusively. The fluid gradually disappeared from the pluræ and peritoneum. There was for some days a friction sound near the angle of the left scapula, but ultimately the chest sounds became quite normal. His general health improved. The albumen gradually diminished, but a slight trace remained when he left the hospital on July 14. For about a month before he left the hospital he was allowed fish and mutton in addition to the milk diet.

The cases of serous effusion into the peritoneum which I

have here briefly recorded appear to me to be analogous to cases of so-called 'latent pleurisy,' in which a copious pleuritic effusion is often found unassociated with pain or febrile disturbance. Case III. is the only one in which there was elevation of temperature, and that case was complicated, not only with a simultaneous effusion into the peritoneum and into both pleuræ, but also with the subsequent occurrence of albuminuria. It is probable that the serous effusions and the albuminuria were all results of some temporary blood-contamination, consequent, perhaps, on a partial suppression of the cutaneous secretion.

Obviously it is of importance, with reference both to prognosis and treatment, to distinguish these cases of sub-inflammatory effusion into the peritoneum, the result, apparently, of a chill, from the much more intractable cases of ascites consequent on cirrhosis of the liver. It is also important to distinguish them from cases of tubercular peritonitis.

The *British Medical Journal* of September 23—the week after the publication of the preceding communication—contains a paper which had been read before the annual meeting of the Yorkshire branch of the Association by Mr. R. H. Meade, of Bradford. Mr. Meade's communication is entitled *On some Forms of Idiopathic Peritonitis*; and that he is referring to the same class of cases as those which I have recorded is evident from the following opening paragraph, in which the resemblance to cases of latent pleurisy is mentioned in terms almost identical with those which I had employed. Mr. Meade says:—

'Peritonitis is so frequently traumatic in its origin, or comes on during the course of other diseases, that its existence as a primary affection, like pleurisy, is often ignored, or very briefly alluded to by most authors. The peculiar form of peritonitis to which I wish to call attention mostly arises from cold; it is generally subacute, and sometimes latent in its character, and speedily causes effusion of serum into the abdominal cavity. This complaint often bears a great resemblance to the latent forms of pleurisy not uncommon in children and young persons; in which, after exposure to cold or damp, the patient complains of slight pain in one side,

followed by shortness of breathing; and, upon examination, one pleural cavity is found full of fluid.'

Two cases are briefly mentioned by him—one subacute, with a high temperature, the other latent. Both patients quickly recovered.

Sir Thomas Watson refers to such cases in the following terms:[1]—'There is another species of ascites, not very common, which approaches in its character to inflammation, and which is, therefore, called *active* ascites. I mean that we sometimes see persons, who were previously in good health, become rapidly ascitic after exposure to cold and wet, and rapidly recover again under the remedies which are used to subdue inflammation.' And further on he says: 'The balance of the circulation between the skin and the internal surfaces appears to be destroyed, on these occasions, by the operation of external cold upon the tegumentary membranes.'

[1] *Lectures on the Principles and Practice of Physic*, 5th ed. vol. ii. p. 438.

CHAPTER XLII.

ON A CASE OF SIMPLE STRICTURE OF THE COMMON BILE-DUCT, CAUSING JAUNDICE AND ASCITES.[1]

Jaundice and Ascites removed by Tapping the Abdomen—Return of the Symptoms—Temporary Relief by Tapping, then a Rigor, Vomiting, and Death—Stricture of the Common Bile-duct causing Dilatation of the Ducts in the Portal Canals, Pressure on the Branches of the Vena Portæ, and Ascites.

The following case, which came under my care in the hospital, presents some points of unusual clinical interest.

Mary Ann N——, aged 38, was first admitted on July 9, 1879. She is married; has had seven children, the youngest a year old. Habits temperate. In December 1878, four months after her last confinement, she noticed that her skin was becoming yellow, and her urine high-coloured. These symptoms had continued until the time of her admission. She was then jaundiced, and the urine was deeply bile-tinged. The liver-dulness extended from the fifth rib to two inches below the costal margin. The lower edge of the liver was thin and firm. There was some evidence of liquid in the abdomen.

After remaining three weeks in the hospital, she was sent to the convalescent home on August 2, and readmitted on September 25. The jaundice had continued since her discharge, and the abdomen had greatly increased in size, the measurement at the level of the umbilicus being forty-three inches. Fluctuation was distinct, and there was dulness on percussion everywhere except at the epigastrium. The feet were œdematous.

On September 27, 344 ounces of bile-tinged liquid were

[1] *British Medical Journal*, August 7, 1880.

removed by tapping, with great relief from pain and distension. The margin of the liver could be felt as before described, and below the margin a distended gall-bladder was sometimes distinctly felt. The fluid reaccumulated, and again caused great pain and distress. On October 28, a second operation removed 284 ounces of fluid, and afforded much relief. After the second tapping, the urine gradually became lighter coloured, and the skin was less deeply jaundiced; but the abdomen again enlarged; and on November 26, 271 ounces of liquid were removed by a third tapping. Again she was much relieved by the operation, and the fluid did not reaccumulate. The abdomen now measured only thirty-five inches. The urine gradually lost its bile-tinge, and the skin and conjunctivæ nearly regained their normal colour. She recovered her appetite and strength, and was discharged, apparently convalescent, on January 10, 1880.

She was readmitted on June 21, 1880. After leaving the hospital, she had remained quite well until six weeks ago, when she noticed that her skin was again becoming deep yellow; and a month since her abdomen again began to enlarge. On admission, the skin and eyes were deeply jaundiced; the urine contained an abundance of bile; the abdomen was much distended, measuring forty-four inches and a half at the navel. She had pain and dyspnœa from abdominal distension. On the 24th, 303 ounces of liquid were withdrawn by paracentesis, after which the liver was felt with its thin edge two inches and a half below the ribs, as before noticed. Great relief was afforded by the tapping; but four days after the operation she had a rigor. Temperature 102°·3; abdomen tender, and again becoming distended. There was occasional vomiting; the pulse became rapid and feeble, the tongue dry; and she died on July 4, ten days after the last tapping.

Inspection by Mr. Barrow Twenty-four Hours after Death—The abdomen contained a large amount of dark turbid liquid. The peritoneum was intensely congested; the intestines covered by recent yellow lymph. The liver was stained of an olive-green colour, somewhat enlarged; the lower margin was thin, moderately firm. The gall-bladder, distended by dark bile to the size of a turkey's egg, extended some distance

below the margin of the liver. The cystic and hepatic ducts were much dilated; the dilatation of the hepatic ducts extending into the interior of the liver; sections of which showed the ducts in the portal canals large enough to admit a middle finger. The common duct, just below the junction of the cystic and hepatic ducts, was obstructed by a fibrous thickening of its coats. Very firm pressure on the distended gall-bladder caused only a slight oozing of bile through the common duct into the duodenum. The omentum was thickened, folded upwards, and adherent to the under surface of the diaphragm. The capsule of the spleen was thickened. The kidneys were soft and stained yellow. A cyst, of the size of a small cocoa-nut, was connected with the left ovary.

Remarks.—Until the inspection of the body revealed the true nature of the pathological changes in this case, it was impossible to refer the symptoms to their true cause. The main phenomena were—deep jaundice, followed by great ascites; the disappearance of both the jaundice and the ascites after the third tapping; the reappearance of jaundice and ascites after an interval of about five months.

The first link in the chain of morbid processes resulting in jaundice and ascites was evidently the constriction, amounting to almost complete obliteration, of the common bile-duct. The exciting cause of the inflammatory process which resulted in this stricture of the gall-duct is not apparent. There was no indication of syphilis. There was no history of gall-stones, the passage of which might have caused ulceration and subsequent stricture of the duct. But, starting from the obstruction of the duct, the resulting phenomena are sufficiently intelligible. The impeded escape of bile caused the jaundice and the gradual dilatation of the hepatic ducts. The dilated ducts compressed the portal veins within the canals, thus obstructing the whole portal circulation, and causing the ascites. The jaundice was a direct mechanical result of the constricted gall-duct; the ascites an indirect result. The force which dilated the gall-duct, and, by this means, so impeded the flow of blood through the portal vein and its tributaries as to cause ascites, was evidently derived from the active secretory function of the liver. The temporary passing away of the jaundice

and ascites after the third tapping is explained by supposing that, in addition to a permanent constriction of the duct by inflammatory exudation, there was a congested and swollen condition of the lining membrane, which subsided after removal of the dropsical pressure by tapping; thus allowing the accumulated bile to escape, and removing the pressure from the portal veins. In like manner, we have seen that, in cases of simple stricture of the sigmoid flexure of the colon, temporary obstruction of the bowel may result from congestion and swelling of the mucous membrane at the constricted part. The swelling of the mucous membrane subsiding under the sedative influence of opium, the fæces again pass, and the bowel unloads itself. Then, after an interval of weeks or months, from the irritation of indigestible food or a drastic purgative, congestion and swelling of the mucous membrane, with complete obstruction of the bowel again occur, and perhaps a fatal result.

The liver, unfortunately, was not weighed; but there was evidently a combination of enlargement, resulting from dilatation of the hepatic ducts, with some degree of atrophy of the glandular tissue; and some sections, which my friend and colleague Mr. Barrow was good enough to make for me, showed atrophy of the lobules, with an increase of connective tissue in the interlobular spaces. These atrophic and hyperplasic changes are exactly analogous to those which occur in the kidney in consequence of an impeded escape of urine from the gland, whether resulting from obstruction in the urethra, in the neck of the bladder, or in the ureter.

It is interesting to note that, although the last tapping, when her strength had been much impaired, was followed by fatal peritonitis, the previous three tappings not only afforded great immediate relief, but unquestionably prolonged her life in comfort for several months. The satisfactory result is an encouragement to repeat the operation of paracentesis in cases of recurring ascites, when other means have failed to remove the dropsy.

CHAPTER XLIII.

CLINICAL LECTURE ON A FATAL CASE OF PERITONITIS EXCITED BY THE ESCAPE OF HYDATIDS FROM THE LIVER.[1]

Pain in the Abdomen, Vomiting, and Jaundice—Relief from Opium—Return of Symptoms in an Aggravated Degree—Fatal Peritonitis the Result of Hydatids from the Liver having escaped into the Cavity of the Peritoneum.

GENTLEMEN,—A case of unusual interest ended fatally the day before yesterday. We are presently about to make a *post-mortem* examination of the body, but before doing so I propose to give you a brief history of the disease, condensed from the report of my clinical clerk, Mr. (now Dr.) Tirard, and to state to you my opinion as to its probable nature and cause.

The case is that of Kate L——, aged 27, married, but without children, a book-folder, who was admitted into Twining ward on October 19. She stated that she had been in good health until five weeks before her admission, when she was seized with pain in the pit of the stomach; she had frequent retching, the urine became high-coloured like coffee, and her eyes and skin were yellow. The very severe pain and vomiting had ceased some time before her admission, but she remained very weak and ill, with great tenderness of the belly. When admitted there was pain and extreme tenderness over the whole front of the abdomen, the abdominal walls were tense, and palpation gave the impression of the intestines and omentum being matted together. The eyes and skin were still yellow, but the urine was free from bile and of normal character. The temperature was 102°·4 Fahr., the pulse rapid and feeble, but its exact frequency was not noted. The symptoms clearly indicated peritonitis, and for this I pre-

[1] *Medical Times and Gazette*, Jan. 1, 1876.

scribed fomentations, and a grain of opium in a pill three times a day.

Under this treatment the pain and tenderness gradually subsided, and the improvement was so great that at the beginning of November, it seemed probable that she might recover. Meanwhile, the jaundiced state of the skin and eyes had quite passed away.

On November 5 she complained of a 'bearing-down' pain in the abdomen. On the 8th the pains had recurred. On the 10th the urine was tinged with bile, and the skin and eyes were again becoming yellow. On the 11th the pain was more severe, and attended with vomiting; urine dark-coloured; jaundice increased; great tenderness over the epigastrium. Pulse 170, temperature 105°. The opiate treatment was continued. On the 12th the symptoms continued unabated; delirium had come on. Temperature 101°·2, pulse 160. On the 13th the vomiting had been frequent during the night; temperature 103°·2, pulse 148; countenance anxious. She died at 2 P.M.

Now, what is the nature of this disease? We shall presently get a decisive answer to this question; meanwhile I hope to excite your interest by telling you what I expect to find. It is quite certain that peritonitis was the immediate cause of death; and I think it probable that this intense peritonitis was excited by the escape of some irritant into the cavity of the peritoneum. Beyond this we cannot go with certainty; but my theory is that the attack of pain, with vomiting, high-coloured urine, and jaundice, which occurred at the commencement of her illness, was caused by the passage of a gall-stone into the common duct. Then I think it probable that the gall-stone got impacted, and, causing ulceration of the duct, escaped together with some bile into the cavity of the peritoneum, and excited peritonitis. The further escape of bile would be prevented by the effusion of lymph and resulting adhesions to the neighbouring parts. The peritonitis was subsiding, and all was going on well until, as I suppose, about a week before her death another gall-stone passed into the common duct, which it obstructed, causing a return of the pain, vomiting, bile-tinged urine, and jaundice, and then it

followed its predecessor into the cavity of the belly, thus exciting the fatal increase of the peritonitis. We will now go to the *post-mortem* theatre.

Inspection by Dr. Pritchard Forty Hours after Death.—On opening the abdomen, the viscera were found firmly matted together by recent lymph, so that their separation was effected with great difficulty. Pus and purulent lymph were found in many places, there being a large collection in the left iliac region. Amongst the puriform effusion near the under surface of the liver was found *a globular hydatid*, about the size of a marble. The liver weighed eighty-three ounces. On its surface it appeared healthy, but in the back part of the left lobe there was a hydatid cyst about three inches and a half in diameter, containing numerous secondary hydatids of various sizes. The cyst communicated with the hepatic and the common duct, both of which were dilated to the size of the little finger, and a catheter readily passed from the opening of the duct in the duodenum into the hydatid cyst. When the cyst was pressed, the hydatids passed one after another along the duct in the duodenum. The cystic duct was nearly obliterated, and the gall-bladder contained only mucus. There had been no ulceration or rupture of the bile-duct, but it appeared that the wall of the cyst had given way on its under surface, by a small opening which led into a honeycombed mass of lymph and adhesions.

Concluding Remarks.—You see, then, gentlemen, that I was right in my diagnosis of peritonitis, and I was also right in my conclusion that the peritonitis was excited by the escape of an irritant substance into the cavity of the peritoneum, consequent on plugging of the bile-duct; but I was wrong in supposing that a gall-stone had been the cause of the block. It is evident, from the dilated condition of the hepatic and common duct, that numbers of hydatids had passed through these canals. On two occasions—when the pain, sickness, and jaundice first occurred, and again a few days before her death—it is probable that a hydatid of larger size caused a temporary block of the duct, and one result of this was distension of the cyst by accumulating secretions, and rupture of the cyst, with a partial escape of its contents into the

cavity of the peritoneum. The aperture, which was temporarily closed by lymph after the first escape, was reopened when the second block and resulting distension occurred. Thus the symptoms are completely explained. The stools were repeatedly examined after her admission into the hospital, but nothing decisive was found. The position of the cyst at the back of the liver was such that it could not be felt during life. The only evidence that might have led to the discovery of the cause of the symptoms, so closely resembling those which result from the passage of gall-stones, would have been the detection of one or more hydatids in the stools. If any of you should, hereafter, meet with a similar case, your recollection of this one may assist you in your diagnosis; and, of course, you will not omit to make a careful examination of all the matters discharged by vomiting and by stool.

CHAPTER XLIV.

A CLINICAL LECTURE ON THE TREATMENT OF THE DIARRHŒA OF TYPHOID FEVER.[1]

Injurious Results of Opiates in the Treatment of the Diarrhœa of Typhoid —Contrasted Results of Treatment without Opium—Diarrhœa sometimes caused by Undigested Beef-tea and Eggs—Twenty-nine Consecutive Cases of Fever without a Death—Reply to Objections—The Statistics of Fever Cases during Five Years—Cold Wet Pack in a Case of High Temperature—Opium in Small Doses to soothe, but not to arrest the Discharges.

The diarrhœa of typhoid fever, as it is one of the most frequent symptoms of the disease, so is it one of the most troublesome, and one which often causes the greatest anxiety. It is a fact generally admitted that, in the great majority of cases, the severity and danger of typhoid fever are in direct proportion to the intensity and duration of the diarrhœa. Delirium and other serious cerebral symptoms, pulmonary engorgement, and renal congestion with albuminuria, are comparatively infrequent complications. The treatment of diarrhœa, then, forms a very important part of the management of the disease. During the many years of my connection with this hospital I have had the opportunity of seeing the diarrhœa of typhoid fever treated in very different ways and with very different results; and I propose now to give you, in a few sentences, the results of my experience with reference to this important practical subject.

For many years the practice strongly advocated by Dr. Todd was generally adopted throughout the hospital. This consisted in persevering attempts to arrest the diarrhœa by repeated doses of opiates and other powerful astringents. It was then a common practice to give an enema containing

[1] The *Practitioner*, January 1875, vol. xiv. p. 1.

from ten to fifteen or twenty drops of laudanum after each liquid stool. The result of this treatment, in a large proportion of cases, was that the diarrhœa continued in spite of the repressive treatment, and meanwhile the intestines were distended with gas, and the abdomen became tumid and tympanitic. Then the patients were tortured by the application of turpentine stupes to remove the tympanitis. The results were altogether most unsatisfactory. Nor is it difficult to explain the failure of this opiate treatment. Without entering upon the consideration of disputed pathological theories, it can scarcely be doubted that one effect of opium must be to render the intestines torpid and to lessen their expulsive efforts; and as a result of this, their putrid contents are retained until they decompose and give off noxious gases, by which the bowel is distended and irritated, and so the diarrhœa is perpetuated and increased. It is pretty certain that the healing of the ulcers must be impeded by the continual contact of the fœtid morbid secretions, and that the distension of the bowel must cause pain and increase the risk of fatal perforation or rupture.

Now for a number of years we have entirely changed our treatment, and I have gradually arrived at the conclusion that in the treatment of typhoid fever careful nursing and feeding are of primary importance, while, as a rule, no medicines of any kind are required, and when not required they are often worse than useless. The result of this change of treatment has been that diarrhœa is a less frequent symptom than formerly, and when it does occur it is far more tractable, while tympanitic distension of the abdomen is a rare event. The mischievous opiate enemata and the torturing turpentine stupes have disappeared together. I believe that one of the main reasons why we have less diarrhœa than formerly is, that we carefully abstain from the employment of irritating drugs of all kinds. As a rule, a fever patient has the 'yellow mixture,' which is simply coloured water; and, except an occasional dose of chloral to procure sleep, and a tonic during convalescence, we give no active medicines of any kind. We feed these patients mainly with milk, with the addition of beef-tea and two raw eggs in the twenty-four hours, and we

give wine or brandy, in quantities varying according to the urgency of the symptoms of exhaustion, especially in the advanced stages of the disease; but in many of the milder cases, and especially in the case of children, we find that no alcoholic stimulants are required, from the beginning to the end of the fever, and when not required they are of course best withheld.

I have said that we give no irritating drugs of any kind. For a time I adopted the practice, which has been strongly recommended, of giving repeated doses of diluted mineral acids. I have long since abandoned this practice; for I am sure that it was injurious, and it was injurious in a very obvious and intelligible way: it irritated the ulcerated mucous membrane of the intestines, it caused pain and griping, and I believe that it often increased the diarrhœa. I have no doubt that the comparative infrequency of severe and obstinate diarrhœa amongst my enteric fever patients, during the last few years, is partly attributable to the discontinuance of this mineral acid treatment. The extreme sensitiveness of the intestinal mucous membrane, during the progress of typhoid fever, is obvious and indisputable. It is admitted, on all hands, that the greatest care is required in returning to solid food during convalescence; a want of caution in this respect has often been followed by a return of pain and diarrhœa, an increase of temperature, and not seldom by a decided relapse. If, then, a slice of bread or a morsel of fish can excite such local and general disturbance, even after the subsidence of the fever, how improbable is it that repeated doses of an irritating mineral acid can be given without injury, during the height of the fever, when the ulceration of the intestines is actively progressing!

One more hint I wish to give you with regard to the diarrhœa of typhoid fever, which is, that in all probability it is often increased by the patient's inability to digest the beef-tea and eggs which are sometimes too abundantly given. When you have reason to suspect that this may be the case, I advise you, for a few days, to keep the patient entirely upon milk, which contains all the elements required for the nutrition of the tissues in a form most easy of digestion. I have had a

large experience of the effects of an exclusively milk diet in various forms of disease. In many cases of Bright's disease it is very efficacious; but one of the inconveniences in some of these cases is its tendency to cause troublesome constipation. In many cases of chronic diarrhœa and dysentery, milk diet will effect a cure without the aid of medicines of any kind. There is now in Twining ward a girl, aged 14, who for four months had been suffering from dysenteric diarrhœa, the stools containing much blood and mucus. She was put upon a diet of milk alone, without medicine: within a fortnight the diarrhœa entirely ceased, and she is now convalescent. For the reason, then, that milk has this antilaxative and even constipating effect in various morbid states, it is, when given alone, one of the best antidotes for the diarrhœa of typhoid fever.

That our treatment of fever cases is not unsuccessful is shown by the results. I find on reference to my case-books that during the past year, from November 1, 1873, to October 31, 1874, I have had under my care in the hospital twenty-nine cases of fever—fifteen typhoid, and fourteen typhus. Some of the cases have been very severe, but all have been discharged well; not one death has occurred. This very satisfactory result I attribute mainly to the admirable nursing which our patients receive, and to our abstinence from mischievous medication. To only one of these patients was opium given, and that was for the relief of an irritable condition of bowel which remained after a very severe attack of typhoid. A few doses of opium soon put a stop to this, and the patient made a good recovery.

The preceding lecture having been published in the *Practitioner*, Dr. Kennedy, in a subsequent number of the journal, expressed his disapproval of my treatment, to which I replied as follows:[1]—

In the *Practitioner* for June, Dr. Henry Kennedy comments on my paper, and expresses his belief that 'it embodies a doctrine which is most questionable, and in every way open

[1] Vol. xv. p. 107.

to discussion.' Be it so. I am not about to enter into a controversy with Dr. Kennedy, but I beg to suggest that his objections to my method of treatment would have more weight if he could have said that he had fairly tried it and had found it unsuccessful. In the absence of such experience I venture to say that his adverse judgment is worth very little. I have, at any rate, this advantage over him, that I have myself tried and have seen others follow out the methods of treatment in which he places his trust, and I have abandoned them because I have found them worse than useless. As I stated in my former paper, I do *not* trust entirely to diet and good nursing. I give alcohol in various forms, chloral, quinine, and even opium when required, but not as a matter of routine or without a clear indication of their necessity.

There is but one objection raised by Dr. Kennedy which I consider to have any weight, and that is his statement that the fact of fifteen recoveries occurring in succession affords no proof that the treatment was successful—the numbers being too small to have any value as evidence. To meet this objection our Medical Registrar, Mr. Batterbury, has done me the favour to look through our records and extract for me all the cases of typhoid and typhus fever that have come under my care in the hospital during the last five years, from the beginning of the year 1870 to the end of the year 1874. The result is that there have been during that period sixty-three cases of typhoid with three deaths, and twenty-six cases of typhus with one death. As I have before stated, I attribute this very satisfactory result mainly to the admirable nursing which our patients receive, and to our abstinence from all medication except such as is required to meet particular symptoms. In some of my cases the symptoms were of extreme severity. In one, the temperature having risen above 107°, life was saved only by the frequent repetition of the cold wet pack, which was administered with great skill and perseverance by the then house physician, Dr. Bomford.

Everyone whose opinion is of any weight admits the supreme importance of careful feeding in cases of typhoid fever, not only during the progress of the fever, but also until convalescence is thoroughly established. It is not so generally

known or admitted that the symptoms—the intestinal symptoms especially—are often aggravated by the irritant action of such drugs as mineral acids, and by the retention of fœtid morbid secretions, when the intestines have been rendered torpid by opiates indiscreetly given to arrest diarrhœa. Whether any medicine has the effect of checking the outpour of morbid secretions from the bowel into the intestines, and what would be the effect of such restraint, is doubtful. It is not doubtful that the artificial retention of foul liquids and gases within the bowel must be an unmixed, and often a very serious evil.

[It should be understood that my objection to the use of opium in the treatment of the diarrhœa of typhoid applies, not to the giving of small doses, to soothe or to act as a stimulant, but to the employment of large doses, with the object of arresting the discharges, and with the result of distending the bowel by the morbid secretions and the gases which result from their decomposition.]

CHAPTER XLV.[1]

LECTURES ON BRIGHT'S DISEASE.

SECTION I.

THE MINUTE ANATOMY AND PHYSIOLOGY OF THE KIDNEY.

Cortical and Medullary Portion—Uriniferous Tubes—Malpighian Bodies—Arrangement of the Blood-vessels—Basement-membrane—Epithelium in various Parts of the Tubules—Matrix—Connective Tissue—Nerves—Function of Kidney—Bowman's Theory—Heidenhain's confirmatory Experiments—Source of the Urinary Constituents—Physical Characters of the Urine—Mechanism of Albuminuria.

An exact knowledge of the structure and functions of the kidney is essential for a correct interpretation of its diseases. I therefore beg first to direct your attention to certain points of anatomy and physiology which will be found hereafter to have a direct bearing upon important pathological questions.

A longitudinal section of the kidney shows it to be composed of a cortical and a medullary portion. The medullary portion is arranged in the form of cones or pyramids—pyramids of Malpighi—usually from twelve to fifteen in number, the bases of which are directed outwards towards the surface of the gland, becoming gradually continuous with the cortical portion; while the apices are directed inwards towards the cavity or pelvis of the kidney. The cortical portion occupies the entire surface of the organ, forming a layer about two lines in thickness opposite the bases of the medullary cones, and sending prolongations inwards between the cones; so that each medullary cone is surrounded, except at its apex, by the cortical portion of the gland. The kidney is a tubular gland. The tubes of the cones take, for the most

[1] This chapter is, in fact, a new edition, revised and in part rewritten (with additional illustrations on wood), of my *Lectures on Bright's Disease* (Smith, Elder, and Co. 1873).

part, a straight course; while those of the cortex are extremely convoluted and tortuous. Tracing the tubes from the apex of a medullary cone, on the surface of which their open mouths may be seen, they are found to take a straight course through the pyramid, branching dichotomously, and diverging from each other as they proceed. After reaching the base of the pyramid, their course through the cortical portion varies: many tubes immediately become very tortuous, some of them bending down into the interpyramidal portions of the cortical substance, while others pass on, in sets and in straight lines towards the surface; the tubes on the sides of each bundle diverging successively, and then taking a tortuous course through the cortical substance, so that only a few of the central tubes in each bundle retain their straight course quite up to the surface of the kidney. These all finally turn backwards, making many convolutions in the cortical portion of the gland. After leaving the medullary cones, the branching of the tubes, except in very rare instances, appears to cease. In all the numerous sections of the kidney that I have examined, I have never seen a convoluted uriniferous tube either branching or anastomosing with another tube. Some of the convoluted tubes dip down amongst the straight tubes, forming loops with their convexities towards the apex of the pyramid. Henle supposed that these looped tubes were closed at both ends, and therefore quite distinct from those which open into the pelvis of the kidney. There is, however, good anatomical evidence that, as each convoluted uriniferous tube, at one extremity, forms a globular dilatation, which constitutes the capsule of the Malpighian body, so at the other end it passes into a straight tube which opens into the pelvis of the kidney. For an excellent criticism of Henle's views and of some curious speculations which others have based upon them, I refer you to Dr. Beale's book on *Kidney Diseases*, &c. (p. 10). These speculations have no practical bearing upon the diagnosis or the pathology of renal diseases, and I shall not refer to them further.

We have next to trace the very remarkable arrangement of the blood-vessels within the kidney. The renal artery, entering the hilum of the kidney, sends small branches to the areolar and adipose tissue outside the pelvis, and then, passing

into the substance of the organ, breaks up into terminal branches, which, with a few exceptions to be presently mentioned, correspond in number with the Malpighian bodies. Each terminal artery—the *afferent artery* of the Malpighian body—perforates a Malpighian capsule, and thus passes within the dilated end of the uriniferous tube. It there breaks up into loops of capillaries, which take a more or less tortuous course; these again unite into a single *efferent vein*, which leaves the Malpighian capsule near the entrance of the afferent artery; it then enters the capillary plexus which lies

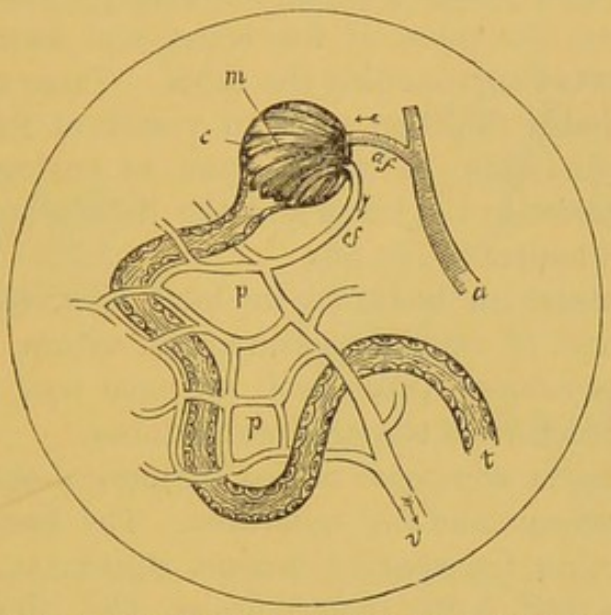

FIG. 6.—PLAN OF THE MINUTE STRUCTURE OF THE KIDNEY.

a. artery, sending an afferent branch, *a f*, which breaks up into *m*, the Malpighian capillaries. *e f*. efferent vessel, which conveys the blood from the Malpighian capillaries into *p*, the plexus of capillaries between the tubes. These again unite, and form the vein *v*. *t*. the uriniferous tube. *c*. the capsule of the Malpighian body. The course of the circulation is indicated by the arrows.—× 60.

outside the tubes—the *intertubular plexus* (see fig. 6). The intertubular capillaries anastomose freely on all sides, so as to form a continuous network, whence the blood is ultimately collected into the commencing branches of the renal vein. The course of the circulation through the kidney, then, is as follows:—The blood passes from the renal artery through the afferent arteries into the Malpighian capillaries; from these it is carried by the efferent veins to the intertubular capillaries; and thence it passes out of the kidney by the renal vein. The greater part of the blood which passes into the interior of the

kidney takes the course which I have described; but amongst the straight tubes of the pyramids there are certain *vasa recta* which have a different distribution. Some of these *vasa recta* are efferent veins from Malpighian bodies near the bases of the pyramids, which, as originally described by Bowman, take a straight course towards the apices of the cones, and terminate in capillaries, from which the blood is returned by venous radicles, which also take a straight course and join the renal vein. But, in addition to these venous branches, it has been shown by Virchow, Beale, and others, that there are arterial *vasa recta* which pass off from the artery, take a straight course between the tubes of the cones, and terminate in a capillary network surrounding the tubes. These arterial *vasa recta* are probably the chief nutrient vessels of the pyramids. They may, therefore, be looked upon as analogous to the bronchial arteries in the lungs and the hepatic artery in the liver. (See Chapter IV. p. 53.)

Each Malpighian body, as we have seen, consists of a globular plexus of capillaries contained within the dilated end of a convoluted tube; and we have now to consider briefly the structure of the uriniferous tubes.

Each tube is composed of two anatomical elements—the *basement-membrane* and the *epithelium*. The basement-membrane is a thin transparent lamina, appearing, as a rule, structureless and quite homogeneous, the slightly fibrous appearance which it sometimes presents being probably due to contraction and corrugation by the sections which must of necessity be made for microscopic examination. This membrane is in direct contact on its outer surface with the intertubular capillaries, and on its inner surface with the epithelial lining of the tubes.

The epithelium in the convoluted tubes differs from that in the straight tubes. In the convoluted tubes, the epithelium is of the true glandular character. The cells are somewhat angular in outline; and between this and the central nucleus there are a number of granular particles. The cell-wall is often indistinct, and readily disintegrated by the action of water. The cells form a single layer within the tubes; and this cell-lining occupies from one-third to one-half of the

diameter of the tube, leaving a clear canal in the central axis of the tube. (Fig. 7.)

The epithelium in the straight tubes is flatter, less granular, and has more the character of pavement-epithelium; so that the clear canal within a small straight tube is wider than that of a convoluted tube of larger size.

At an early period of development of the mammalian kidney, a delicate epithelium may be seen lining the Malpighian capsule and covering the capillary loops.

Some writers describe a delicate connective tissue binding together the Malpighian capillaries, so that the glomerulus,

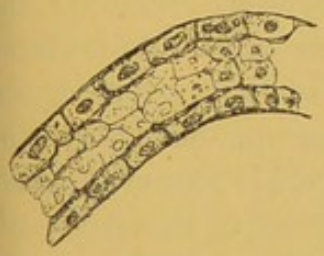

FIG. 7.—PORTION OF A CONVOLUTED URINIFEROUS TUBE.

The lining of glandular epithelium leaves a clear canal in the middle, which is equal to about half the diameter of the tube. —× 200.

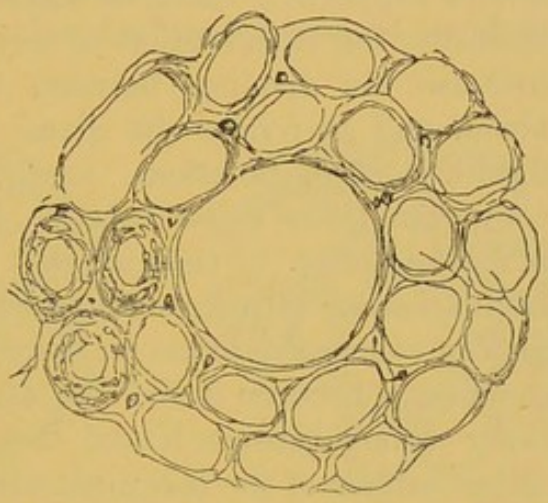

FIG. 8.—SECTION OF THE CORTEX OF THE KIDNEY, AFTER WASHING IN WATER TO REMOVE THE GLAND-CELLS.

The smaller rings are sections of the basement-membrane of the tubes; the larger ring is a section of a Malpighian capsule. In three sections of a tube the gland-cells remain. Sections of capillaries are seen here and there in the angular spaces between the tubes.—× 200.

when picked out from the surrounding capillaries, maintains its original form and does not collapse.

In the kidney of the newt and the frog a delicate layer of ciliated epithelium may be seen within that portion of the Malpighian capsule which lies next to the opening of the tube; and, in the newt's kidney, vibratile cilia may be seen throughout the entire length of the uriniferous tubes.

The appearance to which Goodsir originally gave the name of the *matrix* of the kidney has been a source of much perplexity to anatomists and pathologists. Fig. 8 represents an appearance which results from washing a thin section of the

cortex of an uninjected kidney in water, so as to remove the gland-cells. The appearance is that of a fibrous network enclosing circular and oval spaces. The explanation of the appearance is this. The tubes lie in close contact with each other, having the intertubular capillaries between them. A thin transverse section gives a reticular appearance; the rings being formed by the basement-membrane of the tubes, with the capillaries in the interspaces and angles. The so-called matrix has no existence apart from the basement-membrane and capillaries. The convolutions of the tubes and the network of capillaries mutually support each other. No connective or supporting tissue is required; and, as Dr. Beale well remarks, the intervention of any such tissue would tend to increase the distance between the secreting cells and the blood, and so render the gland less perfectly fitted for the discharge of its function. Ludwig states that 'no fibrillated connective tissue exists between the tortuous portions of the urinary tubules.' [1] Only isolated fusiform cells lie between the capillaries and the convoluted tubes. There is no more appearance of connective tissue on the *outer* surface of the basement-membrane between it and the capillaries than there is on the *inner* surface between it and the gland-cells. The tissues on either surface of the basement-membrane adhere to it without the intervention of another tissue to which the term connective tissue can be given (see fig. 9). If there be any connecting medium it is a homogeneous and structureless element between the convoluted tubes. The medullary cones contain a fibrous connective tissue separating the straight tubules, and increasing in amount towards the apices of the cones.

You may make a coarse imitation of the fibrous network of the kidney by taking half a dozen india-rubber tubes, and cementing them together side by side, so as to form a bundle of parallel tubes. Transverse sections will then form a network, the rings of the meshes being formed by the divided india-rubber tubes, as the reticular appearance in the kidneys is the result of sections of the basement-membrane of the uriniferous tubes.

[1] Stricker's *Manual of Histology*, New Sydenham Society's Translation, vol. ii. p. 106.

Some connective tissue passes in with the blood-vessels from the pelvis and calices, and some passes off from the fibrous capsule into the substance of the cortex. It is especially abundant in the divisions between the lobes, which in the earlier stage of development are quite distinct, and separated from each other by deep fissures.

Bear in mind, then, that there is no distinct structure to which the term 'matrix' can be applied. The fibrous appearance represented in fig. 8, which has been often described as a morbid formation of fibrous tissue surrounding and constricting the tubes, is, I hope, rendered quite intelligible by the description which I have given you.

The diameter of the convoluted tubes is remarkably uniform,

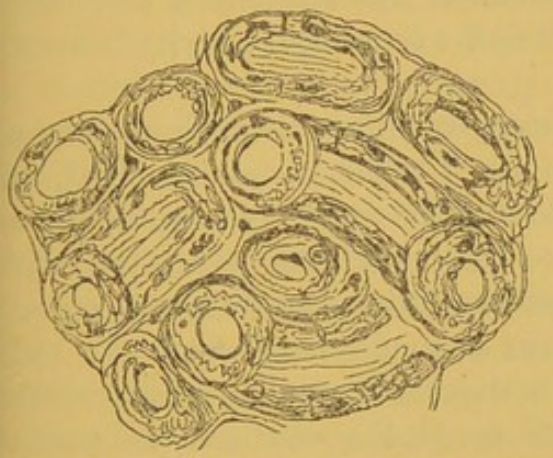

FIG. 9.—SECTION OF THE CORTEX OF THE KIDNEY.
The gland-cells are here attached to the inner surface of the basement-membrane. The light interspaces between the gland-cells of adjacent tubes correspond with the rings of the basement-membrane in fig. 8.—× 200.

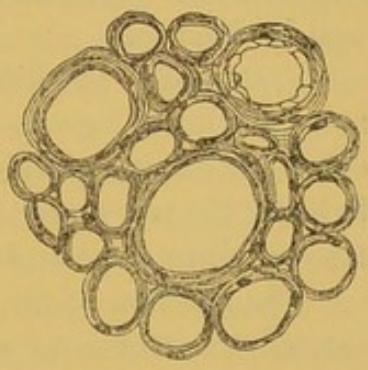

FIG. 10.—SECTION OF A MEDULLARY CONE.
The rings which are here of unequal sizes are sections of the straight tubes. In one section the epithelium remains.—× 200.

and equals about one five-hundredth of an inch. That of the straight tubes is much more variable. While many straight tubes have a narrower outline than the convoluted tubes, but a wider canal, in consequence of the more flattened form of the epithelium, others, especially near the apex of a cone, are more than twice as large as the convoluted tubes. This may be seen by a comparison of figs. 8 and 10, the latter representing a section of the straight tubes in a cone. The basement-membrane of the straight tubes is somewhat thicker than that of the convoluted tubes, and the medullary cones are firmer than the tissue of the cortex.

Nerves.—The nerves of the kidney are chiefly derived from the sympathetic. In man and in the higher animals it is difficult to trace their distribution; but in the kidney of the newt Dr. Beale has found that not only are the terminal branches of the nerves distributed to the small arteries and veins, but also to the convoluted tubes and to the Malpighian and intertubular capillaries. The nerve fibres are all connected with ganglion-cells, from each of which two or more fibres proceed in different directions, and so establish a communication between various parts of the organ. It is probable, as Dr. Beale suggests, that the nerves which are distributed over the uriniferous tubes constitute an afferent system, which, through the nerve-centres and the efferent nerves distributed to the arteries, are capable of influencing and regulating the blood-supply to the capillaries, and so the functional activity in health and in disease.

Practically, the kidney may be said to be made up of two sets of tubular vessels—one set of tubes containing blood, the other containing gland-cells; and the organ is so constructed as to bring the two sets of tubes—the sanguiniferous and the uriniferous—into close and intimate relationship with each other.

The function of the kidneys is to discharge from the body superfluous water, together with certain peculiar urinary solids. There appears no reason to doubt the essential accuracy of Mr. Bowman's original doctrine that, while the convoluted tubes, with their lining of gland-cells, are the agents by which the solids of the urine (the urea, uric acid, &c.) are secreted, the watery portion of the secretion is, chiefly filtered through the Malpighian bodies.

The convoluted tubes resemble, in all essential points, the secreting tissues of true glands, and especially in the character of their epithelial cells; while the Malpighian bodies, in their structure and arrangement, form a striking contrast. The epithelial cells either cease altogether or entirely change their character within the Malpighian capsules. The Malpighian capillaries lie within the dilated ends of the tubes, and are covered, if at all, only by the most delicate epithelium. 'It would be difficult,' as Mr. Bowman says,[1] 'to conceive a disposi-

[1] *Philosophical Transactions*, 1842.

tion of parts more calculated to favour the escape of water from the blood, than that of the Malpighian body. Each afferent artery breaks up into a number of minute capillaries of far greater aggregate capacity than itself. Hence must arise an abrupt retardation of the blood-stream. The vessels in which this delay occurs are uncovered by any structure. The interior of the capsule, certainly in the lower animals, and probably in the higher, is lined by cilia whose motion directs the current of liquid towards the orifice of the tube. Why is so wonderful an apparatus placed at the extremity of each uriniferous tube, if not to furnish water, to aid in the separation and solution of the urinous products from the epithelium of the tube?'

The epithelium of the straight tubes, as I have before mentioned, is allied to the lamelliform or pavement variety. It probably has no glandular function, the tubes which form the medullary cones being merely ducts for conveying away the secreted products from the convoluted tubes into the pelvis of the kidney.

The ingenious and most interesting experiments of Heidenhain [1] afford confirmatory evidence that the epithelium of the convoluted tubes secretes the solid constituents of the urine. He injected indigo-carmine into the veins of animals in which the secretion of urine had been arrested by division of the spinal cord below the medulla. By killing the animals at variable intervals after the injection, he ascertained that the pigment was separated from the blood by the renal epithelium, and thence passed into the tubules, where it was precipitated in the form of solid particles. While there was no trace of the pigment having passed through or stained the Malpighian capillaries, the cells which could be seen to have taken up and ejected it were those lining the convoluted tubes, which from their microscopic characters are believed to have an active secretory function. As regards this particular substance then, it is certain that the epithelial cells of the convoluted tubes, in the discharge of an excretory and not a formative function, are the means by which it is withdrawn from the blood; and in like manner it may be inferred the urinary solids are excreted.

[1] Pflügers *Archiv*, ix. (1874), quoted in Foster's *Physiology*, 3rd ed. p. 375.

The precise mode in which the glandular epithelium separates its peculiar products from the blood and discharges them into the duct is a mystery which has not yet been solved.

It is probable that the epithelial cells are continually becoming liquefied and passing away with the secretion, and that they are constantly replaced by new formation; but whatever may be the process by which these changes are effected, no entire gland-cells, nor even the débris of renal epithelium, are visible in normal urine. The appearance of renal epithelium in the urine affords unquestionable evidence of a pathological process.

Conflicting results have been obtained by different experimenters in their attempts to solve the question whether the peculiar urinary constituents exist ready formed in the blood and are only separated by the kidney, or whether they are formed, wholly or in part, by the gland. It had long been the accepted doctrine that urea and uric acid exist normally in the blood, that they are thrown out by the kidneys, and that they accumulate and cause uræmia when the secretory function of the kidney has been impaired by disease. Dr. Oppler, of Berlin, threw a doubt upon this doctrine. He found, as he believed, much more urea in the blood of dogs whose ureters had been tied than in the blood of those whose kidneys had been extirpated, and he concluded that the excess was due to the formation of urea by the kidneys in the first class of cases.

More recent experimenters have obtained different and apparently more trustworthy results. Thus Gréhant and Gscheidlen, quoted by Foster,[1] found a large and an equal excess of urea in the blood whether the kidneys were extirpated or the ureters tied. In the latter case the distension of the tubules soon destroys the epithelium, so that an animal with ligatured ureters is practically in the same condition as one from which the kidneys have been removed.

While these and other experiments prove conclusively that urea and uric acid exist ready formed in the blood, their primary source being doubtful, the question remains for solution whether any of the urinary constituents are actually

[1] *Physiology*, p. 404.

formed and not merely excreted by the kidneys. For all practical purposes it is sufficient to be assured that the urinary constituents are largely brought to the kidneys by the blood, whence they are discharged through the uriniferous tubes of the gland, and that hence arises the contamination of the blood by urinary materials when the kidneys are structurally changed and their excretory function suspended or much impaired.

Physical Characters of the Urine.—Healthy urine is a transparent sherry-coloured liquid, having an acid reaction and a density usually ranging between 1015 and 1025, but it may temporarily fall much below or rise considerably above these limits without being morbid. The daily secretion of urine has been estimated by some observers to be as low as 35 ounces; by others as high as 81 ounces;[1] the mean being 50½ ounces. The amount secreted depends upon the measure of fluid taken in and the amount passed off by other channels, especially by the skin.

Referring you for a detailed account of the chemistry of the urine to the works of Parkes, Thudichum, Beale, &c., I may remind you in passing that, as the lungs and the liver are large eliminators of carbon, so the urinary secretion is remarkable for the abundance of its nitrogenous constituents. Urea, the chief urinary solid, contains a large proportion of nitrogen, and the amount of urea discharged by an adult male in twenty-four hours ranges, according to different observers, from 286 grains to 688 grains, the mean being 512 grains.[2]

The Mechanism of Albuminuria.—Now, before I proceed further, let me show you, by referring to the anatomy of the kidney, that the peculiar position of the Malpighian capillaries, within the dilated ends of the uriniferous tubes, is attended with this result, that any interference with the circulation through the kidney is apt to be associated with an escape of blood-constituents through the Malpighian capillaries, which, mingling with the urine, render it either bloody, or, if the serum alone escape, simply albuminous.

Looking at the plan of the renal circulation (fig. 6, p. 627), you see that, whether the escape of blood constituents be

[1] Parkes *On the Urine*, p. 5.

[2] Parkes, pp. 7 and 8.

traceable to an altered physical relation between the blood and the walls of the vessels, or to engorgement of the Malpighian capillaries—the result of an increased afflux of blood through the arteries or of an impeded return of blood through the intertubular capillaries and veins, consequent on an obstruction within the kidney itself, or beyond, in the heart or lungs —in each and every case the blood-materials, transuding through the walls of the Malpighian capillaries into the tubes, mingle with the urine and render it bloody or albuminous. There are many interesting points of analogy between the liver and the kidney as regards structure, functions, and pathology; but in the liver there is nothing analogous to the intratubular Malpighian capillaries, and therefore, while albuminuria is of very common occurrence, an albuminous or sanguineous condition of the bile is a rare event.

SECTION II.

ON THE VARIOUS MODES OF TESTING FOR ALBUMEN IN THE URINE.

Heat: Mode of Applying—Fallacies.—Nitric Acid: Mode of Using—Fallacies —A Drop of Nitric Acid prevents Coagulation by Heat—Excess of Nitric Acid redissolves Albumen—Mode of Testing for a Minute Trace of Albumen. —Picric Acid as an Albumen Test—Mode of using in Solution—Comparison with Nitric Acid—Distinction between Albumen and Urates—Distinction between Albumen and Peptones—Picric Acid as a Test for Peptones—Detection and Separation of Peptones when mixed with Albumen—Vegetable Alkaloids precipitated by Picric Acid, but redissolved by Heat—Mucin is *not* Precipitated by Picric Acid—Practical Advantages of a delicate Test for Albumen—The Fallacy of Testing a *Morning* Specimen only—Influence of the Erect Posture and Exercise in the Production of Albuminuria—The Urine in Cases of Small Red Kidney rarely free from Albumen—Boiling after adding Acetic Acid sometimes fallacious—The smallest Trace of Albumen is Pathological—Estimation of the Amount of Albumen—History of the Use of Picric Acid as a Test for Albumen.

I propose now to describe, demonstrate, and critically review the various modes of testing for albumen in the urine, in the light of recent knowledge and research.

The two tests for albumen which have hitherto been most generally used are heat and nitric acid. I pour into a test-tube a column of urine about two inches in height, and place

it over a spirit-lamp. Before the liquid reaches the boiling point, the albumen begins to coagulate; and the more abundant the albumen, the lower is the temperature at which coagulation commences. When the urine is highly albuminous, as the heating proceeds and coagulation commences, the tube must be continually and briskly shaken, to prevent the sudden expulsion of the partially coagulated liquid by the steam generated beneath a dense film on the surface. Now observe that, when albuminous urine is alkaline, heat alone will not show the presence of albumen. I take some of this albuminous urine, and before applying heat, I add a drop or two of liquor potassæ. You see that, when boiled, it remains clear; the potash has combined with the albumen, and the resulting albumanite of potash is not coagulable by heat; but if now I add a few drops of strong nitric acid, coagulation at once takes place.

Again, some specimens of urine, when boiled, give a precipitate which has the appearance of albumen, though none is present. The non-albuminous urines which are thus acted on by heat, are usually neutral or alkaline, and the turbidity is due to the precipitation of phosphates. To a specimen of normal urine I add a drop or two of liquor potassæ, and, when heated over the lamp, you see that it becomes turbid.

This phosphatic turbidity is easily distinguished from an albuminous coagulum by the addition of a drop or two of nitric acid, or even of acetic acid, or citric acid solution, which dissolves the phosphates while coagulated albumen remains undissolved.

You see, then, that if heat alone were relied upon as a test for albumen, it might mislead in two opposite directions: (1) by not detecting albumen in alkaline urine, and (2) by giving a delusive phosphatic precipitate in non-albuminous urine.

Nitric acid has long been known and employed as a valuable test for albumen. To this clear specimen of urine I add a few drops of strong nitric acid, and an abundant coagulum of albumen is immediately formed. In most cases, nitric acid alone is a sufficient test for the presence of albumen in the urine, but there are some exceptional conditions, and some sources of fallacy, which I now proceed to point out.

Here is a specimen of albuminous urine, which, as you see, is highly turbid with urates. As I warm it over the lamp, it first becomes clear by the solution of the urates, and then, by the further application of heat, the albumen coagulates, and renders the urine milky. Such a specimen requires to be cleared by heat before nitric acid can be satisfactorily used as a test; although, as you see, a slight excess of nitric acid added to the turbid urine decomposes and dissolves the urates, and then precipitates the albumen.

It sometimes happens that, when nitric acid is added to highly acid urine, it causes a turbid deposit of urates, which might be mistaken for an albuminous coagulum. The distinction is readily made by the application of heat, which dissolves the urates, but not the albuminous precipitate.

In the urine of patients who are taking copaiba, nitric acid, acting on the resin, causes a slight milkiness, which has sometimes been mistaken for albumen. Such urines have a peculiar resinous odour; although acid, they are not coagulated by heat, nor, as I shall presently show you, by picric acid, which is a most delicate test for albumen.

When an excess of nitric acid is added to a highly concentrated urine, it sometimes happens that a copious crystallisation of nitrate of urea gradually occurs. This slowly formed crystalline deposit could scarcely be mistaken for an amorphous coagulum of albumen.

With regard to nitric acid as a test for albumen I have yet to show you certain facts which require attention. To this specimen of highly albuminous urine I add a single drop of strong nitric acid. You see that it forms a white coagulum which, on shaking the tube, is redissolved; a second drop may be added with the same result, while the further addition of acid causes a permanent precipitate. Now, note this fact, that, when a drop or two of nitric acid has caused a coagulum which has been redissolved, the subsequent application of heat will not precipitate the albumen. The explanation is, that a nitrate of albumen has been formed, which is not coagulable by heat, but readily by an excess of the acid. Albumen is a neutral substance which is capable of combining,

on the one hand, with alkalies, as we have before seen; and, on the other, with acids; and, in both instances, to form compounds not coagulable by heat.[1]

As a drop or two of nitric acid will prevent the coagulation of albumen by heat, so a *great excess* of nitric acid added to the urine will either redissolve the coagulum which first forms, or, if rapidly poured in, entirely prevent coagulation, the mixture remaining quite clear.

Nitric acid, carefully added to cold urine, is one of the most delicate tests for a slight trace of albumen in the urine. For this purpose, a few drops of the acid may be placed at the bottom of a test-tube, and the urine slowly poured into the sloping tube, so as to rest on the surface of the acid; or, the urine being first placed in the tube, a few drops of the heavy acid are allowed to fall through the urine to the bottom. The opalescent albuminous coagulum appears at the junction of the two liquids.

When the amount of albumen is small the opalescence with nitric acid does not appear until the tube has been allowed to stand for a few minutes, and is then looked at with a dark background.

In *picric acid* we have a more delicate test for minute traces of albumen than either heat or nitric acid, or than both these tests combined. Picric acid is used in the form of a saturated aqueous solution. An ounce of water at 60° Fahr. retains in solution 5·3 grains of the dry acid. A saturated aqueous solution may, therefore, be made by dissolving the powder in boiling distilled or filtered rain-water, in the proportion of six grains to the ounce. A portion of the acid will crystallise out on cooling, leaving a transparent yellow supernatant liquid.

I now show you that this solution of picric acid, being added to an equal volume of albuminous urine, in a test-tube, immediately coagulates the albumen. The coagulum is made more dense by the subsequent application of heat. The coagulated albumen picrate is soluble in alkalies. If, therefore,

[1] The influence of a small amount of nitric acid in preventing the subsequent coagulation of albuminous urine by heat was first published by Mr. George Padley, of Swansea, in the *Medical Gazette*, 1845, p. 242.

the albuminous urine be highly alkaline, it is necessary to acidulate it with acetic or citric acid, before adding the picric solution. This, however, is very rarely necessary. The picric acid solution is itself sufficiently acid to dissolve the phosphatic sediment which results from boiling a neutral or alkaline specimen of urine.

When the albumen is very copious, a few drops of picric acid cause cloudiness, which disappears on shaking, like the cloudiness caused by a drop or two of nitric acid. A further addition of the solution causes a permanent coagulation. A small amount of picric acid solution does not prevent the subsequent coagulation of the albumen by heat, and an albuminous precipitate by picric acid is not soluble by an excess of the acid. In these two respects, picric acid differs advantageously from nitric acid.

To detect a very minute trace of albumen, I adopt the method which I will now show you. Into a test-tube six inches long I pour a four-inch column of urine ; then, holding the tube in a slanting position, I gently pour an inch-column of the picric acid solution on the surface of the urine, where, in consequence of its low specific gravity (1007), it mixes only with the upper layer of the urine ; and, as far as the yellow colour of the picric solution extends, the coagulated albumen renders the liquid turbid, thus contrasting with the transparent unstained urine below. Bear in mind that, for the action of the test, there must be an actual *mixture*, and not a mere surface-contact, of the two liquids. When, in consequence of the scantiness of the albumen, the turbidity is very slight, the application of heat to the upper part of the turbid column is found to increase the turbidity.

Any previous turbidity in the urine interferes with the detection of a minute trace of albumen. The turbidity, which is caused by any floating particles in the urine, is increased by the yellow stain which is given by the picric acid solution. The turbidity which is due to lithates may be removed by heat, more moderate in degree than that required to coagulate albumen, and that caused by mucus by a previous filtration of the urine to be tested.

I have proved, by numerous observations, that picric acid,

applied in the manner described, is a more delicate and trustworthy test than nitric acid added to cold urine.

The simplest method of comparing the two tests, as regards their relative delicacy, is to dilute a specimen of albuminous urine until one or the other test fails to act; when it will be found that the picric acid solution shows the presence of albumen in a specimen diluted much beyond the point at which the nitric acid fails to give any indication of its presence. The picric acid often gives an immediate albuminous opalescence in urine, when nitric acid only slowly, after an interval of some minutes, gives a similar, but perhaps a doubtful, indication. I shall presently have something to say of the practical value of a test for minute traces of albumen.

Here let me remark that the albuminous opalescence with picric acid, which always occurs immediately, if at all, and which is increased by heat, may readily be distinguished from the coarse granular particles of sodium urate which, after a delay of some minutes or hours, sometimes result from the acidity of the picric solution. The granular masses of urates, often mixed with crystals of free uric acid, quickly fall to the bottom of the test-tube, and are readily distinguished, not only under the microscope, but even by the unaided eye.

It rarely happens that the picric solution, in common with all acid tests, produces an immediate turbidity in non-albuminous urine by the precipitation of urates. This turbidity, like the similar turbidity sometimes caused by nitric acid, is readily distinguished from albumen by its speedy and complete disappearance when heat is applied. In very rare instances uric acid crystals are quickly set free by the addition of picric acid, in sufficient quantity to cause a slight turbidity which is not removed by heat. The appearance of the crystals under the microscope would distinguish this from an albuminous opalescence.

It has been objected to picric acid as a test that it gives with *peptones* a precipitate not distinguishable from albumen. To this statement, I reply, first, that the precipitate with peptones is most easily distinguished from that with albumen by its ready solubility when heated. I have here a peptonised solution of mutton-fibre; the addition of picric acid solution

causes a copious yellow precipitate, which, as you see, is entirely dissolved over the spirit-lamp before the liquid reaches the boiling-point. The distinction, then, between albumen picrate and peptone picrate is sufficiently easy. In fact, *there is no known substance, occurring in either normal or abnormal urine, except albumen, and, in very rare cases, uric acid, which gives a precipitate with picric acid insoluble by the subsequent application of heat.*

Precipitated peptones resemble a sediment of urates in the fact that both are dissolved by heat, but they are readily distinguished by the microscope, which shows the urates to be composed of large granules of sodium urate and uric acid crystals. Freshly precipitated peptones appear under the microscope quite homogeneous and free from granular solid particles; but when, after being dissolved by heat, they are reprecipitated on cooling, they contain exceedingly minute granules, which are incessantly dancing about with the so-called 'Brownian movement.'

This granular condition occurs at once if the cooling be allowed to take place *slowly* in the air, but if the liquid be *suddenly* cooled, by plunging the test-tube in cold water, the precipitate does not assume the granular appearance until it has been allowed to stand for some hours.

The test for peptones which is commonly employed is Fehling's copper solution, which gives a rose-red colour at the junction of the two liquids, when the urine is gently poured on the surface of the solution previously introduced into the test-tube. The utility of the test is, however, much lessened by the fact that albuminous urine, treated in the same manner with Fehling's solution, yields a colour which cannot be distinguished from that which occurs in peptonous urine.

That picric acid is a much more delicate test for peptones than Fehling's solution is shown by the fact that the addition of artificially prepared peptones to normal urine, in amount sufficient to give a copious precipitate with picric acid soluble by heat, affords only a slight colour indication with Fehling's solution.

If peptones are associated with albumen in the same specimen, their detection and separation may be readily

effected by the picric acid and heat tests. The precipitate with picric acid, instead of being increased and rendered more dense by heat, as when albumen alone is present, will be lessened and dissolved in proportion to the amount of peptones present. If the boiling liquid be then poured on a filter, the dissolved peptone picrate will pass through and precipitate again on cooling, while the coagulated albumen remains on the filter.

By testing in this manner I have found a trace of peptones in many specimens of highly albuminous urine, but hitherto I have not met with a single instance of peptonuria apart from albuminuria.

Dr. J. Frank Nicholson, writing to the *Lancet*,[1] mentioned the fact that, in the urine of patients taking large doses of quinine, he got, with picric acid, copious opalescence, which was cleared by heat. A similar precipitate occurs when cinchonidine is taken in large doses. In fact, most of the vegetable alkaloids, such as morphine, atropine, &c., are precipitated by picric acid and by the potassio-mercuric iodide; but the cinchona alkaloids are alone likely to be taken in sufficient doses to render the urine opalescent with either of these tests, and then the complete clearance by heat at once distinguishes them from an albuminous precipitate.

It has sometimes been asserted that picric acid coagulates *mucin*, and so may cause an opalescence in normal urine. This erroneous statement is explained by the fact that those from whom it emanates have combined either acetic or citric acid with the picric; a combination which is quite unnecessary except, as before mentioned, when the urine is highly alkaline.

As neither acetic nor citric acid coagulates albumen without the application of heat, while both acids coagulate mucin, when an alkaline urine is suspected to contain a *trace* of albumen, it should be acidulated with acetic or citric acid, and filtered to separate the mucin. The filtrate may then be tested with picric acid and heat as usual. When an alkaline urine is *highly* albuminous, the slight opalescence caused by the coagulation of the mucin by acetic or citric acid may be disregarded.

[1] November 10, 1883, p. 835.

I have shown you that picric acid is a most delicate and trustworthy test for albumen in the urine; and if you ask whether there is any practical advantage to be gained by the employment of so sensitive a test, I reply that, unquestionably, there is. For instance, during the convalescence from acute nephritis with albuminuria, whether the result of scarlet fever, of exposure to cold, or of the other well-known exciting causes, it is of vital importance to be assured that the urine is entirely free from albumen before the patient is allowed to pass from medical supervision and to return to his usual habits. For the want of such exact and careful observation, it too often happens that, in these essentially curable cases, the recovery is incomplete; a trace of albumen remains, but unattended by any signs of functional disorder, until, it may be many years afterwards, the patient presents himself with some of the many distressing symptoms and unequivocal physical signs in the urine, the heart, the arteries, the eyes, and various other tissues, of advanced and incurable degeneration of the kidneys.

And here let me warn you to avoid the not uncommon mistake of *testing for albumen only one specimen of urine, and that one which has been passed before breakfast.* It frequently happens that, while the urine which is passed after resting in bed and before breakfast is quite free from albumen, that which is secreted after food and exercise contains albumen in abundance. It is, therefore, obvious that, if a morning specimen alone were tested, a patient might be considered and pronounced convalescent while disease of a dangerous tendency is still present. It is always desirable to have for testing both a night and a morning sample of urine; the former showing the effect of food, exercise, and change of temperature, the latter of abstinence, rest, and warmth.

It will very commonly be found that the amount of albumen in the urine secreted after food and exercise is twice as great as that passed after some hours of abstinence and rest in bed.

The erect posture alone has sometimes a remarkable influence. In one of my patients lately, a youth recovering from acute albuminuria, it was found at one period that, although no albumen appeared after his breakfast, so long as he

remained in bed; soon after he assumed the erect posture, a trace of albumen was found in the urine, whether before or after breakfast, although he remained in the same warm room. The only probable explanation of this is that the erect posture caused some congestion of the kidneys, and consequent escape of albumen, by the gravitation of blood into their weakened and relaxed vessels.

In many cases, however, food has a very decided influence in the production of albuminuria. Thus in hospital practice I have often found that in cases of acute albuminuria in which, under an exclusive milk diet, the albumen had disappeared, the addition of solid food, *while the patient is still kept in bed*, had caused a reappearance of albumen. In order to ascertain the cause of an occasional recurring albuminuria, to which the very inappropriate term 'Cyclical Albuminuria' has lately been applied, it is often necessary to test the urine at short intervals during the twenty-four hours. In this way only is it possible to determine, in each case of recurring albuminuria, the respective influence of food, temperature, posture, and exercise.

There is yet another class of cases in the investigation of which it is of practical importance to employ a delicate and trustworthy test for albumen. I refer to that common form of Bright's disease in which the small red granular, or so-called cirrhotic, kidney is found after death. In this class of cases, the amount of albumen is often small, and is no index or measure of the gravity of the disease; indeed, more than one recent observer has asserted that an entire absence of albumen is of common occurrence in this form of disease. This statement, however, is not in accordance with my experience. The cases of this disease that have come under my observation have been very numerous, and I am constantly meeting with fresh examples; and when the existence of the disease has been unequivocally established by other signs and symptoms, I have very rarely found that a careful testing of the urine at different periods of the day has failed to detect albumen. The idea that the urine is often free from albumen in these cases must, I think, have resulted from carelessness or want of skill in testing.

One method of testing which is in common use is very likely to mislead; I refer to the practice of adding acetic acid to the urine before boiling. The result often is that, albumen not being coagulable by an excess of the acid, an albumen acetate is formed, which, like the albumen nitrate before mentioned, is not coagulable by heat; the albumen, therefore, remains in solution, and is not detected.

I cannot too emphatically insist upon the fact that the smallest trace of albumen in the urine, whether with or without the appearance of renal tube-casts, is always abnormal and pathological. Not long since, during a discussion at the Clinical Society, it was suggested by one speaker that an amount of albumen so minute as to require for its detection any test more delicate than heat and nitric acid should be looked upon as physiological. I scarcely need say that such a distinction would be purely artificial, and not in accordance with facts.

Without doubt, the correct interpretation of the significance of albuminuria, whether in small or large amount, is a subject of great practical importance; but if the absence of symptoms and the superficial appearance of perfect health are to be taken as evidence that albuminuria is physiological, I can affirm that the largest possible amount of albumen in the urine might, not rarely, be looked upon as physiological. The notorious absence of symptoms in many cases of albuminuria accounts for, but does not excuse, the too common neglect to test the urine until renal degeneration has reached an incurable stage. Until the practice of testing the urine in all cases of disordered health, no matter how trivial, becomes general, cases of unsuspected latent albuminuria will continue to abound.

The easiest and the most exact mode of estimating the *amount* of albumen in the urine is by the use of Esbach's graduated tubes.[1] The albumen is coagulated by a solution of picric acid, either with or without citric acid: the amount of sediment, after being allowed to stand for twenty-four hours, indicates

[1] See papers by Dr. Veale (who had carefully tested the accuracy of this method), *British Medical Journal*, May 1884, p. 898; by Mr. Bloomfield, *Lancet*, January 23, 1886; by Dr. Cruise, *Dublin Journal of Medical Science*, June 1886, p. 499; and by Dr. Sidney Coupland and myself, *Lancet*, July 1886. p. 63.

the number of grammes of dried albumen per litre of urine. The graduated tubes, with printed directions for their use, and for making the test solution, are sold by Mr. E. Cetti, of 36 Brooke Street, Holborn.

This method, which is very accurate when the amount of albumen is not less than 1 gramme per litre (0·01 per cent.), is not applicable for the estimation of very small quantities of albumen. To such specimens it is convenient to apply the terms slightly opalescent, opalescent, opalescent to milky, and milky, to express varying degrees of coagulability.

The *potassio-mercuric-iodide* solution, prepared by adding a solution of potassium iodide to a solution of mercuric chloride until the red precipitate is just re-dissolved, has been much praised as a highly sensitive test for albuminuria. The test is entirely untrustworthy, for the simple reason that with the addition of acetic or citric acid, without which it does not coagulate albumen, it causes an opalescence in all normal urines, by precipitating mucus and apparently some other normal constituent of the secretion.

And now, gentlemen, having, as I trust, convinced you of the great value of picric acid as an albumen precipitant, I shall, in a future lecture, show you that, in combination with potash, it is an admirable qualitative and quantitative test for sugar.[1]

[1] I was induced to use picric acid as a test for albumen by a suggestion from my son, G. Stillingfleet Johnson, who found, as he states in a paper on the Compounds of Albumen with Acids, published in the *Journal of the Chemical Society*, August 1874, that picric acid causes coagulation of albumen in solutions of all the acid compounds of that substance. It was not until after I had (in the *Lancet*, November 4, 1882, p. 737) published my experience of picric acid as an albumen test that I became aware of the fact that the same test, although known to very few, had for some years been occasionally used by others (see my letter in the *Lancet*, November 11, p. 823). As I shall explain hereafter when we come to the consideration of the tests for sugar, my first observation of the reaction of *glucose* in a boiling solution of picric acid and caustic potash was the result of what I have ventured to call 'a happy accident.'

SECTION III.

THE GENERAL CHARACTERS OF BRIGHT'S DISEASE.

Bright's Disease—History and Definition of the Term—General Propositions relating to Bright's Disease:—1. It is of Constitutional Origin: Proof of this—2. The Primary and Chief Changes occur in the Gland Cells of the Kidney—3. Changes in Basement-membrane of Tubes and Malpighian Capsules often misinterpreted—4. Changes in Vessels of Kidney and other Organs later and less constant—5. Morbid Products appearing as Tube-casts in the Urine are of great diagnostic value—Mode of examining Urinary Sediments.

Definition of Bright's Disease.—Having made yourselves acquainted with the structure and functions of the kidney, you are prepared to enter upon the study of its diseased conditions; and I now proceed to give you some account of a most important and interesting class of cases which are usually included under the name of Bright's disease. The history of this term may be very briefly told. Before the time of Dr. Bright, it was known that dropsy and disease of the kidney were sometimes associated. It was also known that some dropsical patients had albuminous urine.[1] Dr. Bright's great merit and originality consisted in this, that he pointed out the frequent association of dropsy and albuminuria with very striking pathological changes in the kidney. In the first volume of his *Reports of Medical Cases*, published in 1827, he described and represented by beautiful coloured drawings various morbid appearances in the kidney; some kidneys being large and congested; others large and anæmic; and others, again, contracted and granular. He showed that these forms of renal disease are of every-day occurrence; that they are frequently associated not only with dropsy, but with many other formidable secondary diseases; and thus he opened up the great field of renal pathology, which had previously been, for all practical purposes, an almost unknown region. These morbid conditions of the kidney having been made known, it became necessary to give them a name, and

[1] See *Observation on the Nature and Cure of Dropsies*, by John Blackall, M.D., 3rd ed. 1818.

various names have been proposed. Rayer used the term 'néphrite albumineuse' to designate this class of diseases. The objections to this term are, first, that every form of inflammation of the kidney may be associated with albuminous urine; and second, that some forms of the disease under consideration are not of an inflammatory nature. Dr. Christison called the disease 'granular degeneration.' The kidneys, it is true, are often granular; but in some of the most characteristic cases they are quite smooth, and not at all granular. Each of these terms, then, being insufficient and objectionable, it has become the custom to designate the morbid states of the kidney by the name of the distinguished physician who discovered them; and so the term 'Bright's disease' has come into very general use both in this country and abroad. The term is sufficiently convenient and unobjectionable, if only we can agree upon a definition. The designation Bright's disease seems to involve the idea of unity; and some pathologists have maintained that all the morbid changes in the kidney to which attention was directed by Dr. Bright are the result of a single morbid process in different stages and of various grades of intensity. I shall have no difficulty in convincing you that this view is erroneous. Meanwhile, however, I must ask you to bear in mind that under the name of Bright's disease are included various forms of acute and chronic disease. I believe the following to be the best definition of the term Bright's disease—'a generic term indicating several forms of acute and chronic disease of the kidney, usually associated with albumen in the urine, and frequently with dropsy, and with various secondary diseases resulting from deterioration of the blood.'

Accepting this definition of Bright's disease, we shall find that it is nearly synonymous with albuminuria—nearly, but not quite. For, on the one hand, in some rare and quite exceptional cases, both acute and chronic, albuminuria is sometimes absent; and, on the other hand, albuminuria may be unassociated with Bright's disease. For example, the mixture of blood or pus with the urine of course renders it albuminous; but hæmaturia and purulent urine, although often associated with Bright's disease, may result from other and

quite distinct pathological conditions, either general or local. And, again, in the advanced stages of valvular disease of the heart, and in some cases of extreme emphysema of the lungs with bronchitis, albuminuria may be caused by passive congestion of the kidney resulting from an impeded circulation through the heart and lungs, and a consequent engorgement of the whole systemic venous system; yet albuminuria thus originating from purely mechanical causes would not be correctly designated a form of Bright's disease. With these limitations, however, the terms 'albuminuria' and 'Bright's disease' may be looked upon as practically synonymous; and, to avoid wearisome reiteration, I shall employ sometimes one and sometimes the other term.

Now let me impress upon you that, according to the nomenclature and definition which I have given you, Bright's disease is not always, and of necessity, an incurable malady. Under this common designation will be included on the one hand cases of renal disease as curable as a simple bronchitis or a slight pneumonia, and on the other hand cases as intractable as advanced pulmonary phthisis. The first great division of cases of Bright's disease is into acute and chronic; and, in any case that comes under your notice, there always arises this most important practical question, Is the disease acute, and therefore probably curable? or is the case one of chronic and advanced degeneration of the kidney, and therefore probably irremediable? A careful study of the entire history of the disease, and of each particular case that comes under your observation, will alone enable you to give a true and trustworthy answer to this question.

Before I proceed to discuss the various forms of Bright's disease, I wish to direct your attention to certain general propositions which are true of all forms of the disease.

Proposition I.—Bright's disease is not a merely local malady, but a disease of constitutional origin; and the proximate cause of the renal disease is, in all probability, a morbid condition of the blood.

The proofs of the blood-origin of Bright's disease are to be found in the entire pathological history of the disease. Much of this evidence will come under our consideration hereafter, but

some facts bearing upon the question may with advantage be referred to now.

First, then, the disease is a *bilateral* disease. The rule is, that both kidneys, receiving the same morbid blood, are both affected, and both by the same form of disease, although the degeneration is sometimes more advanced in one kidney than in the other. The exceptions to this rule are easily explained. For example, one kidney may be absent or undeveloped; or it may have been destroyed by an abscess or by the impaction of a calculus. Bright's disease occurring in such cases would of necessity be unilateral. But the most instructive case of unilateral Bright's disease that I am acquainted with has been published by the late Dr. Moxon.[1] In a woman aged 34, who died of dropsy, the right kidney had the characters of a large, pale, granular Bright's kidney. 'The left, on the contrary, was rather small, and of the colour and appearance of a healthy kidney.' A microscopic examination showed the large kidney to be much diseased; the smaller 'practically healthy.' The explanation of this remarkable difference was found in the fact that the left renal artery was plugged by a very old fibrinous coagulum, probably derived from the interior of the heart. Dr. Moxon, in his interesting comments on this case, suggests that, while one kidney was saved by a diminution or rather a suspension of its function, the other was destroyed by an excess of function. We know, as he says, that an excess of normal function (as, for instance, when double work is thrown upon one kidney in consequence of the other having been destroyed by an impacted calculus) causes not Bright's disease, but simple hypertrophy of the kidney. In this case, the result was to aggravate a disease which probably had already commenced. But, with reference to the theory of blood-poisoning, which I am now endeavouring to illustrate, I would suggest that the fibrinous plug saved the left kidney by excluding morbid blood from its gland-cells, while it damaged the right by diverting to it a double supply of the same morbid blood. The bare nutrition of the left kidney was probably maintained by anastomoses between the renal artery and other branches from the aorta (the kidney may be partially injected from the aorta

[1] *Pathological Transactions*, vol. xix. p. 268.

after ligature of the renal artery); but the secretory function, in the discharge of which, as we shall hereafter see, the pathological changes occur, was suspended, and therefore the organ underwent no other change than slight wasting, the result of a defective supply of nutrient blood. This exceptional case, therefore, confirms and helps to explain the rule that Bright's disease is bilateral.

Secondly, confirmatory evidence of the blood-origin of Bright's disease is derived from the fact that the malady occurs in association with constitutional states in which a morbid condition of blood may confidently be assumed to exist. Albuminuria, varying in degree and in duration, has been found more or less frequently associated with scarlet fever, diphtheria, measles, small-pox, erysipelas, pyæmia, typhus and typhoid fever, yellow fever, rheumatic fever, malarious fevers, cholera, purpura, scurvy, diabetes, syphilis, certain forms of pneumonia, pregnancy, the absorption of secretions from the interior of the uterus after parturition, gout, the abuse of alcoholic liquors, excessive eating, certain forms of dyspepsia, resulting, as may be supposed, in the passage of crude materials into the circulation, a poor and insufficient diet, purulent and other exhausting discharges, and, lastly, suppressed action of the skin by exposure to cold, and especially to cold and wet combined.

Proposition II.—The morbid blood, which is assumed to be the proximate cause of Bright's disease in all its forms, exerts its influence primarily and chiefly upon the gland-cells which line the convoluted tubes. Look at a diseased kidney, or at one of Dr. Bright's beautiful plates, and you see at a glance that the cortex or secreting portion of the kidney is the main seat of the disease; while the medullary cones, even in the advanced stages of the malady, are left comparatively intact. Examine the diseased gland with the microscope, and you find that the morbid process has been concentrated, and, as it were, brought to a focus upon the secreting cells within the uriniferous tubes. The kidney, as a great blood purifier, forms an outlet and a means of escape for many useless and noxious materials which have been developed within the system or introduced from without; and in the discharge of this excretory

function, the gland undergoes the degenerative changes which constitute Bright's disease. The blood in the vessels of the kidney probably contains no more noxious materials than an equal volume of blood in any other tissue—that of the voluntary muscles, for instance; but, during the process of excretion, these products are withdrawn from the blood and concentrated within the gland-cells of the kidney, where they effect the morbid changes in question. The intertubular pathological products which sometimes occur, either alone or associated with other structural changes, are comparatively infrequent.

Proposition III.—The structural changes which occur in the basement-membrane of the tubes and in the Malpighian capsules are direct results of the intratubular cell-changes.

These changes in the basement-membrane are often very obvious and striking, but they have frequently been misinterpreted. For example, thickening and corrugation of the membranous walls of the tubes in the advanced stages of a contracted granular kidney are often described as being the result of a primary formation of connective tissue between the tubes—an interstitial fibrosis, as it is called—but I shall demonstrate to you hereafter that the greater part of this supposed new formation of fibrous tissue consists of the atrophied remains of intertubular capillaries and of the basement-membrane of uriniferous tubes, whose epithelial lining has been destroyed by a primary pathological process.

Proposition IV.—During the progress of chronic Bright's disease, the blood-vessels in the kidney and in many other tissues and organs undergo very interesting changes, but these occur later and less constantly than those which affect the secreting tissues of the gland.

Proposition V.—The pathological products of the structural changes within the tubes, being carried out by the liquid secretion, escape with the urine and appear in the form of cylindrical casts of the uriniferous tubes; and a microscopical examination of these tube-casts affords most interesting and valuable information as to the nature and the stage of the renal disease. I shall hereafter show you the various forms of tube-casts, and explain to you their pathological and diagnostic significance.

Let me impress upon you that the examination of urinary sediments of all kinds is much facilitated by allowing the urine to stand for a few hours in a four-ounce conical glass. Then a portion of the sediment can be taken up with a pipette and put into a glass cell, and this, covered with thin glass, is now placed beneath a quarter-inch object-glass. A magnifying power of about two hundred diameters is quite sufficient for the examination of urinary sediments; and with a higher power it would be impossible to focus to the bottom of a cell. The most convenient cell is made by cementing with marine glue a circular flat ring of glass, or a thin square of glass with a circular perforation, upon the ordinary microscopic slip of glass. These cells are sold by all microscope-makers. One cell, with ordinary care, will last for months. When the sediment is scanty it is a waste of time to hunt for tube-casts in a drop of urine placed between two flat pieces of glass. The glass cell before mentioned holds several drops of sediment, and therefore greatly facilitates the investigation.

When making a microscopical examination of the urine, care must be taken not to mistake fibres of cotton, or of wool, or human hairs, which are often accidentally present, for tube-casts. A practitioner once brought to me a specimen of supposed tube-casts of various colours—green, blue, and red—and I regret to say that he was not grateful when I told him that his coloured tube-casts, which he had looked upon as an interesting discovery, were fibres of wool, probably from the sweepings of a carpet. Many years since I was called into the country to see a case of supposed cancer of the kidney; when I was told that the chief evidence of cancer was afforded by the appearance of cancer cells in the urine. The supposed cancer cells, I saw at once, were grains of starch, whose presence in the urine was accounted for by the nurse having applied violet powder to the patient's vulva. The lady recovered, and no one but the two gentlemen whom I met in consultation heard of the microscopic mistake.

SECTION IV.

ON ACUTE BRIGHT'S DISEASE.

Synonyms—General Symptoms—Microscopic Appearances in the Urine—Morbid Anatomy of the Kidney—Physiology of the Morbid Process—Varieties of Acute Bright's Disease: 1. With Epithelial Desquamation (Desquamative Nephritis)—2. Without Desquamation, and with or without small Hyaline Casts—3. With Leucocyte Casts (Glomerulo-Nephritis), with or without Epithelial Desquamation—4. Without Albuminuria—Changes in the Blood—Etiology—Suppressed Action of the Skin artificially produced in Rabbits—Diagnosis—Prognosis.

I have told you that the first great division of cases of Bright's disease is into acute and chronic, and I now proceed to give you the pathological history of acute Bright's disease. The synonyms for acute Bright's disease are 'acute albuminuria,' 'acute desquamative nephritis,' 'acute parenchymatous nephritis,' and 'acute renal dropsy.' The renal disease associated with dropsy, which often occurs in connection with scarlet fever, may be taken as a type of acute Bright's disease. I will first give you a sketch of the ordinary course of this form of disease, and I will then point out to you the chief varieties and modifications of the malady.

The attack is sometimes ushered in by a sense of chilliness, which may amount to actual rigors; a quick and throbbing pulse, a hot and dry skin, a dry and coated tongue, thirst, loss of appetite, pain in the back and limbs, headache, and restlessness. In some cases, frequent vomiting occurs at the commencement of the attack. In most instances, dropsy is a very early symptom; the patient's attention, or that of his friends, being arrested by an appearance of unusual pallor and puffiness of the face, and especially of the eyelids; the swelling soon becomes general, affecting the subcutaneous areolar tissue throughout the body, and often one or more of the serous cavities. The urine is at this stage more or less scanty, occasionally almost or even altogether suppressed; usually it is dark-coloured from admixture with blood, the colour varying from a slight smokiness to a deep blood tinge, and it contains so large an amount of albumen as to become nearly

solid with albumen tests. The specific gravity varies considerably, being oftener above than below the normal point. There is usually more or less pain and tenderness in the loins; the pain is sometimes, though rarely, severe, and occasionally it extends to the inside of the thighs and to the testicles. There is frequent desire to pass water, and sometimes a sense of pain or scalding in the urethra. There is often more or less of uneasiness in the epigastrium, with flatulent distension of the stomach, especially after food, and nausea and vomiting are of common occurrence. In some cases, inflammation of one or more serous membranes—the pleura, the pericardium, or the peritoneum—occurs; or respiration is impeded by an œdematous or inflammatory effusion into the pulmonary air-cells and the smaller bronchi; or the headache, which is often present from the commencement, becomes more severe, and is followed by one or more attacks of convulsions, from which the patient may recover, or which may be followed by fatal coma.

When the progress of the disease is favourable, one of the earliest signs of amendment is an increased secretion of urine. It may be that, for some days, only a few ounces of urine have been passed in the twenty-four hours; it then becomes more copious, of paler colour, of lower specific gravity and less albuminous, and the amount secreted may be as much as from four to six pints, the dropsy meanwhile diminishing daily. After an interval, varying from a few days to a month or more, the secretion of urine is reduced to the normal amount, the sediment diminishes, and at length disappears, while the urine gradually resumes its normal colour and ceases to be albuminous. At any time during the convalescence, there may be a temporary increase of blood and albumen, with a diminished secretion of urine and a return of dropsy, if the congestion of the kidneys be increased by exposure to cold or by errors of diet. In most cases the dropsy disappears for days, and it may be for weeks or even months, before the urine has ceased to be albuminous. In some cases, although the dropsy and the pallor of the skin pass away, the urine remains albuminous, and the renal disease passes into a chronic form.

Acute albuminuria is sometimes, though rarely, unasso-

ciated with dropsy from its commencement to its termination. The terms 'acute albuminuria' and 'acute renal dropsy' are, therefore, not strictly synonymous.

Microscopic Appearances in the Urine.—A portion of the urinary sediment in the early stage of acute Bright's disease,

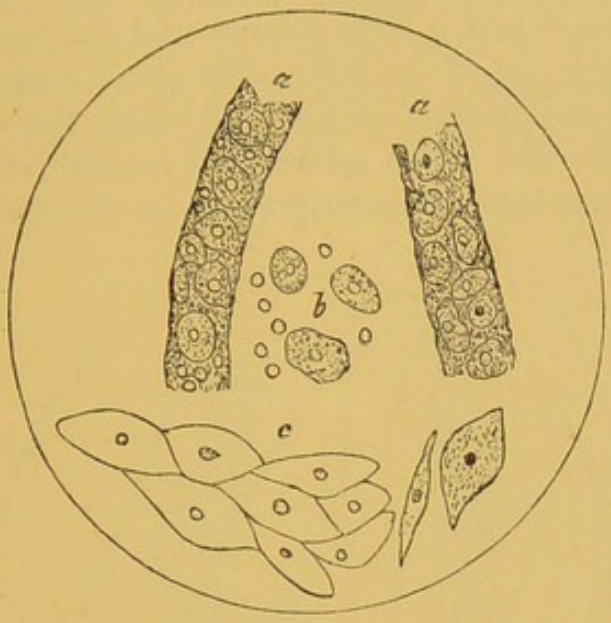

FIG. 11.—*a a*. epithelial casts. Casts of the uriniferous tubes entangling renal epithelium and blood-corpuscles. *b*. scattered renal gland-cells and blood-corpuscles. *c*. pavement-epithelium from the vagina. This is broader, flatter, and less granular than the renal epithelium.—× 200.

when examined with a power of about two hundred diameters, presents very characteristic appearances. The most striking objects are solid cylindrical moulds of the uriniferous tubes. The basis of all renal tube-casts is fibrine which has coagulated within the uriniferous tubes; but these casts assume various appearances according to the nature of the products which

FIG. 12.—BLOOD-CASTS COMPOSED OF FIBRINE ENTANGLING BLOOD-DISCS.—× 200.

they contain and the condition of the tubes in which they have been moulded. The casts which are most characteristic of acute Bright's disease are 'epithelial casts' (fig. 11). These casts contain gland-cells evidently derived from the convoluted tubes of the cortex; they also entangle blood-cor-

puscles, and some casts are entirely composed of coagulated blood; these are called 'blood-casts' (fig. 12). Together with the tube-casts, many scattered renal gland-cells and blood-discs may usually be seen. In addition to the epithelial and blood-casts, we find in most cases of acute Bright's disease some small and large hyaline casts (fig. 13). The difference between a small and a large hyaline cast is readily explained by referring to fig. 6, p. 629. The small casts, which are composed of pure fibrine, are moulded within the canal formed by the gland-cells, which retain their normal position within the uriniferous tubes; the large casts, on the other hand, are

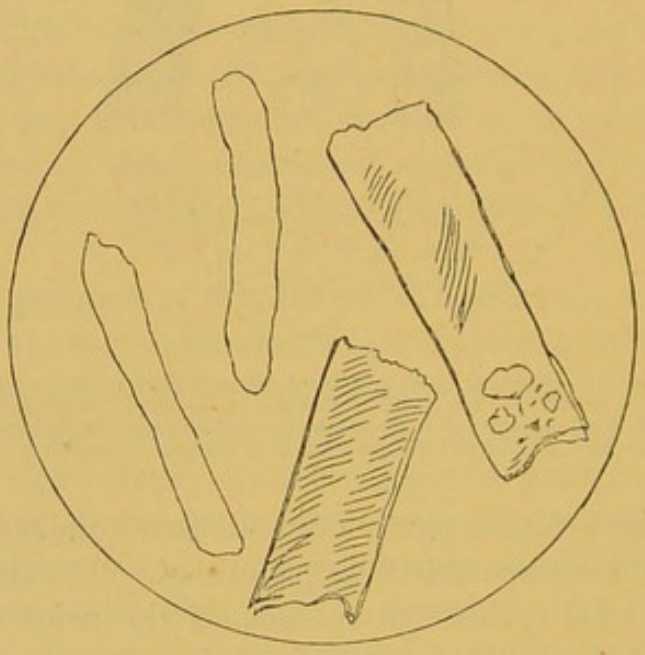

FIG. 13.—SMALL AND LARGE HYALINE CASTS COMPOSED OF PURE FIBRINE.—× 200.

formed within tubes whose gland-cells have been removed. The diameter of these casts, therefore, is about twice that of the small casts, and equals that of the tubes whose basement-membrane constitutes the mould in which they have been formed. The larger casts may be simply hyaline, or they may entangle here and there a cell-nucleus or the fragment of a cell.

In cases of acute Bright's disease, the small hyaline casts are often present in considerable numbers, while the large hyaline casts are usually less numerous, and may be entirely absent. On the other hand, there are unquestionably acute

and curable cases in which the large hyaline casts are very numerous. When the disease has lasted beyond a month or six weeks, we find often in adults, more rarely in children, that more or less oil begins to appear in the tube-casts and in the desquamated renal epithelium. The appearance of oily casts and cells (fig. 14) excites less alarm now than it formally did. It indicates that in certain parts of the kidney the secreting cells and the inflammatory exudations are undergoing fatty transformation; but I have seen many cases of

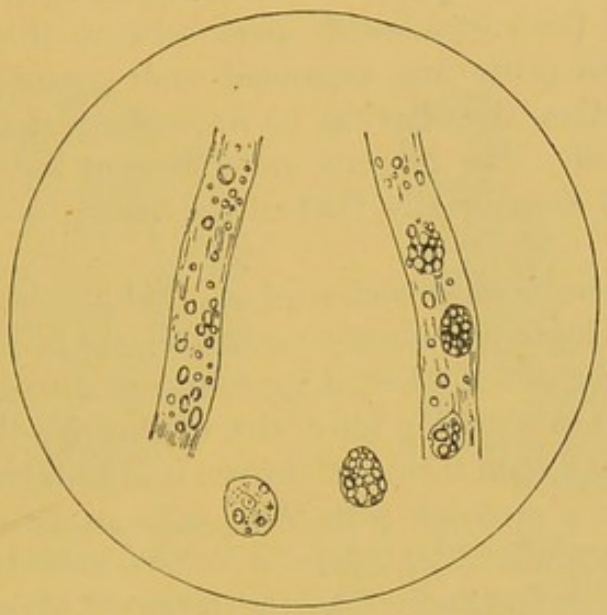

FIG. 14.—OILY CASTS AND CELLS.—× 200.

complete recovery after oily casts and cells, in great numbers, had appeared in the urine continuously for many weeks.

Morbid Anatomy of the Kidney.—When acute Bright's disease (acute desquamative nephritis) has proved fatal, both kidneys are always found diseased; they are enlarged and their weight is increased, each kidney weighing from six to eight ounces, or even more. The increased weight of the kidney is partly due to infiltration with serous fluid; the tissues contain an excess of water in proportion to solids; the capsule readily peels off the surface, which is smooth and mottled, presenting an irregular combination of red vascular engorgement, with anæmia and pallor. The fine lobular divisions formed by the minute venous radicles on the surface are more or less obliterated. Some of the stellate veins are

much enlarged and distended. Here and there appear spots of hæmorrhage—some the size and shape of a pin's head, others irregular in form. On section, there appears a marked distinction between the cortical and the medullary portions; the former presents the same mixture of congestion and anæmia as appears on the capsular surface; the spots of ecchymosis, too, are here visible, but they sometimes take a linear course, especially near the base of a medullary cone. The cones are much darker than the cortex, as if from venous congestion. They appear to be compressed by the swollen portions of the cortex which pass between them, while the bases of the cones are expanded and spread out into the cortical portion, thus having, as Rayer suggested, the form of a wheatsheaf. The mucous membrane of the pelvis of the kidney, and occasionally that of the ureter, is more or less congested.

A microscopic examination of the kidneys shows that the morbid products are mostly contained within the convoluted tubes of the cortex. Most of the tubes are abnormally opaque, in consequence of being filled with epithelial cells which have been formed within them and thrown into their cavity (fig. 15). The tubes are crowded with these cells in different degrees, some being stuffed full, while in others there is little or no evidence of desquamation having occurred, there being only a single layer of epithelium on their walls, and this being quite normal, or appearing more than usually opaque, granular, and swollen. This condition has been called the 'cloudy swelling' of the epithelium. The most crowded tubes are usually found in those portions of the cortex which to the naked eye appear pale and anæmic. In these parts the intertubular capillaries are compressed and emptied by the distended and swollen tubes. In some exceptional cases leucocytes are found collected between the tubes. Occasionally, when examining a section of the tubes, portions of their contents, being squeezed out, present exactly the appearance of the 'epithelial casts' which have been before described as existing in the urine.

The hæmorrhagic spots before spoken of, as appearing here and there on the cortical surface and on the face of a

section, are seen to be tubes injected with blood which has flowed into them from ruptured Malpighian capillaries (fig. 16). In some tubes the epithelium is found to contain more or less oil. Most of the straight tubes of the cones appear to be quite normal, while others are opaque and filled with cells more or less disintegrated, which seem to have been washed into them from the convoluted tubes. There is no evidence that the epithelium of the straight tubes has been morbidly changed.

Some Malpighian bodies are of a deep red colour, with fully injected capillaries; more frequently, however, the Mal-

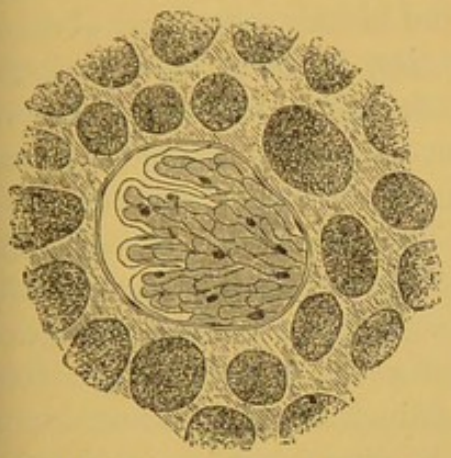

FIG. 15.—SECTIONS OF TUBES RENDERED OPAQUE BY THEIR ACCUMULATED CONTENTS.

A Malpighian body near the centre more transparent than the surrounding tubes.—× 200.

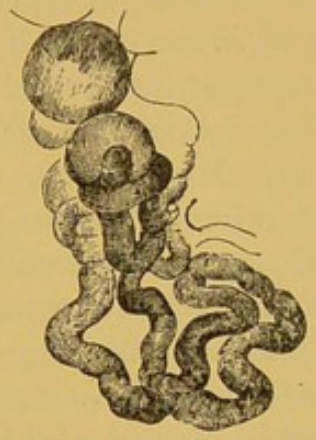

FIG. 16.—MALPIGHIAN CAPSULE AND CONVOLUTED TUBE—THE FORMER PARTIALLY, THE LATTER COMPLETELY FILLED WITH BLOOD FROM RUPTURED MALPIGHIAN CAPILLARIES—THUS FORMING A HÆMORRHAGIC SPOT IN THE CORTEX OF THE KIDNEY.

The lighter part of the capsule is occupied by the collapsed Malpighian capillaries.—× 45.

pighian capillaries appear of a lighter colour than the surrounding opaque tubes; their walls are more opaque than in the normal state, and their surface often appears rough and finely granular, as if from the coagulation upon them of some of the fibrinous materials which have transuded through them during life. The nuclei in the walls of the capillaries are abnormally conspicuous. Occasionally, as I have before mentioned, there is evidence of rupture of the capillaries, in the fact that the capsule and the adjoining tube are filled with extravasated blood (fig. 16).

Physiology of the Morbid Process.—The morbid anatomy of this form of Bright's disease being such as I have described

it to be, it remains that we attempt a physiological explanation of the phenomena. I say a physiological explanation, because I wish to impress upon you that these morbid phenomena are modifications of normal physiological processes, and admit of explanation only by reference to physiological principles. The structural changes in a kidney affected with Bright's disease are the result of a modified process of secretion. The cortical or secreting portion of the kidney is obviously the part which is chiefly implicated, and a microscopical examination shows that the gland-cells which line the convoluted tubes are the structures primarily and essentially affected. The secreting cells of the kidney, like those of other glands, have the power of separating from the blood and discharging from the body not only the constituents of their own proper secretion, but also other materials foreign to that secretion. Many salts, and many odorous and colouring matters, when introduced into the circulation through the stomach, are speedily and completely eliminated through the kidneys, and apparently without causing structural change or inconvenience. We often give for weeks or even months consecutively large doses of such medicines as iodide of potassium, a salt which is known to be largely eliminated by the kidneys. It is, however, important to observe, that certain materials, which when secreted by the kidneys in moderate quantities for a short time are quite harmless, may cause decided structural change by their long-continued secretion in larger quantities. We have an instance of this in the case of diabetes. Diabetes is not primarily a disease of the kidneys, but kidney disease is a frequent result of diabetes. The urine of most patients who are passing large quantities of sugar contains more or less decided traces of albumen, and healthy kidneys are rarely, if ever, found in subjects who have died of diabetes. The probable reason is, that the long-continued secretion of large quantities of sugar so alters the creting cells of the kidney, rendering them granular, swollen, opaque, and oily, that at length they lose the power of secreting urine; the urine becomes albuminous, and complete suppression of the secretion is often the immediate cause of death. Again, when in consequence of obstruction of the

gall-duct, or other disease or accident, causing an accumulation of bile in the blood, bile-products in large quantities are secreted by the kidneys, desquamated renal epithelium, tube-casts, and sometimes albumen, are found in the urine. The excretion of these new products sometimes excites a mild form of desquamative nephritis.[1] I refer to these facts to illustrate a physiological principle. It is certain that neither renal gland-cells nor tube-casts are ever found in normal urine, and, with the exception of such local and mechanical causes of disturbed nutrition as embolism and the distension of the renal tubules by retained secretion, the desquamative process is always an indication and a result of the excretion of some abnormal materials by the gland. It will scarcely be denied that scarlet fever is associated with a blood-poison. This poison does not always and of necessity implicate the kidneys, as in the vast majority of cases it affects and inflames the skin; but we know, from abundant experience, that the risk of renal complication is greatly increased by exposure of a patient to cold while the rash of scarlet fever is out, or even while the skin is desquamating after the disappearance of the rash. It would seem that by exposure to cold the cutaneous inflammation and desquamation are suppressed or checked, and an analogous morbid process is set up in the uriniferous tubes of the kidney. That a poison is thrown off from the skin of a scarlet fever patient, and that the poison is contained in the epidermic scales, we have very good reason to believe; and analogy renders it in the highest degree probable that the implication of the kidneys is associated with the secretion of a morbid poison by their gland-cells. The morbid phenomena result from a modified physiological function. This explanation can scarcely be considered hypothetical; it appears to be the obvious interpretation of unquestionable facts. Meanwhile, the modified and excessive cell-growth within the kidney chokes and distends the tubes with desquamated epithelium, the circulation through the gland is impeded, the secretion of urine is checked, and urinary constituents, both liquid and solid, accumulate in the blood. The circulation of urinous

[1] This effect of the excretion of biliary products upon the renal gland-cells I first published in 1852 (*Diseases of the Kidney*, pp. 108-109).

blood causes general febrile excitement, with a quick, full, and throbbing pulse; usually a more or less extensive dropsical effusion, and in some cases inflammation of the serous membranes or of other tissues, or serious disorder of the cerebro-spinal functions.

When, under favourable circumstances, the morbid poison which excited the renal disease has been eliminated, or in part, perhaps, decomposed, the desquamation of epithelium ceases, the secretory process again becomes normal, the urine is copiously secreted, the blood and the tissues are then freed from retained impurities, and from excess of water.

The copious flow of urine which occurs during convalescence from acute Bright's disease is thus explained: during the acute stage of the disease, the constituents of the urine, both solid and liquid, have accumulated in the blood, and have thence been effused into the areolar tissue and into the serous cavities. Now, urea is a most powerful diuretic: when injected into the veins of a dog, it quickly excites an abundant flow of urine; and as soon as the circulation through the kidney again becomes free, the retained urea exerts its natural diuretic influence upon the gland. The accumulated water serves as a vehicle for washing out the urea, and the copious flow of urine thus induced speedily removes the retained urinary solids and water from the blood, the areolar tissue and the serous cavities into which they had been effused, and thus the dropsy is removed.[1]

This abundant flow of urine, in favourable circumstances, takes place without aid from diuretics or drugs of any kind. I have seen it occur while only a bread-pill or coloured water was given as a *placebo*.

Varieties of Acute Bright's Disease.—I have described to you the usual course of acute Bright's disease associated with a copious desquamation of renal epithelium. For this form of disease I originally proposed the name of *acute desquamative nephritis*.[2] This acute desquamative nephritis is the most common and typical form of acute Bright's disease. But the terms acute Bright's disease and acute desquamative nephritis are not strictly synonymous. There are cases of acute Bright's disease

[1] See *ante*, Chapter III. [2] *Med.-Chir. Trans.*, vol. xxx. p. 170.

with dropsy which, in all their general features, resemble the cases which I have described as acute desquamative nephritis; but they differ in this respect, that, from first to last, whether they terminate in recovery or in death, there is no evidence of that process of renal desquamation which forms the characteristic anatomical feature of the cases to which I have before referred. The urine is as scanty and as highly albuminous as in the other class of cases; but it either contains no tube-casts, or it contains, in variable numbers, the small hyaline casts (see fig. 13), moulded within the clear canal of tubes which retain their lining of gland-cells. When the disease terminates fatally, the kidney presents to the naked eye the same appearances which characterise the acute desquamative cases; but, on microscopic examination, the sections of the convoluted

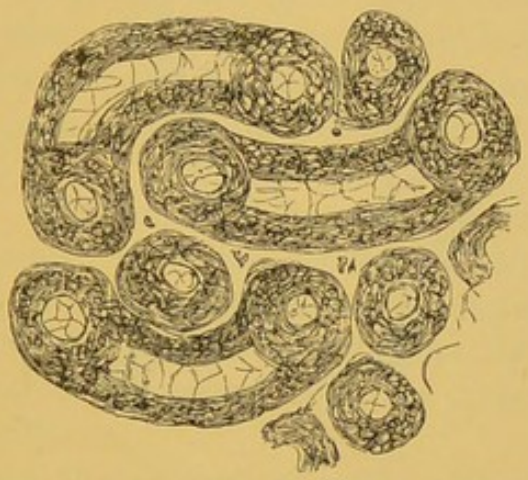

FIG. 17.—SECTIONS OF TUBES HAVING DARK GRANULAR EPITHELIUM WITH THE CENTRAL CANAL CLEAR.—× 200.

tubes appear very different. The gland-cells are unusually bulky, granular, and opaque; but the central canal of the tube, instead of being filled with desquamated epithelium, is clear and open; so that, while the 'cloudy swelling' of the epithelium renders the margins of the tubes darkly granular and opaque, the epithelial nuclei being indistinctly seen or even quite concealed, the central canal of the tube appears comparatively light and clear (fig. 17).

Glomerulo-Nephritis.—In other cases of acute Bright's disease, the urine contains few or no epithelial casts; but it deposits a sediment in which are found numerous casts, mostly of the small size, which indicates that they have been formed

within the central canal of tubes which are lined by gland-cells; and these small casts contain numerous round cells, which are identical in appearance with white blood-cells (see fig. 18).

In my *Lectures on Bright's Disease*, published in 1873, I gave a woodcut illustration of these tube-casts, and in explanation of them I stated (pp. 34–35) that I had not been able, by a microscopic examination of the kidney, to ascertain the source of the small round cells thus entangled in the tube-casts; but I added that 'since the publication of Cohnheim's researches it has occurred to me that these exudation cells may probably be white blood-cells—leucocytes—which have

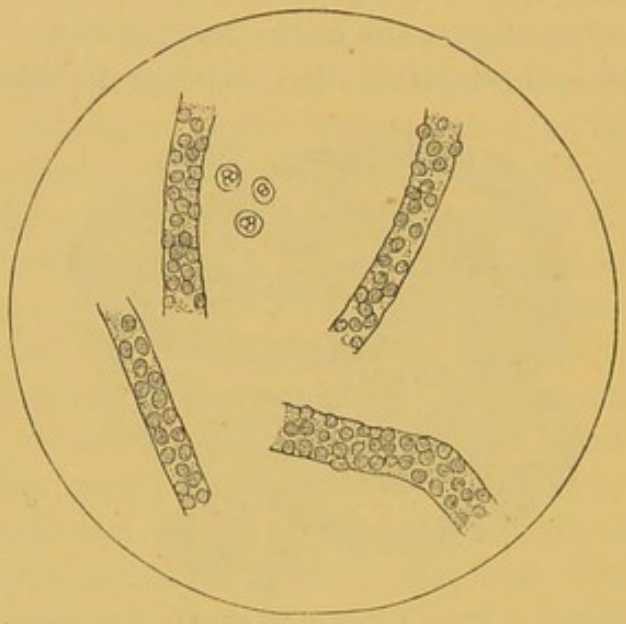

FIG. 18.—CASTS ENTANGLING SMALL ROUND WHITE CELLS—LEUCOCYTES—FROM A CASE OF ACUTE BRIGHT'S DISEASE.—× 200.

migrated through the walls of the Malpighian capillaries, and subsequently have become moulded into small cylindrical casts within the central canal of the uriniferous tubes.'

Since then additional light has been thrown upon the source and the significance of what I now designate 'leucocyte-casts.'

Dr. E. Klebs, in his *Handbuch der Pathologischen Anatomie* (pp. 644–647), describes peculiar structural changes affecting the kidney in some cases of scarlatinal nephritis, to which he has given the name of 'glomerulo-nephritis.'

Klebs states that in the cases in question the intratubular

changes usually found in cases of scarlatinal nephritis are absent, but he describes the interior of the Malpighian capsules as being filled with a number of small angular nuclei embedded in a finely granular mass. These nuclei he believes to result from proliferation of the corpuscles of the connective tissue, which binds together the capillaries of the Malpighian tufts. In kidneys affected by glomerulo-nephritis, he describes an enormous increase in the number of these corpuscular bodies, which so completely fill the Malpighian capsules as to compress and even empty the capillaries, thus seriously impeding the circulation and secretion, and causing dropsy and uræmia. According to Klebs, this intracapsular accumulation is the only structural change observable in these cases.

The observations of Klebs have been confirmed in their main points by Dr. Klein,[1] and by Dr. Bryan Charles Waller;[2] but both these observers describe and demonstrate, in addition to the intracapsular accumulations mentioned by Klebs, certain interstitial changes outside the capsules and in the intertubular stroma.

Dr. Waller considers that the cell-accumulations within Bowman's capsules have two sources: firstly, from direct proliferation of the connective-tissue corpuscles between the Malpighian capillaries; and secondly, from the migration of leucocytes, which have passed from the blood through the walls of the capillaries.

The migratory theory receives confirmation from the fact that corpuscles exactly similar to those within the capsules are found outside the capsules, and around some of the arteries and small veins of the cortex. Further, the lumen of many cortical capillaries is seen to be occupied by numerous leucocytes, which are often so closely aggregated that the circulation through them must have been seriously retarded, if not completely arrested. This cramming of the vessels with leucocytes, apparently ready to migrate, affords, as Dr. Waller maintains, additional evidence of the migratory source of those cells which have accumulated outside the cortical vessels as well as within the Malpighian capsules.

[1] *Pathological Transactions*, vol. xviii., 1877.

[2] *Journal of Anatomy and Physiology*, vol. xiv. p. 432.

Dr. Waller states that the intratubular changes are limited to those portions of tubules which are in the immediate neighbourhood of the Malpighian bodies, and consist chiefly of fatty degeneration of the tubular epithelium, though cloudy swelling and granular degeneration were also observed in a few instances.

I now pass on from the consideration of the morbid anatomy of the kidney to the clinical diagnosis of this class of cases.

During the last thirty years, at least, I have often observed and demonstrated microscopical appearances in the urine, which it now appears are closely connected with the structural changes in the kidney, designated glomerulo-nephritis by Klebs.[1] In these cases of acute albuminuria the urine deposits a sediment containing numerous tube-casts crowded with leucocytes. The casts are for the most part of small size, indicating, as I have before said, that they have been moulded within the lumen of uriniferous tubes which possess their normal lining of epithelium (fig. 18, p. 666).

These clinical observations of mine, which were made and published long before I had any knowledge of Klebs's anatomical researches, have evidently an intimate supplementary relation to them. The white-cell casts in the urine observed during life are the counterparts and the diagnostic clinical indication of the intracapsular accumulations which are found in the kidneys after death. I have observed these casts in numerous cases, both in hospital and in private practice. In some cases the leucocyte casts have existed alone, while in others they have been associated, in variable proportions, with the epithelial casts indicative of the intratubular changes which constitute acute desquamative nephritis. It is evident, therefore, from clinical observation, that the two conditions designated respectively 'glomerulo-nephritis' and 'desquamative

[1] I have in my collection specimens of these leucocyte casts which were obtained in 1856, and, the disease having been fatal, a section of the kidney, although roughly prepared in comparison with modern methods, shows the remarkable opacity of the Malpighian capsules resulting from the accumulation of leucocytes therein. This condition was quite unintelligible when the specimen was prepared.

nephritis ' may either occur separately or they may co-exist in the same subject.

In some instances the glomerular disease has been fatal with acute dropsy and uræmia, while in others it has passed into a state of chronic albuminuria; but in most of the cases that have come under my observation complete recovery has taken place, and the urine has lost all trace of morbid products. The prognosis in cases of glomerulo-nephritis as indicated by the microscopic character of the urine is, I believe, not more unfavourable than in ordinary cases of desquamative nephritis.

Then, as regards etiology, there appears to be no material difference between the intracapsular and the intratubular form of disease. While some of the cases that I have seen have been associated with scarlet fever, others have resulted from exposure to cold and wet, and the other well-known exciting causes of acute desquamative nephritis.

With our present more exact knowledge of the significance of the leucocyte casts in the urine, and of their relationship to the intracapsular accumulations in the kidney, we shall, in future, find it easier to collect precise data relating to the etiology as well as the prognosis of this form of renal disease.

You see, then, that while acute Bright's disease is usually associated with a more or less copious epithelial desquamation, there may be no desquamation of renal epithelium, and either no tube-casts or only small hyaline casts in the urine; while in other cases, either with or without epithelial casts, there may be casts crowded with leucocytes.

Acute General Dropsy without Albuminuria.—In all the cases of acute Bright's disease to which I have hitherto referred, although the microscopical appearances in the urine are various, the general symptoms and the physical and chemical characters of the secretion are alike, and in particular the presence of a large amount of albumen is a constant phenomenon. Now, I have to tell you that we sometimes, though rarely, meet with cases of acute general dropsy in which the urine, although scanty, contains not a trace of albumen. In the great majority of cases, acute Bright's disease and acute albuminuria are synonymous terms; but in these exceptional cases the latter term is inapplicable, for the

urine is not albuminous. Dr. Blackall described two cases of acute general dropsy after scarlet fever in which the urine was not coagulable either by heat or by nitric, or, as he calls it, 'nitrous' acid.[1] Dr. Roberts gives the history of one fatal case after scarlet fever.[2] Dr. Basham recorded the case of an adult in whom general dropsy followed exposure to wet and cold. He recovered.[3] Dr. Dickinson has published one fatal case in a child eighteen months old.[4] In that case the cortex of the kidney was anæmic and firm, and 'the cortical tubes were closely packed with epithelial cells and granular matter.' In a fatal case recorded by Dr. Cayley,[5] the two kidneys of a child 9 years old weighed $9\frac{3}{4}$ ounces, and had to the naked eye the appearance of the 'large white kidney.' The morbid change consisted 'in the deposition of masses of nuclei, having the characters of lymph corpuscles, between the uriniferous tubes and round the Malpighian bodies.' The epithelium of the uriniferous tubes appeared normal, or at the most was only slightly swollen and granular. Scarlatina was probably the primary cause of the disease. It is difficult to understand the absence of albumen with such an amount of intertubular deposit as must have caused great retrograde engorgement of the Malpighian capillaries.

I have notes of four cases that have come under my own observation. Three of these cases recovered, and the fourth was improving when he was lost sight of. In two of my cases, the dropsy followed scarlet fever, and in the other two it was probably a result of exposure to cold. In two of the cases, neither albumen nor tube-casts could be discovered throughout; in one, a trace of albumen was found on one occasion; and in the fourth, after general dropsy had existed for six weeks without albumen or tube-casts, a trace of albumen and some hyaline casts appeared.

Now what is the explanation of these remarkable and exceptional cases? I have neither seen nor heard of any satisfactory explanation of them, and I am not prepared to give you one.

[1] *Observations on the Nature and Cure of Dropsies*, pp. 12 to 21.
[2] *On Urinary and Renal Diseases*, 4th ed. p. 28. [3] *Lancet*, August 1867.
[4] *On the Pathology and Treatment of Albuminuria*, 2nd ed. p. 99.
[5] *Pathological Transactions*, vol. xxi. 259.

Dr. Wilks has published in the sixth volume of the *Pathological Transactions* a remarkable case of general dropsy, with a peculiar form of renal disease, but without albuminuria, in a woman aged 35. The urine passed amounted to about twelve ounces in the day, of specific gravity 1,012, and not albuminous. A few days before death, the urine became less in quantity, and for the last four days none was obtained. The kidneys were pale and large, their combined weight being seventeen ounces. The cortical portion was seen by the naked eye to be scattered over with small round dots like grains of sand. On a microscopic examination, these were found to be the Malpighian bodies, the capillaries of which were covered over with mulberry-like masses of oil-globules, while the tubes were healthy. It seems not unlikely that this may have been a case of Klebs's glomerulo-nephritis which had passed into a chronic stage.

It may hereafter happen to some of you to have the opportunity of throwing additional light upon the pathology of these rare and exceptional cases of general dropsy, not dependent on heart disease and unassociated with albuminuria.

We have seen that the chief varieties and modifications of acute Bright's disease with albuminuria are the following :— (1) with epithelial desquamation (desquamative nephritis) ; (2) without desquamation, either with or without small hyaline casts ; (3) with leucocyte casts, either with or without epithelial casts and desquamation. Lastly, we have, as an entirely distinct class of cases, rare, exceptional, and obscure in their pathology, acute Bright's disease, or at any rate acute general and febrile dropsy, without albuminuria.

Changes in the Blood.—The effect of acute Bright's disease is not only to cause an admixture of blood constituents with the urine, but also to bring about a large accumulation of urinary materials in the blood. While the urine is usually more or less bloody, the blood becomes in a greater or less degree urinous. Dr. Christison was the first to announce the fact that the blood in these cases contains a large amount of urea, and that urea is found in the dropsical and inflammatory effusions.[1] Not only is the blood altered by an accumulation

[1] *Edinburgh Medical and Surgical Journal*, October 1829.

of urinary materials, but also by a loss of its own normal constituents. The density of the serum is reduced from 1,030 or 1,031 to 1,022 or even 1,020. The loss of density is greatest when the urine has been most albuminous; and it is probably in part explained by the escape of albumen through the kidneys, and partly by the retention of water. The hæmoglobin or colouring matter also diminishes rapidly, the normal proportion being 1,335 in 10,000. Dr. Christison found it reduced, after a few weeks' illness, to as low as 955 in one case, in another to 564; and in a young man ill for three months and a half subsequent to scarlet fever, who had never been bled, it was only 427. At the commencement of the disease, the loss of colouring matter is less rapid than the extreme pallor of the patient would seem to indicate; and it is probable that the blanched appearance of the skin is partly occasioned by the quantity of water in the subcutaneous tissue.

Etiology.—Acute Bright's disease may occur at all ages from infancy to extreme old age. The two most frequent causes of acute Bright's disease with dropsy are exposure to wet and cold, and scarlet fever. Either of these causes is alone sufficient to excite the disease; but their combined action—exposure to cold during the progress of scarlet fever—is a most powerful determining cause of the malady.

Another not uncommon cause of albuminuria in young men and boys is imprudently prolonged cold bathing. In the *Transactions of the Clinical Society*, vol. vii. p. 42, I have given some particulars of four cases of temporary albuminuria excited by cold bathing. Since the publication of that paper, I have met with several cases of confirmed degeneration of the kidney clearly traceable to repeated and prolonged cold bathing. One case of this kind I saw with my friend and former pupil, Mr. Alfred J. Bell, of St. John's Wood. A young man aged 19, in the summer of 1878, after bathing repeatedly in Teddington lock, noticed that his urine became 'almost as black as ink.' This was probably hæmaturia; but no chemical examination of the urine was then made. The dark colour passed away after a few days, and he had no symptoms of illness. He continued to take active exercise—fishing, shooting, and bicycling; and

seemed to be in good health until near the end of October 1879, when considerable dropsical swelling of the legs occurred. The urine was then found of normal colour, of specific gravity 1020, albuminous to the extent of one-half, and it deposited a sediment in which were found numerous hyaline and oily casts. In tracing back the history of this case, we found good reason to believe that the renal disease commenced eighteen months before, with congestion of the kidneys and hæmaturia, excited by the cold bathing; and that the albuminuria had continued from that time until the present.

This case is a type and illustration of many others that have come under my observation. Thus, in one case, albuminuria in a previously healthy young man was a result of wading through a river up to the shoulders at the end of a twenty-mile walk; and in another it was excited by swimming his horse through a river in fox-hunting, and allowing his wet clothes to dry upon him and to chill him. From what I have seen of the effects of cold bathing, I have arrived at the conclusion that more people are injured than are benefited by the practice; and I am confident that, if the urine of all the men, women, and children who paddle about in the sea until they are blue and cold, were tested within a few hours after their prolonged immersion, it would be found to be more or less albuminous in a large proportion of cases.

The cold probably acts by repressing the secretory function of the skin and so causing a form of blood-poisoning, the noxious influence of which may be felt in one case by the kidneys, in another by the lungs or other viscera.[1]

Diphtheria is a frequent cause of albuminuria; but, as I have before said, diphtheritic albuminuria is rarely associated with dropsy. Amongst the less frequent causes of acute albuminuria are measles, erysipelas, septicæmia, the absorption of poisonous materials from the uterus after parturition, rheumatic fever, the malarious poison, sewer poison, typhus and typhoid fever, cholera, and, lastly, excessive eating and drinking, more especially when combined with dyspepsia. The chain of events which connects albuminuria with dyspepsia is probably this—

[1] At the end of this section I have reported the result of suppressing the cutaneous secretion by varnishing the skin of an animal.

imperfectly digested food passes into the blood and loads it with impurities. The gland-cells of the kidney excrete these ill-digested products, and in doing so undergo structural changes, while the imperfectly assimilated albuminous materials pass more readily by exosmosis through the Malpighian capillaries. Further, the malnutrition resulting from chronic dyspepsia causes a general nervous exhaustion, with loss of vaso-motor nerve force, and consequent diminution of tone and contractile power in the muscular walls of the minute arteries generally, including those of the kidney, while the walls of the capillaries are probably weakened by depraved nutrition. Thus the filter and the fluid to be filtered are both materially changed, while the increasing inpurity of the blood throws more work upon the kidneys, and favours the passage of the altered albumen through the kidneys, the amount of albumen being often notably increased after food.

Various experimenters, and, amongst others, Professor Semmola, of Naples, have caused albuminuria and hæmaturia by injecting white of egg into a vein, or by repeated injections into the subcutaneous tissues. The introduction of this crude material causes the elimination of a larger amount of albumen than that which was injected.

In the majority of cases, acute albuminuria resulting from other causes than scarlet fever and exposure to cold is unassociated with dropsy, and its history belongs to that of the disease with which it is associated as a complication. We shall find hereafter that albuminuria resulting from one or other of the various causes here referred to sometimes leads to a chronic and incurable degeneration of the kidney. Excess of alcohol is a more frequent cause of chronic than of acute Bright's disease, but in a former chapter (pp. 299–300) I have given the particulars of a remarkable case of acute transient alcoholic albuminuria.

Diagnosis.—In most cases of acute Bright's disease, the symptoms are so obvious that the disease can scarcely be overlooked or mistaken for any other. The only cases in which there is a possibility of acute albuminuria being unrecognised are those in which it is unassociated with dropsy. But, the existence of albuminuria being discovered, it is not always

easy to determine whether this is the result of a recent acute attack, or of a chronic degeneration of the kidney. We shall be in a better position to discuss this important practical question after we have studied the various forms of chronic Bright's disease. Meanwhile, however, I may tell you that, as a rule, high-coloured, smoky, and blood-tinged urine, of high specific gravity, is usually an indication of a recent acute attack; and equally so is a copious sediment composed of epithelial and blood casts (figs. 11 and 12, p. 657), or of leucocyte casts (fig. 18, p. 666), alone or mixed with epithelial casts. The appearance of oily casts and cells (fig. 14, p. 659), in combination with numerous epithelial casts, does not materially affect the diagnosis. On the other hand, urine of low specific gravity and very pale in colour, yet highly albuminous, is usually evidence of chronic disease; and this evidence is strengthened by the appearance of numerous oily casts and cells unassociated with epithelial or leucocyte casts. Large hyaline casts (fig. 13, p. 658) in *pale*, highly albuminous urine point to disease not only chronic, but in an advanced stage. We shall return to this subject, and discuss it more fully in a future section.

Prognosis.—Acute Bright's disease has a tendency to terminate in complete recovery. It is essentially a curable disease, as much so as acute bronchitis or acute pneumonia. The earlier the patient comes under treatment, the better is his prospect of recovery; and, on the other hand, the longer the symptoms have continued without signs of amendment, the more grave does the prognosis become. The prognosis is, on the whole, more favourable in the young and middle-aged than in those more advanced in years; but the disease may prove mild and tractable even in very aged persons. For obvious reasons, the prospect of recovery is better in the case of those who can avoid exposure to cold and other injurious influences than when the patient's circumstances are less favourable.

In favourable cases, a copious secretion of urine, of comparatively low specific gravity and of paler colour, with a diminishing amount of albumen and decrease of dropsy, are amongst the earliest signs of amendment. Albuminuria is

usually the last symp om to disappear. The time of its disappearance varies, in different cases of recovery, from a few days to many months. If the urine continue albuminous for more than six months, it becomes more and more doubtful whether it will ever cease to be so; but I have seen cases of complete recovery after albuminuria had continued for one, two, and even three years; and in one case the urine ceased to be albuminous after seven years of persistent albuminuria excited by an attack of scarlet fever. So long as the urine continues albuminous, in however slight a degree, although the dropsy and all other general symptoms may have passed away, recovery must be considered incomplete. Acute Bright's disease, although, as a rule, a curable, is not unfrequently a fatal disease. There are some symptoms and complications which indicate a case of more than ordinary peril; such as a very scanty secretion of highly albuminous urine; frequent and distressing vomiting; great anasarca, with a tendency to erysipelatous inflammation of the skin; dropsical effusion within the chest, either in the pleura or the pericardium, or both, with urgent dyspnœa; inflammation of the lung, or pleura, or pericardium, or endocardium; severe and persistent headache, which is apt to be followed by convulsions and by coma, with a brown and dry tongue. All these are symptoms of grave, though not always of fatal, import. When the renal disease is acute, and therefore essentially curable, recovery sometimes occurs after the most formidable symptoms of uræmic poisoning have been present.

A consideration of the exciting causes of the renal disease forms an element in the prognostic indications. When Bright's disease results from some inherent constitutional defect, without obvious exciting cause, it is generally more intractable than when it is directly due to exposure to cold or to the influence of some specific blood-poison, as, for instance, that of scarlet fever or erysipelas. To all general rules of this kind there are exceptions, and each case requires a separate and careful study.

Let me impress upon you one point of practical importance. Before you pronounce a patient to be entirely free from his malady, be careful to test the urine, not only after

rest and fasting—*i.e.* in the morning before breakfast—but after food and exercise. Albuminous urine is usually more copiously so after food and exercise; and you will sometimes find that, while the urine before breakfast is quite free from albumen, that which is secreted after a meal is decidedly and even copiously albuminous. In some cases, exercise has even more influence than food in exciting renal congestion and albuminuria.

An attack of acute Bright's disease confers no immunity from future attacks; on the contrary, the disease may occur more than once in the same subject, a result either of inexplicable predisposition or of a liability resulting from a first attack. I think my experience warrants the statement that when acute albuminuria has resulted from some non-specific cause, such as exposure to cold and wet or excessive eating and drinking, it is more likely to recur than when it has been excited by a specific morbid poison, such as that of scarlet fever, which, as a rule, does not occur a second time in the same individual; but I have known patients so unfortunate as to have two attacks of scarlet fever, and each attack complicated with albuminuria. I shall defer the important question of treatment until we have passed in review the various forms and complications of chronic Bright's disease.

The Deleterious Results of Suppressed Action of the Skin illustrated by Experiment.

The rapidly fatal result of varnishing the skin of an animal after removing the hair affords the most striking illustration of the deleterious influence of suppressed cutaneous secretion.

In October 1873, Professor Rutherford, having closely cut the hair of two rabbits, covered the skin, first with melted gelatine, and, when this had been dried before the fire, a coating of ordinary spirit varnish was applied. One animal was then covered with a thick layer of cotton wool, the other was left uncovered. Both rabbits were kept in a room with a fire. The experiment was completed at 5 P.M. At 11 A.M. the next day the uncovered rabbit was dead, cold, and stiff. The other animal was still alive, but at 4 P.M. he was nearly dead. The next morning he was dead and stiff. So that one rabbit died

in less than eighteen hours, the other in about twenty-four hours after the skin had been coated.

In No. 1, the uncovered rabbit, there was some ecchymosis beneath the peritoneum, and at the lower part of the middle lobe of the right lung. The mucous membrane of the trachea was slightly congested. The bladder was half-full of highly albuminous urine. The kidneys were red, their structure was normal.

No. 2. Nearly the whole of the left lung was in a state of intense engorgement passing into hepatisation, and a portion of the lower lobe of the right lung was in the same condition. The mucous membrane of the trachea and bronchi was dark red and much congested. The bladder was full of urine of normal colour but highly albuminous. The kidneys were red, their structure normal. This animal having lived some hours longer than the other, the changes in the lungs were much greater; whether life was prolonged by the covering of cotton wool it is impossible to say from a single experiment.

SECTION V.

CHRONIC BRIGHT'S DISEASE WITH A SMALL RED GRANULAR KIDNEY.

Small Red Granular Kidney—Synonyms—Outward Appearance of the Kidney in different Stages—General History of the Disease—Chemical and Microscopical Characters of the Urine—Microscopic Appearances in the Kidney—The Structural Changes are essentially tubular and intratubular—Changes in the Blood-vessels of the Kidney—Physiological Explanation of the Structural Changes in the Kidney and of the Condition of the Urine—Local and General Symptoms of Contracted Granular Kidney; frequent Micturition—Pain in the Back—Dyspepsia as a Cause and a Consequence of Renal Disease—Dropsy—Hypertrophy of the Heart—Inflammation of Serous Membranes—Hæmorrhage from various Surfaces—Cerebral Hæmorrhage—Impairment of Vision—Albuminuric Retinitis—Cerebral Symptoms the Result of Uræmia—Theory of Uræmia—Nervous Dyspnœa—Disease of the Liver—Inaction of Skin—Diagnosis—Prognosis.

Classification of Chronic Bright's Disease.—Cases of chronic Bright's disease arrange themselves, anatomically and clinically, in two very distinct classes. In one class of cases, the

kidney is found small, red, and granular; in the other class, on the contrary, the kidney is large, pale, and usually smooth on the surface.

Small Red Granular Kidney.—The clinical history of the two classes of cases is as distinct as are their anatomical characters. For various reasons, it will be more convenient to take first in order those cases which are associated with the small red granular kidney. The disease is usually designated 'granular kidney,' with the *synonyms* 'contracted granular kidney,' 'chronic desquamative disease,' 'gouty kidney,' 'interstitial nephritis,' 'cirrhotic kidney.'

Outward Appearance of the Kidney in Different Stages.—At no period of the disease is there an appreciable enlargement of the kidney, but from the commencement a process of wasting occurs. In the early stage, when death has resulted from some other disease, the capsule is found adhering firmly to the surface of the gland, so that it is difficult to tear it off without bringing away some of the adherent glandular tissue. The fine lobular markings are less distinct than in the normal state, and the surface of the kidney is slightly uneven and granular. As the disease advances, there is progressive wasting of the cortical glandular portion of the kidney, with granular unevenness of the surface and diminution of the thickness of the cortex; so that by degrees the bases of the medullary cones approach nearer to the surface of the gland. In extreme cases, the kidney may be reduced to one-half or even one-third of its normal size and weight. The cortical secreting portion of the gland is evidently the part chiefly implicated, while the medullary cones are nearly intact. The contracted kidney is somewhat firmer and tougher than natural. In all stages of the disease, one or more, sometimes numerous, serous cysts may be seen projecting from the surface, varying in size from a pin's head to a pea, but sometimes as large as a filbert, or even larger. Even in the most advanced stages of atrophy, the organ retains more or less of its normal red colour and its vascularity, with only here and there pale patches, where, with atrophy of other tissues, the blood-vessels have become obliterated; hence it is called the *red* granular kidney, to distinguish it from certain cases of

chronic Bright's disease to be referred to hereafter, in which the kidneys are granular, but white and anæmic.

The appearance of the red granular kidney in an advanced stage is represented by Plate V. (erroneously printed Plate III.) in my paper *On the Forms and Stages of Bright's Disease of the Kidneys.*[1]

General History and Causes of the Disease.—Some general facts relating to the disease it may be well to point out now. The disease is essentially chronic from the commencement, and rarely, if ever, a sequel of an acute attack. Its commencement, therefore, is, as a rule, insidious, and in its early stages it is often unsuspected and latent. It is a comparatively rare disease in early life, though not uncommon between the ages of twenty and thirty: bnt the majority of cases occur in persons at and beyond middle age. It is often associated with the gouty diathesis, as one of its synonyms indicates; and it is of common occurrence in persons who eat and drink to excess, or who, not being intemperate in food or drink, suffer from certain forms of dyspepsia, without the complication of gouty paroxysms. In some cases, the disease probably results from habitual exposure to cold and wet, and consequent suppression of the cutaneous secretion. There is reason to believe that chronic poisoning by lead is, at any rate, a concurring cause of the disease amongst painters, and others who are exposed to the influence of this pernicious metal. Sir A. B. Garrod was the first to direct attention to the influence of lead in the causation of gout: and Dr. Dickinson states that, out of forty-two men exposed to lead-poisoning who had died in St. George's Hospital, twenty-six had granular degeneration of the kidneys, which in most cases was so advanced as to have caused death.[2] Allowing, as we must, that the lead had great influence, it is probable that habits of intemperance and other causes may have co-operated with the lead. Granular kidney is occasionally, though rarely, found as a sequence of the albuminuria which is associated with pregnancy. I have seen one well-marked instance of this.

[1] *Med.-Chir. Trans.*, vol. xlii. p. 153.

[2] *On the Pathology and Treatment of Albuminuria.*

Dr. Clifford Allbutt has directed attention[1] to the influence of mental anxiety in causing granular degeneration of the kidney. I do not agree in opinion with Dr. Allbutt that 'mental anxiety is one of the chief, if not the chief, cause of granular kidney;' but I believe that there is a real etiological relation between mental anxiety and some cases of albuminuria; and I have often seen, in cases of chronic renal disease, a great increase of albumen under the disturbing influence of mental emotion. In my own experience, however, saccharine diabetes has much more frequently than albuminuria been traceable to mental and emotional influences; and it has appeared to me that as, in the diabetic cases, the saccharine urine, whether in the rabbit, whose medulla oblongata has been mechanically irritated by the operator's needle, or in the human subject, whose brain has been tortured by mental anxiety, the primary influence of the nerve-disorder is upon the sugar-forming liver, so, in an analogous manner, the albuminuria which results from mental anxiety is a secondary result of a nervous influence acting primarily on the liver and the stomach —the so-called chylopoietic viscera. In fact, it seems to me that the albuminuria which appears to have resulted from mental and emotional influence is a form of albuminuria from dyspepsia; and the immediate cause of the renal disorder is the excretion of some abnormal products of imperfect digestion. In many of these cases, too, it is obvious that more than one etiological agency has been operative. The man or the woman depressed by mental anxiety, with a failing appetite and disturbed sleep, often seeks temporary relief from misery in an alcoholic stimulant; and so it is often found that the noxious influence of alcohol, as a substitute for wholesome food, has to be taken into account in explaining the albuminuria and the renal degeneration.

The atrophy with granulation which results from passive congestion of the kidney consequent on valvular disease of the heart or emphysema with chronic bronchitis, has a different pathological history; and I shall refer to it in a future section.

During the progress of the disease which results in the contracted granular kidney, dropsy rarely forms a prominent

[1] *British Medical Journal*, February 1877.

symptom, and in the majority of cases it is entirely absent. The disease is often associated with high arterial tension and with hypertrophy of the left ventricle of the heart, even when there is no valvular defect or disease of the walls of the larger arteries to explain the cardiac hypertrophy. In a large proportion of cases, the immediate cause of death is uræmia or cerebral hæmorrhage.

Now, in the course of these lectures, I shall as much as possible avoid all controversial topics; but, in proceeding to give you what I believe to be the true account of the minute anatomy and pathology of this disease, I am bound to tell you that I dissent from the opinions of some pathologists for whom I entertain great respect, but not sufficient to induce me to follow them into what I believe to be an erroneous reading and interpretation of facts. Virchow, in his *Cellular Pathology*, stated that as there are three anatomical elements in the kidney—namely, tubes, vessels, and interstitial tissue; so, in accordance with this, there are three forms of Bright's disease—what he calls parenchymatous nephritis, having its seat in the tubes; amyloid degeneration in the blood-vessels; and interstitial nephritis, consisting essentially, as he believes, in thickening of intertubular tissue and consequent atrophy and granular contraction of the kidney. Virchow admits that two and sometimes all three of his forms of disease may coexist in the same kidney; and I maintain that in every case of Bright's disease, all the tissues are implicated; the various forms of disease depending, not upon the implication of different anatomical elements in the morbid process, but upon the varying nature of the structural changes which these elements undergo in the different classes of cases.

Many of the exciting causes of the acute desquamative disease are also influential in the causation of the chronic degeneration which results in the red granular kidney; and there is a strong *à priori* improbability in the assumption that an identical pathological cause should, in one individual, act primarily and mainly upon the gland-cells, and in another upon the connective tissue of the gland. Again, it is obvious to the naked eye that, as in other forms of Bright's disease so in this, the part of the kidney which is primarily and

chiefly affected is the cortical glandular part, where, to say the least, the connective tissue is the scantiest. If the pathological process were essentially a hyperplasia of connective tissue, it would surely have its seat mainly in the medullary cones, where the connective tissue is admitted to be most abundant.

My doctrine with regard to the minute anatomy and pathology of the granular kidney is, that it consists primarily and essentially in a disintegration and destruction of the gland-cells which line the convoluted tubes, the *débris* of the gland-cells appearing in the urine as granular tube-casts; that the destruction of the gland-cells induces atrophy and contraction of the tubes; that this shrinking of the tubes, with fibrillated thickening of their membranous walls and of the Malpighian capsules, gives the delusive appearance of a greater amount of newly formed fibrous tissue than actually exists; and that thickening of the walls of the arteries, the nature of which I shall presently describe, forms one of the most constant and conspicuous features of the disease.

Chemical and Microscopical Characters of the Urine.—You will find that the minute structural changes in the contracted kidney are rendered more easily intelligible if you study them in connection with the clinical history of the disease, and in particular with the chemical and microscopical characters of the urine. I have told you that the disease, although not exclusively of gouty origin, is often associated with chronic gout. Examine the urine of a man who has had repeated attacks of gout, and you will not unfrequently find in it the earliest indications of incipient renal degeneration. The urine may be of normal colour and specific gravity, and without a trace of albumen; but, after standing for a few hours in a conical glass, it deposits a light cloud, which, on microscopic examination, is found to consist of scattered granular *débris* and tube-casts such as are represented in fig. 19, p. 684. These casts contain epithelial cells, some entire, others in various grades of disintegration, and hence arises their 'granular' appearance. Every granular cast is not, of necessity, composed of disintegrated epithelium. Blood-corpuscles may become disintegrated within the uriniferous

tubes, and appear in the urine as granular blood-casts, distinguished from granular epithelial casts by their reddish-brown colour, and often by containing some entire blood-corpuscles; so, disintegrated hyaline casts may assume a granular appearance; but by a comparison with other casts associated with them, and by noting the various grades of change, we trace them to their true source. The presence of granular epithelial casts and of scattered epithelial *débris* is evidence that a process of epithelial desquamation and disintegration has commenced in the kidney. In the earlier

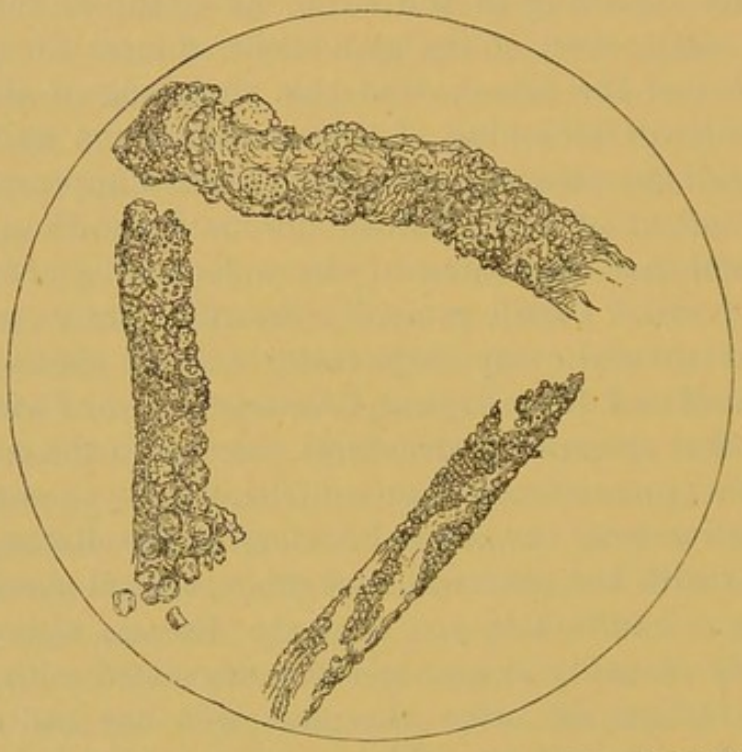

FIG. 19.—GRANULAR CASTS, COMPOSED OF MORE OR LESS COMPLETELY DISINTEGRATED EPITHELIUM AND FIBRINE.—× 200.

stages of the renal disease, the granular casts are found only during, or immediately after a gouty paroxysm, and, as I have already said, unassociated with albuminuria. In the intervals between the attacks of gout, no tube-casts are visible. At a later stage, granular casts and epithelial *débris* are always present in a greater or less amount; and the urine becomes albuminous, at first only during a fit of gout, the tube-casts also being more abundant during the paroxysm. At a still later period, tube-casts and albumen are more or less constantly present, though both may, in exceptional

cases, be absent, even in the most advanced stages of this form of disease.

Not only is the particular form of renal disease indicated by the microscopic appearance of the urinary sediment, but the number of granular tube-casts and the amount of epithelial *débris* indicate the rate at which the disease is progressing. The more copious the sediment, the more rapid is the destruction of the gland-cells, and the consequent atrophy of the kidney. In the more advanced stages of the disease, large hyaline casts are often found associated with the granular casts (fig. 20).

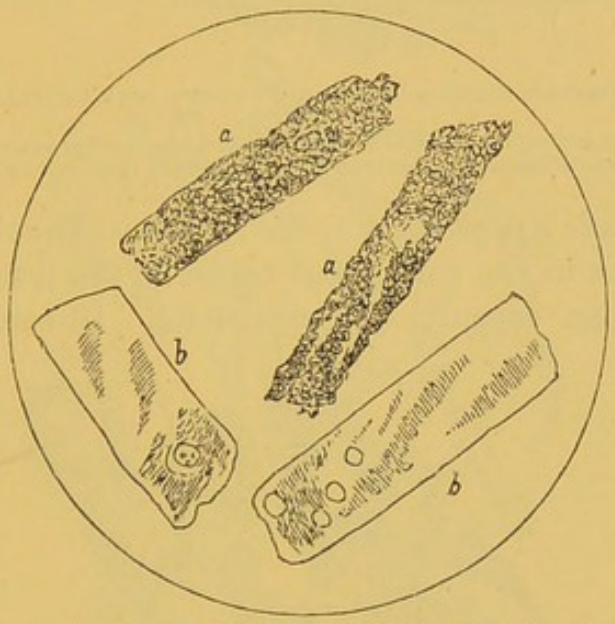

FIG. 20.—*a a*. granular casts. *b b*. large hyaline casts from tubes denuded of epithelium.—× 200.

As the disease makes progress, the urine undergoes remarkable physical changes. The quantity secreted is usually in excess of the normal amount; and with the increase of quantity there is commonly associated a loss of the natural colour and a diminution of the specific gravity, which, usually as low as 1010 or 1012, sometimes falls to 1005. The low specific gravity indicates a relative decrease of the normal solid constituents, especially of urea, uric acid, and extractive matters. In one of Dr. Christison's cases, the total solids discharged were reduced to one-fifth, and in another nearly to one-twelfth, of the healthy average. This defective discharge of solids is partly explained by the rest in bed, the scanty

diet, and the general anæmia. The amount of albumen varies considerably. Absent or scanty in the early stage, it may be rather copious in the middle periods, and again scanty in the stage of extreme degeneration of the kidney.

FIG. 21.—TRANSVERSE SECTIONS OF TUBES, CONTAINING ONLY GRANULAR DÉBRIS OF EPITHELIUM.
At one end of the section, the contents of the tubes have been washed away, and the sections of the basement-membrane form three empty rings.—× 200.

Microscopic Appearances in the Kidney.—The chief changes will be found in the convoluted tubes, in the arteries, and in the Malpighian capsules and capillaries. In some tubes the

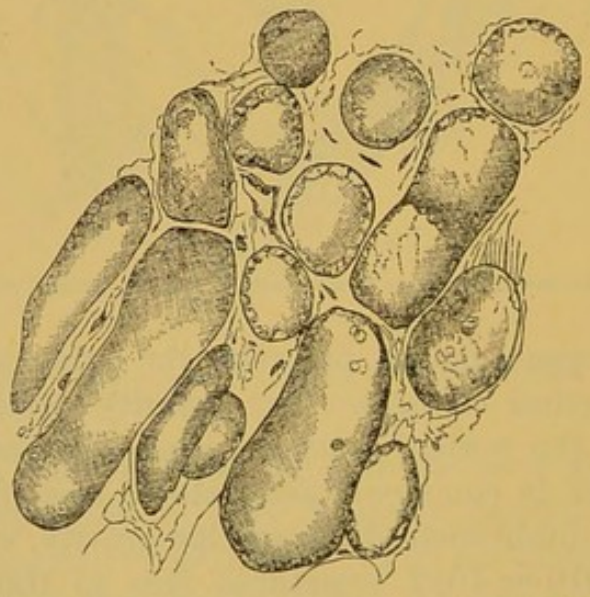

FIG. 22.—TUBES MORE OR LESS COMPLETELY DENUDED OF EPITHELIUM.
Some transversely divided and cyst-like; others seen lengthwise.—× 200.

gland-cells have their normal appearance, or they are opaque and granular, with a clear central canal (see *ante*, fig. 17, p. 665). Other tubes are filled and rendered opaque with desquamated epithelium more or less disintegrated (see fig. 15, p. 661).

Others, again, present the characteristic appearance represented in fig. 21. Their epithelial lining has become disintegrated and removed, appearing in the urine in the form of the granular casts before described (figs. 19 and 20). A few granular particles of epithelium only remain, and these appear to be held together by fibrinous coagula. The transverse sections of tubes in this condition have somewhat the appearance of oval or globular cysts, and many years ago they were described as microscopic cells by a very able observer.[1] When, in the same section, some tubes appear transversely divided, while others present themselves lengthwise, as in fig. 22, all having the same general structure and contents,

FIG. 23.—TUBES IN PROCESS OF ATROPHY AND CONTRACTION AFTER THE DESTRUCTION OF THEIR EPITHELIAL LINING, A FEW GRANULAR PARTICLES ONLY REMAINING WITHIN THEM.—× 200.

it is easily seen that the cyst-like appearance is given by transverse sections of partially or completely denuded tubes.

The number of tubes thus denuded of their epithelial lining varies much in different cases. In some kidneys, which to the naked eye present comparatively little deviation from the normal state, the destruction of gland-cells is found to have been very extensive. Other tubes are found, as regards their appearance and contents, in the same condition as those just now described, but shrunken and atrophied, with wide inter-

[1] See Simon's paper on Subacute Inflammation of the Kidney, *Med.-Chir. Trans.*, vol. xxx.

spaces between them—the interspaces being occupied by the remains of other atrophied tubes and capillaries (see fig. 23). Atrophy and contraction of the tubes appears to be the usual result of the destruction and removal of their gland-cells. But the opposite condition of dilatation is found in some of the tubes, which may be seen often as large as Malpighian bodies, and even larger (fig. 24); and there can be no doubt that these dilating tubes at length form the cysts, which are visible by the naked eye.

Some tubes which have lost their epithelial lining are filled

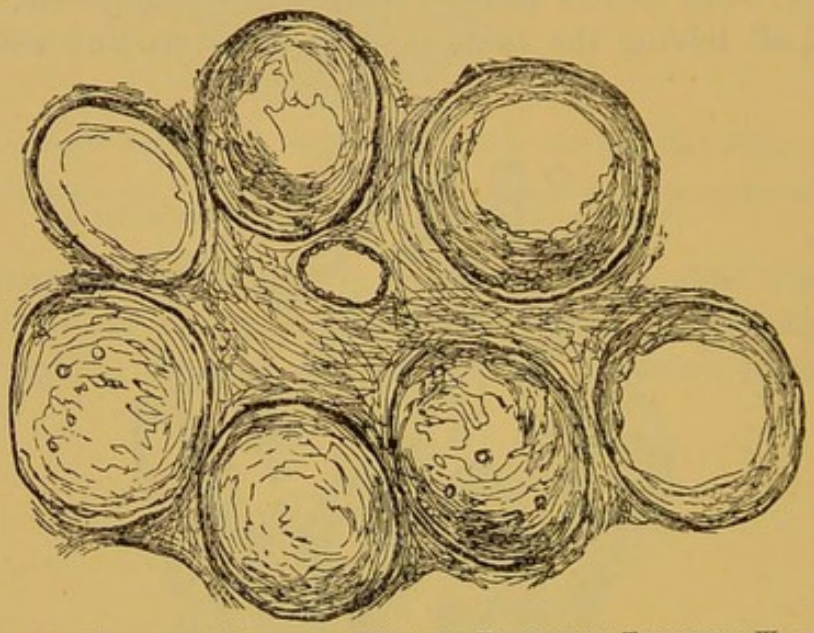

FIG. 24.—TRANSVERSE SECTIONS OF DILATED TUBES WITH THICKENED WALLS.
In some sections the open mouth of the divided tube is seen—one section of a denuded tube of normal size.—× 200.

with an unorganised material having the same appearance as the 'large hyaline casts' which are commonly present in the urine, and which, as we have seen, are moulded within tubes which have been deprived of their epithelium (figs. 20, 22, and 25).

There is another appearance of the tubes in the granular kidney, which probably has a close relation with the formation of dilated tubes and cysts. This appearance is represented in fig. 26. The tubes are lined by a layer of small transparent cells of a more or less globular form, and each having a single nucleus.[1] Some tubes in this condition may be found in every

[1] These uni-nucleated cells were described and figured forty years ago, and

granular kidney, but their number varies considerably. In some contracted kidneys the tubes with the uni-nucleated round-cell lining are few in number, while in others they are very numerous; and there appears to be a close and special relationship between these tubes and the cysts with which, in the same kidney, they are always associated. That the cysts are dilated tubes is now generally admitted; and it is probable that the tubes are dilated by the accumulation of a secretion which is formed by this peculiar cell-lining. This, at any rate, is certain, that, as the contracted granular kidney is the only form of Bright's disease with which, as a rule, cysts and tubes lined by the peculiar cells in question are associated; so when a large white kidney has reached the stage of atrophy

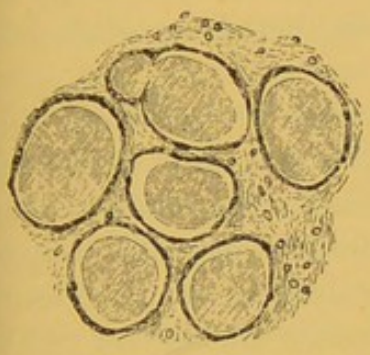

FIG. 25.—DENUDED TUBES FILLED WITH HYALINE MATERIAL.

The shading indicates the stain of the hyaline material by the logwood dye.

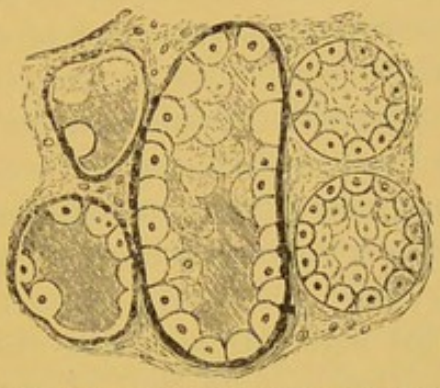

FIG. 26.—SECTIONS OF TUBES IN WHICH A LAYER OF TRANSPARENT CELLS, EACH WITH A SINGLE NUCLEUS, HAS TAKEN THE PLACE OF THE NORMAL EPITHELIUM.

One tube is seen lengthwise. In three of the segments a hyaline material, coloured by the logwood dye, is seen within the cell-lining.

and coarse granular contraction, cysts visible by the naked eye, and the microscopic appearance of tubes whose normal epithelium has been replaced by uni-nucleated cells, are again found in combination.

Dr. Conway Evans has recorded a remarkable case, in which one kidney was of the natural size, and appeared outwardly quite normal, while the other was honeycombed with cysts. Both kidneys, however, presented the microscopic appearances

quite independently, by both Sir John Simon and myself, *Medico-Chir. Trans.* vol. xxx. It will be seen that they differ essentially from white blood-cells and from the nuclei of epithelium; from the latter especially in the fact that they are in close contact with each other, so as to form a continuous lining for the tube.

which I have described as characteristic of the red granular kidney; and in particular, many tubes were lined by 'round or oval transparent cells, each containing in its interior a single well-defined nucleus.'[1]

It is probable that the dilatation of the tubes by their accumulated liquid contents, and their conversion into cysts, is promoted by the compression to which they are subjected by the surrounding tubes, in a more advanced stage of degeneration, whose thickened and shrunken basement-membrane constricts the neighbouring tissues. But the case before mentioned, as recorded by Dr. Conway Evans, proves that a great development of cysts may occur with little or no wasting of the gland. In that case the right (healthy-looking) kidney weighed four ounces and a quarter, while the left, which was in a more advanced stage of cystic degeneration, weighed five ounces.

I have yet to mention that the lumen of the tubes which are lined by the round uni-nucleated cells is often filled by an unorganised fibrinous material similar to that which is found in the tubes represented in fig. 25, which have no cell-lining of any kind; and in like manner the hyaline material is coloured by the logwood violet dye, while the cells themselves remain colourless (see fig. 26). These hyaline, or, as they are sometimes called, 'colloid,' tubes may be seen in every granular kidney; and it seems probable that the hyaline material may be secreted by the layer of cells within which it lies. Sometimes, though rarely in this form of disease, a tube is found injected with blood which has escaped from ruptured Malpighian capillaries. Here and there a tube may be seen whose contents have undergone a fatty transformation; the oil globules being either contained within cells, or free and irregularly accumulated within the tube.

The basement-membrane of tubes whose gland cells have undergone degenerative and destructive changes is often more or less thickened; and this thickening and fibrillation of the tubular membrane, together with the wide spaces occupied by the atrophied remains of shrunken tubes and intertubular capillaries (fig. 23, p. 687), have given rise to the doctrine of a

[1] *Pathological Transaction*, vol. v. p. 183.

primary new formation or increase of connective tissue, as being the essential cause of the intratubular changes.[1] When a granular kidney has been reduced to the half or one-third of its normal size and weight, there can be no question that this is mainly due to the destruction and disappearance of the gland-cells, and that, in what remains of the wasted organ, there must be an enormous relative excess of fibroid tissue, composed of fibrilliform basement-membrane, thickened Malpighian capsules, and bloodless capillaries.[2]

From an examination of the most advanced stage of degeneration it would be impossible to ascertain, amidst the wreck and ruin of the tissues, the starting-point and the sequence of the pathological changes. A microscopic section of a portion of kidney in the stage of extreme degeneration shows little more than such a perplexing mixture of fibrous tissue, with granules and globules, as is well represented in fig. 1, Plate V., in the paper *On Chronic Bright's Disease with Contracted Kidney*, by Sir William Gull and Dr. Sutton.[3]

For a clear understanding and a correct interpretation of the origin and sequence of the structural changes in a granular

[1] In a case of extreme emaciation, the result of pulmonary phthisis or diabetes or cancerous cachexia, there is a great relative excess of bone, cartilage, and tendinous and fibrous tissue in the dead body; but the atrophy of muscle and fat is not supposed to result from an overgrowth of the osseous cartilaginous and fibroid tissues.

[2] With regard to the increase of the fibrous tissue in the granular kidney, I am in entire agreement with such excellent authorities as Drs. Wilks and Moxon, who make the following statement (*Lectures on Pathological Anatomy*, 2nd ed. p. 509):—'Some authors, especially Dr. Dickinson, describe an increase of fibrous tissue; we think there is a little increase round the vessels, but not much. The patches apparently of fibrous tissue figured in Dr. Dickinson's excellent drawings we have always seen, but high powers resolve these patches into the remains of tubes, as indeed would probably follow from consideration of the drawings themselves. His drawing of healthy kidney has seven Malpighian corpuscles, but a drawing of granular kidney on the same scale, but of half that size, has twenty-one of these corpuscles with but little tissue between; now, this shrinking to one-sixth of the bulk is accompanied by dilatation of some tubes, so that a considerable space must be occupied by the compact walls of the necessarily numerous shrivelled ones. We have found that a fibrillated condition prevails in the tube-walls, which become coarse-looking; but all large patches of apparent fibre we have always found to be chiefly made up of apparently wasted tubes.'

[3] *Med.-Chir. Trans.*, vol. lv.

kidney, it is absolutely necessary to examine carefully the primary stages of the pathological process.

I have before stated that the earliest indication of a structural change in the kidney is afforded by the appearance of granular casts in the urine, and that is an important item of evidence in support of the view that the gland-cells are the primary seat of the disease.[1]

Amongst the many and insuperable objections to the theory of a primary intertubular fibrosis, is the fact that the granular kidney retains more or less of its red colour and its vascularity, even in the most advanced stages of the disease. It is called, and correctly called, the small *red* granular kidney. This appearance is quite inconsistent with the doctrine of an intertubular deposit, which must inevitably obliterate the capillaries, and render the cortex more or less pale and anæmic. Such an appearance of anæmia is general over the cortex of a large white kidney, when the tubes, being distended by their contents, compress the intertubular capillaries. Again, such an anæmic appearance is visible over a circumscribed spot when arteries and capillaries are obstructed by embolic particles of fibrine. If in the granular kidney the intertubular capillaries were obliterated, as they must be, by an interstitial fibrosis, we should expect the epithelial lining of the tubes to undergo the same changes as result in it from embolic plugs—namely, a fatty transformation prior to complete atrophy. On the contrary, a fatty condition of the epithelium in cases of small granular kidney is quite exceptional.

In strict agreement with the red colour and the obvious vascularity of the granular kidney, as seen by the naked eye, is the fact that, on a microscopical examination of a section of the kidney, the characteristic intratubular changes may often be seen in various stages of progress, while the intertubular capillaries surrounding these morbidly changed tubes are injected with blood and evidently quite normal. I have seen this in numberless sections of granular kidney, and I maintain that this fact alone is absolutely inconsistent with the interstitial hypothesis. Again, compression of the intertubular capillaries by morbid deposit or growth would evidently

[1] See *ante*, p. 683.

cause great engorgement of the Malpighian capillaries, with resulting copious albuminuria and hæmorrhage into the tubes; but in cases of granular kidney the albumen is, as a rule, less copious than in any other form of Bright's disease. It is often very scanty, and it is sometimes entirely absent. There was no albumen in Dr. Conway Evans's case before referred to (p. 689).

There is often an appearance of fibrous connective tissue round the Malpighian bodies. I am not prepared to say that no connective tissue is ever formed external to and apart from the fibrous capsule of the Malpighian body; but I am convinced that, when the capsule is thickened, as it often is in the granular kidney, and then thrown into folds, the fibrous appearance which it presents, as different depths of the globular capsule are being focussed into view, may very readily be mistaken for fibrous connective tissue outside and surrounding the capsule (see fig. 27).

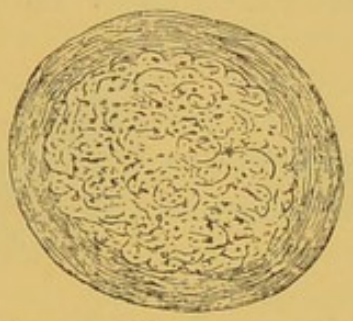

FIG. 27.—MALPIGHIAN BODY—THE CAPSULE THICKENED AND HAVING A FIBROUS APPEARANCE. THE CAPILLARIES THICKENED AND OPAQUE; THE NUCLEI VISIBLE IN THEIR WALLS.—× 200.

Some overgrowth of the fibrous and connective tissues of the kidney may result from these tissues assimilating part of the nutritive material which is no longer taken up by the neighbouring disorganised gland-cells; the overgrowth of the fibrous tissues being a consequence and not the cause of the destruction and disappearance of the epithelium.

In consequence of wasting and contraction of the tubes, some of the Malpighian bodies are brought nearer together, and three, four, or more, may sometimes be seen almost in contact with each other. Sections of dilated tubes, such as are represented in fig. 24 (p. 688), may easily be mistaken for Malpighian bodies; but in the sections of tubes the open

mouth of the cut tube may often be seen and in the Malpighian bodies the capillaries, with the nuclei in their walls, are characteristic; though during the atrophic process which ultimately involves all the tissues, some of the Malpighian capillaries become indistinct, and finally disappear.

Now, to recapitulate. We have found the following pathological appearances in the tubes: the epithelium opaque and granular, in a state of cloudy swelling; the tubes crowded and opaque, with degenerated and disintegrated epithelium; some tubes deprived of their epithelium; some contracted; others dilated in various degrees; some lined by transparent uni-nucleated cells; others filled with unorganised fibrine, rarely with blood, or with oil; lastly, the basement-membrane and the Malpighian capsules are thickened; while the Malpighian capillaries are all thickened, and some are collapsed and shrunken almost beyond recognition. All the changes which I have described, and which you may see in the specimens which I have placed under the microscopes on the

FIG. 28.—NORMAL ARTERY FROM THE KIDNEY. × 200.

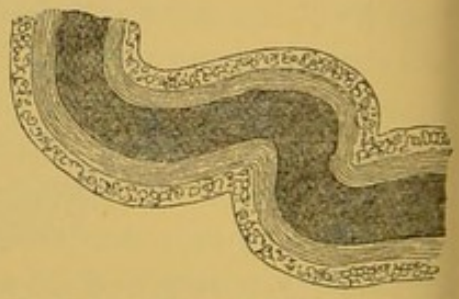

FIG. 29.—ARTERY, WITH HYPERTROPHIED WALLS, FROM THE KIDNEY.
An inner longitudinal and an outer circular layer of fibres, of about equal thickness. The canal is injected.—× 200.

table, are essentially tubular and intratubular. You see, then, with how little reason the contracted kidney is spoken of as the result of an essentially intertubular disease.

Changes in the Blood-vessels of the Kidney.—Amongst the most constant and interesting anatomical changes are those which occur in the minute arteries. In the advanced stages of the contracted granular kidney, the middle and inner tunics of the renal arteries are much hypertrophied, while the external tunica adventitia remains thin and inconspicuous. There is a sharp line of demarcation between the circular

fibres of the muscular layer and the longitudinal fibres of the intima (fig. 29).

The thickening of the arterial coats is due to an excess of normal tissue, with no appearance of structural change or degeneration. The hypertrophy is usually most conspicuous in the smallest arterial branches, and a comparison of the afferent artery of the Malpighian body in a healthy kidney with one from a small granular kidney will often show that the arterial walls in the latter are twice or even thrice the normal thickness.

I discovered this great thickening of the arterial walls many years ago, and published the fact, with an attempt at an explanation, in the *Medico-Chirurgical Transactions*, vol. xxxiii. Figs. 28 and 29 are reproduced from the illustrations contained in my paper.

At that time and long afterwards I believed that not only was the outer circular layer muscular, but the inner longitudinal layer also. I supposed the thickening of both layers to be a result of hypertrophy of muscular tissue; and the explanation which I suggested was that, in consequence of an impeded circulation through the intertubular capillaries, the arterioles, by their forcible action, assisted the heart to drive the blood onwards, and that in so doing they became hypertrophied. The excuse for this erroneous physiology is that it was published in 1850, therefore before the experiments of Bernard and Brown-Séquard had revealed the true function of the muscular arterioles.[1] I am now enabled to give a more correct anatomical account of the thickened longitudinal layer, and to suggest a truer physiological interpretation of the phenomena. The increase of the inner longitudinal layer of the hypertrophied renal arterioles is due to a thickening of the tunica intima. The intima is composed of three distinct layers—an outer wavy elastic layer in contact with the muscular coat, an inner endothelial layer, and an intermediate layer of delicate connective tissue. I am indebted to my friend Dr. Heneage Gibbs for some beautiful stained sections of granular kidneys. From an inspection of these it is manifest that the thickening of the intima in cases

[1] See *ante*, Chapter II. pp, 16 *et seq.*

of granular kidney is caused by *an increase of the connective tissue between the elastic and the endothelial layer* (figs. 30 and 31). Then the question arises, Is this thickening the result of a pathological inflammatory process, which, therefore, probably impedes, more or less, the functions of the vessels in which it occurs, or is it not rather of the nature of a physiological overgrowth, having a conservative purpose and tendency? The chief facts which appear to indicate that the thickening in question is physiological and conservative are the following :—

It is always associated with the hypertrophy of the muscular coat, and bears a constant relation to it. The structural characters of the thickened intima are remarkably uniform, its parallel fibres passing in the direction of the axis of the artery and at right angles to the circular muscular fibres, while the canal of the artery retains its uniform and natural

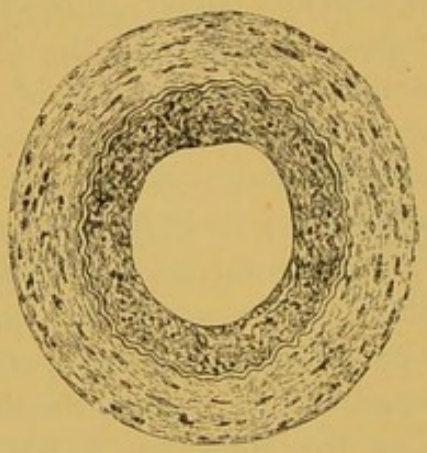

FIG. 30.—TRANSVERSE SECTION OF AN ARTERY FROM A GRANULAR KIDNEY.

The muscular coat much hypertrophied and consisting of several layers of circular fibres. Between the external wavy elastic layer of the intima and the endothelial layer an overgrowth of normal connective tissue. The tunica adventitia, which was inconspicuous, is not represented. — × 200.

diameter throughout its entire course.[1] In a typical example of granular kidney *every* renal artery is seen to have undergone the same change, and *all* the arteries to an almost equal extent. In contrast with this uniform and universal arterial thickening we find that when the arteries have become thickened by

[1] The appearance of the hypertrophied arterial walls is admirably represented in figs. 30 and 31. These figures, as well as figs. 25 and 26 (p. 689), were engraved by Messrs. Danielsson and Co., of 52 Beaumont Street, from accurate drawings of the specimens under the microscope by Mrs. Danielsson.

syphilitic, or tuberculous, or other undoubted pathological processes, not only are different parts of each artery affected in unequal degrees, by changes which tend to distort and often to obliterate their canals, and to confuse and more or less completely destroy all trace of their normal structure, but side by side with arteries in a state of very advanced disease may often be found others in a quite normal condition.[1]

If, as some pathologists maintain, the thickening of the intima in cases of granular kidney is a result of endarteritis—of inflammation excited by the morbid quality of the blood—is it not strange that, unlike the recognised inflammatory thickenings, this process should always be limited by the elastic layer, and that it should never invade the muscular coat ?

The probable result and purpose of the thickening of the intima is that, by strengthening the arterioles in their longi-

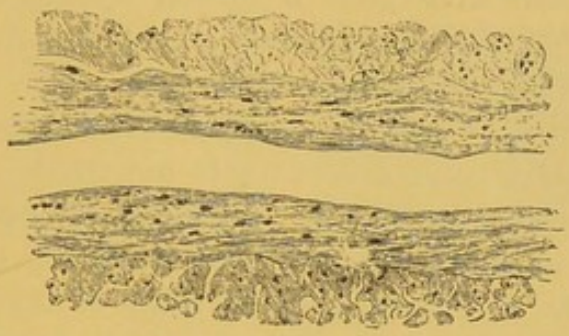

FIG. 31.—LONGITUDINAL SECTION OF AN ARTERY FROM A GRANULAR KIDNEY.

The circular fibres of the hypertrophied muscular coat are seen in section. The longitudinal fibres of the connective tissue of the intima much hypertrophied. The adventitia not shown.—× 200.

tudinal direction, it enables them the better to resist the strain and the resulting tendency to elongation to which they are subjected by the impulse from the greatly hypertrophied left ventricle; with which, as I shall presently explain, this arterial thickening is always associated.

Notwithstanding the increased power of resistance which must be given by this overgrowth of connective tissue, the renal arteries, which normally take a straight course, are often

[1] The striking contrast between general hypertrophy of the walls of the arterioles and the changes which result from inflammation may be seen by referring to two papers with illustrations of syphilitic disease of the arteries—one by Dr. Greenfield, the other by Dr. Thomas Barlow (*Pathological Transactions*, vol. xxviii.).

found in a granular kidney to be more or less tortuous, as seen in fig. 29 (p. 694).

There can, I think, be no doubt as to the thickening of the arterial walls being the result of as true a physiological and conservative overgrowth of normal tissue, as is the hypertrophy of the left ventricle of the heart, which results from a defective aortic valve. The elongation with resulting tortuosity of the renal arteries which occurs, notwithstanding the increased growth and strength of the longitudinal layer of connective tissue, is analogous to the dilatation of the ventricle by the reflux current from the aorta, which the hypertrophy of the muscular walls cannot entirely prevent. I shall presently show you that in cases of granular kidney, not only are the walls of the *renal* arterioles hypertrophied, but that the arterioles in every tissue and organ throughout the body are found to have undergone a similar hypertrophic change, a discovery which I first published in the fifty-first volume of the *Medico-Chirurgical Transactions*.

On comparing the renal arterioles with those from other tissues, this remarkable difference is apparent—namely, that while the hypertrophy of the *muscular wall* occurs alike in the arterioles in all the tissues, the thickening of the *intima* is found, so far as I know, only in those of the kidney; but then, on the other hand, the hypertrophied arterioles in other tissues have their fibrous *tunica adventitia* thickened to about the same extent as the overgrowth of the tunica intima in the renal arterioles. In proof and illustration of this, I borrow from Dr. Dickinson, an able and quite disinterested witness, two illustrations—one representing a normal artery of the pia mater, and another a hypertrophied artery from the same tissue, in a case of granular kidney (fig. 32). It will be seen that the fibrous connective tissue *outside* the muscular layer of that artery is the exact counterpart of the connective tissue *within* the muscular coat of the renal artery. Dr. Dickinson's drawing has the appearance of a hypertrophied renal artery, in which the overgrowth of connective tissue is *external* to the muscular coat.

The probable explanation of this remarkable difference is, that the renal arteries, being embedded in the firm tissue of

the kidney do not need the support of the tunica adventitia which surrounds the arteries of the pia mater and those of most other tissues; and when, during the progress of granular degeneration of the kidneys, the renal arteries require to be braced and strengthened to resist the excessive force of the hypertrophied heart, this is accomplished by the overgrowth of the fibrous connective tissue of the intima.

Since I first discovered the thickening of the renal arterioles I have found this arterial hypertrophy in the advanced stage of every granular kidney that I have examined. It is not present in the earlier stages of the disease, but it comes on gradually, and proceeds *pari passu* with the structural changes within the uriniferous tubes. This thickening of the arterial walls from overgrowth of their normal tissue is quite distinct from that structural change which I shall

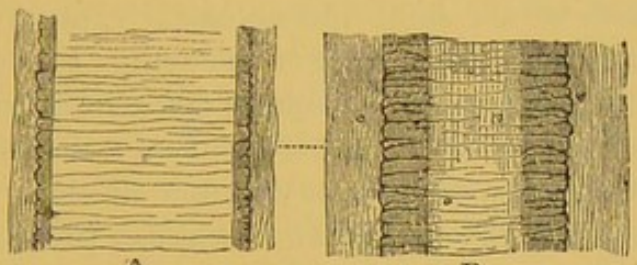

FIG. 32.—A. NORMAL ARTERY FROM THE PIA MATER. B. ARTERY FROM THE PIA MATER IN A CASE OF GRANULAR KIDNEY.
The muscular coat and the adventitia hypertrophied (Dickinson).—× 180.

hereafter show you, and describe as the waxy or lardaceous degeneration of the blood-vessels.

Here and there amongst the hypertrophied arterioles may be seen one with apparently atrophied muscular walls. This is only what might, *à priori*, have been expected, as a result of the general wasting which follows upon the destruction of the glandular epithelium. As the circulation through the disorganised portions of the kidney ceases, the muscular coat of some arterioles which had been hypertrophied gradually wastes and ultimately disappears, while the tougher connective tissue of the hypertrophied intima remains, and adds to the bulk of fibrous tissue, which is found in the extreme stage of granular degeneration of the kidney.

The walls of the Malpighian capillaries are usually thickened and opaque, their surface being sometimes smooth and

wax-like, more commonly finely granular (fig. 27, p. 693). The thickening of the capillary walls, together with that of the Malpighian capsule, tends to conceal the blood within the Malpighian capillaries, and gives the Malpighian bodies a dull grey appearance. The intertubular capillaries, and the veins into which they empty themselves, present no appearance of thickening.

Physiological Explanation of the Structural Changes in the Kidney.—In a paper published long since[1] I designated the disease which I am now describing *chronic desquamative nephritis*. The term 'chronic desquamative *disease*' is, I now think, preferable, since it implies no theory as to the inflammatory nature of the affection. The primary and essential structural changes consist in a desquamation, disintegration, and removal of the renal gland-cells; and the pathological process admits of the same physiological explanation as that which, in the last section, I gave of the acute desquamative disease. The changes in the grandular epithelium are the result of a modified cell-nutrition, consequent on a morbid condition of blood associated with gout or with one or other of those derangements of the general health to which I just now referred, as the usual antecedents of this form of renal degeneration. Gland-cells secreting abnormal products are themselves liable to become abnormal; and, when the process is long continued, the cells are apt to undergo decay and destruction. That appears to me to be the explanation of the disintegration and destruction of the renal epithelium. The wasting and contraction of the tubes, with thickening of their basement-membrane, are results of the destructive changes in the gland-cells. I cannot explain the replacement of the glandular epithelium by the delicate cells which are found lining some of the tubes, and which, apparently, are intimately associated with the conversion of the tubes into serous cysts.

This peculiar cell-formation is obviously the result of an active, though abnormal, intratubular growth, and is not to be accounted for by mechanical pressure from without.

The hypertrophy of the walls of the minute renal arteries is best explained by reference to the analogous results of apnœa.

[1] *Med.-Chir. Trans.*, vol. xxx.

The phenomena of apnœa have been fully explained in Chapter II. Now phenomena precisely analogous occur in the kidney, and probably in all glandular structures. There is the same intimate relation and interdependence between circulation and secretion as there is between circulation and respiration; in fact, the lung may be looked upon as a gas-secreting gland. When the secreting tissue of the kidney is partially destroyed, the gland is reduced to the condition of a lung receiving only a scanty supply of air, the working power of the gland is lessened, and it requires less blood. The minute renal arteries, by their contractile power, under the influence of the vaso-motor nerves, now regulate the blood-supply in accordance with the diminished requirements of the gland. This regulating contraction continues and increases, month after month, year after year; and the physiological result of this persistent over-action of the minute renal arteries is that their walls become hypertrophied. I shall show you, hereafter, that a similar hypertrophy of the renal arteries occurs in the advanced stages of other forms of chronic Bright's disease, but it is most constant and most conspicuous in the contracted granular kidney which we are now considering. The comparatively small amount of albumen in the urine, and its occasional absence in cases of contracted kidney, may be explained by the fact that, while there is but little compression of the intertubular capillaries by swollen tubes, and consequently but little passive engorgement of the Malpighian capillaries, the hypertrophied renal arteries, by their powerful contraction, prevent a too forcible influx of blood. Hence, too, it happens that hæmorrhage into the tubes, which is so common in acute Bright's disease, rarely occurs in this chronic form of the malady.

The Local and General Symptoms of Contracted Granular Kidney.—There are few diseases equally serious whose progress is so insidious as that of the disease which we are now considering; yet there are few maladies whose presence is indicated by more unequivocal signs, if only they be diligently and intelligently sought for.

One of the earliest symptoms in the majority of cases is *increased frequency of micturition*, and especially during the

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night. The more frequent call to empty the bladder may be a result of a more copious secretion of urine and consequent distension of the bladder, or it may be due to irritation of the bladder by some abnormal quality of the secretion. This symptom is sometimes absent, and it may result from other causes than renal disease. When present, it serves to direct attention to the urinary organs; and it is a symptom which should never be neglected.

Pain in the back is not a frequent or an important symptom. In many cases it is entirely absent; and often it is not spoken of until the patient's attention has been directed to the subject. When present, it is more frequently muscular than renal—an aching pain in fatigued and feeble lumbar muscles, often complained of by debilitated patients who have no renal disease. In numerous instances, a patient in the advanced stage of incurable degeneration of the kidney has said, 'I cannot understand how my kidneys can be diseased, since I have never had pain in them.'

Dyspepsia is frequently associated with this form of disease, sometimes, as a cause, sometimes as a consequence. You may often learn that a patient of strictly temperate habits has for months or years suffered from pain or uneasiness after food, flatulent distension of stomach and bowels, occasional nausea and vomiting, habitual looseness or irregularity of bowels, constipation and diarrhœa alternately. With this, there is often turbidity of the urine, which is high-coloured, excessively acid, and deposits urates abundantly. After a time, the urine, which had been scanty, becomes more copious, of pale colour, of low specific gravity, and is found to contain albumen and granular casts. In such a case, probably renal degeneration is a consequence of the long continued elimination of products of faulty digestion through the kidneys. I have seen this sequence of events so frequently that I have no doubt as to their causative relationship. Dyspeptic symptoms such as I have described, and consequent renal degeneration, are in some cases excited or greatly aggravated by habitual excess of alcohol—less frequently, perhaps, by excessive smoking of tobacco.

With regard to the etiological relation between chronic

dyspepsia and degeneration of the kidneys, I am glad to have the confirmatory testimony of so excellent a clinical observer as the late Dr. Murchison.[1] After quoting my observations on this subject in my published *Lectures on Bright's Disease*, Dr. Murchison says: 'Numerous cases which have come under my own observation, and which I have carefully watched, have satisfied me as to the strict accuracy of Dr. Johnson's description.'

In other cases, dyspepsia is a *consequence* of advanced renal degeneration. Urea and other urinary products are vicariously excreted by the mucous membrane of the stomach and bowels, in consequence of the defective action of disorganised kidneys. The gastric secretions are deranged; the digestive functions are disordered; and nausea, vomiting, and diarrhœa are amongst the results of this secondary renal dyspepsia.

The chronic degeneration of the kidney which we are now considering is often preceded and accompanied by such symptoms as the following: a gradual loss of energy, with emaciation to a variable extent; unusual fatigue after exertion, with tendency to rheumatic pains and cramp in the feeble and fatigued muscles; defective perspiration, with a dry and harsh state of the skin; a peculiar pallid or sallow complexion; and a watery condition of the conjunctiva, or of the connective tissue beneath it. Pallor is not a constant symptom; there is sometimes a florid complexion even in the advanced stages of this form of degeneration. The tongue is sometimes dry; at other times, moist and pale. There is often thirst, with loss of appetite and some of the dyspeptic symptoms before mentioned. Not unfrequently there is pain or a sense of weight in the head; sometimes a tendency to drowsiness, and occasional dimness of sight.

Whenever symptoms such as I have described are complained of, the urine should be carefully examined. I need not repeat what I said in the earlier part of this section of the indications afforded by the urine from the earliest to the latest stage of this form of renal degeneration.

Dropsy, as I have before told you, is not a prominent symptom in this form of disease. In the majority of cases it

[1] *Functional Derangements of the Liver*, 2nd ed. pp. 80–81.

is absent throughout the whole progress of the malady. Excluding those cases in which there is the complication of valvular disease of the heart, I found that, of thirty-three fatal cases of contracted kidney, there had been dropsy in only fourteen, the proportion being 42 per cent.; and in most of these fourteen cases the dropsy was only slight and partial, coming on towards the close of the illness.[1] The comparative infrequency of dropsy is explained by the free and often copious secretion of urine, which, as a rule, is not highly albuminous. There is not so great a deficiency of the normal blood-constituents in this form of disease as in most acute cases, and in other forms of chronic disease. The specific gravity of the blood-serum is less reduced, and the proportion of water to solids is less excessive. An excess of urea, however, is often found in the blood, especially in the later stages, when the secretion of urine becomes scanty.

Hypertrophy of the heart occurs in a large proportion of cases of contracted kidney when the disease has reached an advanced stage. In some cases, valvular disease, in others atheromatous and calcareous degeneration of the walls of the large arteries, suffices to explain the hypertrophy; but in other cases, as Dr. Bright pointed out many years ago, there is no such obvious explanation of the hypertrophy, which affects chiefly the left ventricle; and he suggested, as a probable explanation, that 'the altered quality of blood so affects the minute and capillary circulation as to render greater action (of the left ventricle) necessary to force the blood through the distant sub-divisions of the vascular system.'[2] Some years since, it occurred to me that the hypertrophy of the left ventricle of the heart in cases of contracted kidney might be a result of increased contraction of the small arteries throughout the body, this contraction being excited by the abnormal quality of the blood, as the systemic arterioles are excited to contract when the blood passes through the lungs without being duly aërated (see Chapter II.). I went on to argue that, if this were so, we should find evidence of the fact in the existence of

[1] See a paper 'On the Forms and Stages of Bright's Disease,' *Med.-Chir Trans.*, vol. xlii.

[2] *Guy's Hospital Reports*, vol. i.

hypertrophy of the muscular walls of the minute arteries in various tissues. And I have found this hypertrophy not only in the arteries of the kidney, but also in those of the skin, the intestines, the muscles, and the pia mater. It probably exists in the arteries of other tissues which I have not examined.[1]

The probable explanation of the hypertrophied left ventricle in the advanced stage of contracted kidney, then, appears to be this. In consequence of degeneration of the kidney, the blood is morbidly changed. It contains urinary excreta, and it is deficient in some of its own normal constituents. It is, therefore, more or less unsuited to nourish the tissues, and probably more or less noxious to them. The minute arteries throughout the body (under the influence of the vaso-motor nerves) resist the passage of this abnormal blood, and in consequence the left ventricle beats with increased force to carry on the circulation. The result of this physiological antagonism of forces is, that, while the balance of the circulation is maintained, the muscular walls of the arteries, and those of the left ventricle of the heart, become simultaneously hypertrophied.

Some pathologists, apparently not realising the fact, that, in health as in disease, the balance of the circulation is constantly adjusted and maintained by the harmonious co-operation of the propelling heart and the regulating arterioles, have suggested that the hypertrophy of the muscular coat of the arterioles, which they admit to be a fact, is a result of their forcible efforts to aid the heart in driving on the blood.

This theory is precisely that which I put forth in the year 1850 to explain the hypertrophy of the renal arterioles, and which I abandoned in obedience to the teaching of Bernard, Brown-Séquard, and all modern physiologists. If this explanation of the arterial hypertrophy is correct, it is certain that the generally accepted doctrine with respect to the function of the muscular arterioles is entirely erroneous; and the account of the forces concerned in carrying on and regulating the circulation of the blood, which is to be found in every text-book of physiology, will have to be re-written.

The physiological antagonism between the propelling heart

[1] See my paper with illustrations, *Med.-Chir. Trans.*, vol. li.

and the opposing and regulating arterioles, which has for its object to maintain the balance of the circulation, is analogous to the many instances of muscular antagonism with which we are familiar—*e.g.* that of the circular and the radiating fibres of the iris; of the sphincter vesicæ and the detrusor urinæ; of the muscles of inspiration and expiration, &c.; the result of such muscular antagonism being, by mutual adjustment, to secure harmonious physiological co-operation.

Now I wish to direct your attention to the important bearing of the physical signs of hypertrophy of the left ventricle upon diagnosis and prognosis. The chief indications of the cardiac hypertrophy are the beating of the apex below and external to its normal position, with a strong heaving impulse; with this there is reduplication of the first sound over the base, and accentuation of the second sound over the aortic valves,[1] a full resisting pulse and high arterial tension. This combination of physical signs indicates not only that the disease is chronic, but also that it is in an advanced stage, and, therefore, that the prognosis is unfavourable.

Although the systemic vessels are the primary and the main seat of the impeded circulation, yet evidence of obstruction to the *pulmonary* circulation is afforded by the frequent coincidence of granular degeneration of the kidneys with old clots in the pulmonary arteries, a fact long since recorded and commented on by Sir James Paget.[2] The impeded pulmonary circulation may be a direct result of contraction of the arterioles in the lung, or it may be consequent on insufficient compensatory hypertrophy of the left ventricle, with resulting retrograde hindrance of the pulmonary circulation.

Both the investing and the lining membrane of the heart are liable to inflammatory changes, consequent on the blood contamination which occurs during the progress of the renal degeneration.

These complications will be indicated by the local, general, and physical signs of pericarditis or endocarditis, or, it may be, of both combined.

Other *serous membranes* sometimes become inflamed—

[1] For the full explanation of these signs, see *ante*, Chapter XXXVI.

[2] *Med.-Chir. Trans.*, vol. xxviii. p. 367.

the pleura more frequently than the peritoneum. Œdema of the lungs and bronchitis are frequent complications. Pneumonia is comparatively rare, but it does sometimes occur.

Hæmorrhage.—In the advanced stages of the disease, hæmorrhage from different mucous surfaces is a common and often a troublesome and alarming symptom. Epistaxis is the most common form of hæmorrhage; but I have seen it occur from the stomach and intestines, from the lungs, the bladder, and from the uterus in the form of menorrhagia. Amenorrhœa is, however, according to my experience, a more frequent result of advanced Bright's disease than menorrhagia.

Cerebral Hæmorrhage.—The most serious, and by no means the least frequent, form of hæmorrhage is that which takes place within the cranium. In a large proportion, probably half, of the fatal cases of sanguineous apoplexy, the kidneys are found more or less diseased; and the granular degeneration which we are now discussing, is the form of disease which is most frequently associated with cerebral hæmorrhage. The explanation of this common, and too often fatal, accident is not difficult. The minute cerebral arteries resist the passage of the abnormal blood, while the hypertrophied left ventricle is forcibly driving it onwards. Meanwhile, the walls of some of the intermediate arteries undergo atheromatous degeneration —partly, perhaps, in consequence of the circulation of morbid blood, partly as a result of the unusual strain and pressure to which they are subjected. At length, in the struggle between the propelling left ventricle and the resisting muscular arterioles, a brittle artery gives way, and a fatal hæmorrhage occurs.

Impairment of vision is one of the most serious results of granular contraction of the kidney. It occurs in two distinct forms: 1. The impairment of vision may be so sudden in its onset, that in a few minutes or hours there is complete blindness, which usually passes away as suddenly as it came. The attacks of sudden and transient blindness may recur again and again. In these cases, ophthalmoscopic examination discovers no structural change within the eye. This form of amblyopia is believed to be of uræmic origin, and is designated uræmic amaurosis. It is usually associated with other symp-

toms of uræmia, and I shall presently have something more to say of its pathology. 2. In the second form of impaired vision, the dimness of sight usually comes on more slowly, and is more durable. One eye alone may be affected, but both are often implicated simultaneously or in quick succession. The ophthalmoscope reveals peculiar structural changes in the eye, the result of what is called *retinitis albuminurica*. The ophthalmoscopic appearances in the different stages of albuminuric retinitis are well represented in Liebreich's *Atlas of Ophthalmoscopy*. The most characteristic appearances are—a broad glistening white mound surrounding the optic disc, the result of sclerosis of the optic nerve fibres, and fatty degeneration of the connective-tissue elements. The extreme margin of the white mound is broken up into small irregular patches, which assume, in the neighbourhood of the yellow spot, a peculiar stellate arrangement. The retinal arteries are diminished both in size and number, while the veins are dilated and tortuous. Blood extravasations, varying in number and in size, sometimes both numerous and large, occur here and there, chiefly in the internal layers of the retina, but sometimes in the external layers, or between the retina and the choroid. The coats of the blood-vessels are sometimes found in a state of sclerosis or fatty degeneration. These structural changes appear to be of an inflammatory and degenerative character. They are associated more commonly with the contracted kidney than with other forms of chronic Bright's disease. So characteristic are the appearances in the retina, and so insidious is the disease in the kidney, that an ophthalmoscopic examination for determining the cause of dimness of sight has, in many instances, led to the discovery of a previously unsuspected renal disease. It may be well to mention here that the two forms of impaired vision which I have described may occur together or in succession in the same subject. Uræmic amaurosis may, in time, be succeeded by albuminuric retinitis; and the dimness of vision which results from the latter may be temporarily much increased by uræmic amaurosis. The hæmorrhage into the retina may be explained partly by the injecting force of the hypertrophied ventricle, partly by degeneration of the walls

of the retinal vessels, and partly by venous engorgement consequent on pressure upon the veins by inflammatory exudation.

Cerebral Symptoms.—In the advanced stages of contracted kidney, various forms of nervous disorder occur with so great frequency, that the disease may be said to have a natural tendency to terminate with symptoms referable to the brain. These nervous symptoms are very variable. In some cases, epileptiform convulsions or profound coma may occur suddenly, without premonitory symptoms. Much more frequently these formidable symptoms are preceded for a variable period by other indications of brain disturbance. Amongst the commonest of these are more or less severe and constant headache, sudden transient vertigo, equally sudden and transient loss of sight or hearing, temporary inability to speak, or the speech for a time is imperfect and stammering; numbness or neuralgic pains, cramps, chorea-like twitchings, or transient loss of power, may occur in one arm or leg, or in both the arm and leg on one side; there may be confusion of thought, impairment of memory, and an indescribable nervous dread, with a feeling of utter prostration; after one or more of these symptoms have continued for a variable period, or recurred more or less frequently, the secretion of urine being usually scanty, and vomiting of frequent occurrence, the patient perhaps becomes drowsy, with more or less delirium; the tongue is brown and dry; the breath has a most characteristic sour and fœtid odour; the drowsiness gradually increases and deepens into coma; the pupils being natural or equally dilated, and the breathing more hurried than in ordinary cases of sanguineous apoplexy; and so death occurs either with or without convulsions. In some cases, a single attack of violent convulsion is immediately fatal; in others, the convulsions recur again and again for several hours before the fatal termination. The brain after death is usually found extremely pale and anæmic, with some serous effusion beneath the arachnoid and into the ventricles. These are cases of so-called 'serous apoplexy'; but the amount of serous effusion is insufficient to compress the brain, and so to explain the symptoms.

Theory of Uræmia.—In attempting to explain these nervous symptoms, I assume it to be indisputable that they are the result of the blood being deteriorated, partly by diminution of its normal constituents, but chiefly by retention and accumulation of urinary materials. There are two ways in which it is probable that the brain and its functions may be injuriously affected by this blood-deterioration: First, the cerebral tissues, fed with poor and poisoned blood, may have their nutrition impaired, and may in various parts undergo structural changes analogous to those which are often demonstrable in the texture of the retina. Second, it is probable that some of the cerebral symptoms, more especially those which come on and pass away suddenly, are directly due to temporary interruptions or hindrances of the circulation through certain regions of the brain, consequent on excessive contraction of the minute arteries. In my lecture on the Pathology of Epilepsy (Chapter IX.), I adduced many facts and arguments in support of the theory that the immediate cause of an ordinary epileptic convulsion is sudden and extreme anæmia of the brain, the result of excessive contraction of the minute cerebral arteries.

Our increasing experience of the various forms of nervous disorder which may result from so purely mechanical a cause as embolism, in vessels of various sizes and in different regions of the brain, gives additional support and probability to the theory, that many of the cerebral symptoms resulting from uræmia may be explained by a defective blood-supply to certain parts of the brain, consequent on arterial contraction. An arrest of the circulation through a portion of the brain involves immediate suspension of function in that part, with perhaps a disorderly action in subordinate and correlated parts. Thus, amongst other symptoms of nervous disorder, maniacal delirium and acute chorea have sometimes been found associated with, and probably have been directly caused by, mechanical plugging of minute cerebral vessels; the plugging being the result of embolic particles of fibrine detached from the so-called warty vegetations on a damaged mitral or aortic valve. Again, sudden and complete blindness in one eye may result from embolism of the arteria centralis retinæ; partial

and patchy blindness from embolism in one of its branches. It is, therefore, highly probable that uræmic vertigo, amaurosis, delirium, convulsions, and even coma, may in some cases be explained by partial or general cerebral anæmia, the result of excessive arterial contraction, excited by the presence of impure blood, acting through the vaso-motor nerves upon the blood-vessels. I do not ask you to adopt this as a complete and final explanation of the phenomena, but suggest it as a theory to be tested by the results of further observation and research.

Let me add that in some cases, notwithstanding the scantiness and ultimately the almost complete suppression of urine, uræmic symptoms are almost entirely wanting, and consciousness remains until death occurs from exhaustion. In some at least of these cases the uræmic symptoms are probably prevented by the occurrence of incessant vomiting or purging, which, while it rapidly exhausts the patient, favours the escape of noxious impurities from the blood. The cessation of the discharges is sometimes quickly followed by symptoms of uræmia.

Nervous Dyspnœa.—A common and very distressing symptom in the advanced stages of the disease is a peculiar form of dyspnœa. I am not now referring to the persistent dyspnœa which results from the œdema of the lungs, from hydrothorax, or hydropericardium, but to dyspnœa coming on in paroxysms, and especially at night. In some cases the attack resembles asthma, and there are loud sibilant sounds, apparently the result of bronchial spasm; while in other cases the heart's action is rapid and feeble, and the breathing hurried and laborious, with loud puerile respiration over the lungs. There is evidently no want of moving air in the lungs, and the disturbed circulation and breathing appear to result from some morbid influence of the poisoned blood upon the nervous centres. This distressing form of dyspnœa, which recurs in paroxysms night after night, is, in fact, a result of uræmia.

Disease of the Liver.—In a large proportion of fatal cases of contracted kidney, the liver is found more or less diseased, sometimes enlarged and indurated or fatty, more commonly cirrhosed and contracted. Alcoholic excess may, and often

does, excite at the same time cirrhosis of the liver and granular contraction of the kidney. With the cirrhosed liver there is often ascites. When ascites exist without anasarca, or remains after the removal of anasarca, and so forms the prominent dropsical symptom, serious disorganisation of the liver may always be suspected.

Inaction of the Skin.—One of the most constant conditions in the advanced stage of granular kidney is a dry and inactive state of the skin; so that the hot air or vapour bath often fails to excite perspiration. The probable explanation of this state of skin is that the sweat-glands have undergone structural change, in consequence of the long-continued vicarious excretion of urinary products. This is probable from analogy. We have seen[1] that the excretion of biliary matters by the kidney excites pathological changes in the renal gland-cells, and it is probable that the elimination of urinary materials by the cutaneous glands would have a like injurious effect upon them. A careful microscopical examination of the cutaneous structures can alone determine whether this theory is in accordance with the facts.

Diagnosis.—In addition to what I have said of the symptoms and progress of the disease, I have yet some hints to give you on the subject of diagnosis. The state of uræmic stupor or drowsiness, with a dry tongue and sordes on the teeth, may be mistaken for typhus or enteric fever. The difficulty of diagnosis is increased by the fact, that in some cases of typhus and enteric fever, when there is much cerebral oppression, the urine is often scanty and albuminous, and it sometimes contains granular casts. A close attention to the entire history of the case, and a careful examination of the urine, will seldom leave you in doubt. The specific fever eruption, when present, is decisive. The thermometer will assist you. The temperature is higher in fever than in uncomplicated uræmic poisoning. Bear in mind that a patient with chronic renal disease may, in addition, have a specific fever—a complication which is usually fatal. With regard to indications afforded by the urine, remember this, that although during the progress of typhus or typhoid fever there may be

[1] *Ante*, pp. 662–3.

an acute and transient disintegration of the renal gland-cells, as indicated by the appearance of granular casts, not easily to be distinguished from those which occur in cases of chronic desquamative disease—yet there is this difference, that whereas in the advanced stages of chronic desquamative disease the urine is pale and of low specific gravity, the albuminous urine of fever is usually of deep colour and of rather high specific gravity. It is important to bear in mind that granular casts, with albumen, may appear temporarily in the urine as a result of other blood-poisons than those of typhus and enteric fever. I have seen them in cases of pneumonia, erysipelas, and pyæmia. Once, in a case of pyæmia, I found granular and large hyaline cysts exactly like those represented in fig. 20 (p. 685), but the urine was of deep brown colour and of normal specific gravity; and after death, which resulted from pyæmic abscesses in various parts, the only disease found in the kidney was a recent result of pyæmia. You see, then, that, although the observation of the various forms of tube-casts is of great practical value as an aid to diagnosis and prognosis, yet a too exclusive reliance upon this microscopic evidence may mislead you. When, after a careful inquiry into the history of a case, a doubt exists as to renal disease being recent or of long standing, the evidence of hypertrophy of the left ventricle of the heart without valvular disease, but with a full and firm radial pulse, points to chronic disease in an advanced stage.

I have seen several cases of subacute renal disease occurring in men about middle age as a result of overwork and anxiety, in which it was difficult to decide between acute and chronic disease. I have preserved the urinary sediment from three of these cases, and although the first case occurred nearly fifteen years ago, the tube-casts are as well seen as when the specimen was recent. You may see these specimens under the microscopes on the table, and, having carefully inspected them, you may recognise their like when you meet with them in practice. One case was that of a solicitor aged 40, another a merchant aged 56, another a clergyman aged 45. The symptoms and the condition of the urine were alike in all. There was great prostration, vomiting, bleeding

from the nose, and in one case from the gums, no dropsy, ultimately a typhoid condition, and unconsciousness shortly before death. The urine was blood-tinged, the specific gravity from 1009 to 1017, moderately albuminous. A rather copious sediment was composed of dark granular and large hyaline casts, with scattered blood-discs. Some of the granular casts had a blood tinge, and it is probable that they were in part composed of disintegrated blood. After death, in the only case examined, the kidneys were found somewhat enlarged, soft, and congested. Some tubes were injected with extravasated blood, and others, opaque, with desquamated and disintegrated epithelium. In cases of this kind, although the prognosis is very unfavourable—in fact, all the cases that I have seen have died—yet the disease is not so inevitably fatal as the chronic desquamative disease in an advanced stage, and therefore it is important to distinguish between them.

Prognosis.—On the subject of prognosis I have but little to add to what I have already said. Chronic desquamative disease of the kidney, as a rule, tends gradually to a fatal termination. The rate of progress varies much in different cases and at different periods of the same case. You will remember what I said as to the evidence to be derived from the amount as well as the character of the sediment in the urine (pp. 634–5). The most trustworthy prognostic indications are to be obtained by comparing the state of the urine with the general symptoms. When with a condition of urine indicating advanced degeneration of the kidney, there is evidence of hypertrophy of the left ventricle, with high arterial tension and an unperspiring skin; when with a diminishing secretion of urine, or even without a marked decrease, symptoms of uræmia begin to appear, the disease is generally not far from its fatal termination. You cannot, however, be too cautious in giving a prognosis. The symptoms of chronic renal disease are sometimes much aggravated for a time by some imprudence in diet, by fatigue or anxiety, or exposure to cold. The patient may apparently be on the verge of uræmic coma, or he may have a fit of convulsions, yet, under appropriate treatment, these formidable results of his indiscretion, or his misfortune, may pass away,

and, in a few days, he may be apparently no worse than he was before the occurrence of this temporary disturbance. The uræmic symptoms which are not traceable to an obvious external exciting cause are, as a rule, more serious and intractable than those which result from influences capable, to some extent, of being removed, avoided, or counteracted.

APPENDIX TO SECTION V.

In order not to interrupt inconveniently the description of the pathological changes in the kidney and in other tissues and organs, I have reserved for an appendix any reference to the pathological doctrines of Sir William Gull and Dr. Sutton. I have criticised their theories at some length in vol. xxviii. of the *Pathological Transactions*, and I should not have deemed it necessary or desirable to refer to them here, but for the fact that Dr. Sutton, in his recently published *Lectures on Medical Pathology*[1] restates the main points of their conjoint theory, together with their reasons for dissenting from my pathology. Referring to the *Pathological Transactions* for a detailed criticism of the 'hyalin-fibroid theory,' I will here briefly restate a few facts which have relation to the subject.

Both Sir William Gull and Dr. Sutton, after the reading of their paper at the Medico-Chirurgical Society, recognised in a specimen which I showed them, the appearances which they had described as pathological; the specimen consisting of arteries from the normal pia mater of a sheep, the tunica adventitia of which had been distended and rendered hyaline by immersion in glycerine. The authors stated in their paper[2] that all their specimens had been mounted in a mixture of glycerine and camphor water before they were examined.

The effect of glycerine is not only to cause hyaline distension of the adventitia, but also to render indistinct the muscular nuclei of the middle coat; and we have a very interesting proof of this in one of the illustrations appended to the paper of Sir William Gull and Dr. Sutton. Fig. 7 in

[1] Page 175 *et seq.* [2] *Med.-Chir. Trans.*, vol. lv.

Plate VI. represents a transverse section of a thickened renal artery, which is described as 'minute artery of the kidney greatly thickened by hyalin-fibroid changes in the outer layer of the vessel;' and in the text of the paper the following reference is made to the drawing: 'The muscular nuclei were indistinct, and many of them were so altered as hardly to be recognisable. External to the muscular nuclei, there was a quantity of hyalin-fibroid substance, and the layer formed by this material was much thicker than the muscular layer (Plate VI. fig. 7).'

Again, they say (at p. 278, par. 4) 'where the kidney disease was far advanced, hyalin-fibroid changes were seen in the minute renal arteries precisely similar to those observed in the arterioles of the pia mater and of other parts of the body.' It is evident, therefore, that they believe the 'outer' layer in the renal artery to be the counterpart of the so-called 'hyalin-fibroid' layer in the arteries of the pia mater; the fact being that this layer in the pia mater is external to the muscular layer, and is composed of the distended tunica adventitia; while in the renal artery the adventitia is usually inconspicuous, and that which they mistake for it is the hypertrophied circular layer of muscular fibres.

This illustration, with the author's description, completely explains their disbelief in the existence of arterial muscular hypertrophy, and their confirmed belief in the hyalin-fibroid theory. The inner layer in the specimen referred to is, quite obviously, the thickened intima, in which, as it is not muscular, it is not surprising that 'the muscular nuclei were indistinct.' On the other hand, the outer layer is the much hypertrophied muscular coat, but distended, and its nuclei rendered indistinct by the glycerine.

It is a very remarkable circumstance that while Sir William Gull and Dr. Sutton consider the changes which they describe as 'allied with the conditions of old age,' they make no reference to the well-known results of senile degeneration of the blood-vessels which I have described in Chapter XV. (p. 304).

The theory of 'arterio-capillary fibrosis' has been very ably criticised by Dr. Bryan Charles Waller in his work on *Interstitial Nephritis*.

Although, for reasons which have been fully stated elsewhere, I am unable to accept the pathological doctrines of Sir William Gull and Dr. Sutton, I feel personally indebted to them for having done much to direct attention to the vascular changes which are associated with the granular contracted kidney.

SECTION VI.

CHRONIC BRIGHT'S DISEASE WITH A LARGE WHITE KIDNEY.

General History of the Disease: its Causes and Progress—Condition of Urine in different Stages—Various Forms of Tube-Casts, and their Significance—Morbid Anatomy and Pathology of the Kidney in the three Stages of—1. Simple Enlargement—2. Granular Fatty Degeneration—3. Atrophy, with Coarse Granulations on the Surface—Symptoms—Dropsy—Pulmonary Complications—Inflammation of the Serous Membranes—Endocarditis—Dyspepsia Vomiting Diarrhœa Hypertrophy of the Heart Cerebral Symptoms Hæmorrhage, &c. Defect of Vision Hæmorrhage from Mucous Membranes—Diagnosis—Prognosis—Simple Fat Kidney, or General Fatty Infiltration of the Kidney—History—Microscopic Characters of the Kidney—Pathology and Clinical History—Points of Difference between it and the Granular Fat Kidney.

I now propose to give you the pathological and clinical history of those cases of chronic Bright's disease in which the kidneys are found, after death, always more or less anæmic and pale, usually enlarged, soft in consistence, and smooth on the surface, but sometimes contracted, indurated, and coarsely granular.

A large white smooth kidney is often a sequel of an acute inflammatory attack. Acute Bright's disease, the result, it may be, of scarlet fever or of exposure to cold, is imperfectly recovered from. The dropsy passes away; the patient regains his strength and his colour; the urine is normal in quantity, appearance, and specific gravity; but it continues to be more or less albuminous. The albuminuria which thus continues after acute Bright's disease may remain for many months, and even for a number of years, before the appearance of symptoms consequent on chronic and incurable degeneration of the kidney. At length there is, perhaps, a return of dropsy, or the patient is cut off by some of the results of uræmia,

either of an inflammatory or neurotic character. After death, the kidneys may present one of three distinct appearances: 1. They may be large, almost uniformly white, and smooth on the surface. This appearance is admirably represented in Dr. Bright's fourth Plate, figs. 1 and 2. I call this simply a 'large white kidney.' 2. The kidney may present the same general appearance, but with this addition, that the cortical surface and the surface of a section of the cortex are, to use the words of Dr. Bright, 'interspersed with numerous small yellowish opaque specks.' This appearance is represented in fig. 3 of Dr. Bright's third plate. I shall presently show you that these yellow opaque specks are spots of fatty degeneration; and I call this a 'granular fat kidney,' or a 'large white kidney with fatty granulations.' 3. The cortical portion of the kidney may be found more or less atrophied, with an uneven granular surface; the yellow specks of fatty degeneration being in some cases still visible on the surface and on section.

The various appearances which I have described are results of successive stages of the same pathological process; in the majority of cases, but not always, following upon an acute onset. A kidney, which has contracted and become granular, after having been enlarged, differs from the contracted granular kidney which I described in my last lecture, in being of paler colour, of firmer texture, and more coarsely granular on the surface. The microscopic appearances of the kidney, the history, and the symptoms, also differ greatly, as we shall presently see.

There is a class of cases in which, with a clinical history different from that of the cases to which I have just now referred, the kidneys are found, after death, pale and wax-like in colour and consistency, usually enlarged and smooth, but sometimes contracted and granular. This is the 'lardaceous degeneration' of the kidney. The subjects of this form of disease have usually been strumous or otherwise cachectic before the onset of the renal disease, which, in the great majority of cases, begins as an insidious chronic malady. A similar degeneration of the liver and spleen is often associated with that of the kidney. In my next lecture, I shall discuss

the clinical and pathological history of this lardaceous degeneration of the kidney.

I now proceed to give you a more detailed account of those cases which are associated with a large white kidney, with or without the fatty granulations, with or without subsequent contraction and coarse granulations on the surface.

In a large proportion of cases, the commencement of the disease dates from an attack of acute Bright's disease resulting from exposure to cold, scarlet fever, diphtheria, typhus or enteric fever, or other zymotic disease. In several instances, I have traced the disease back to an attack of tropical malarious fever; I have seen it as a sequela of ague in this country; and I have known it follow upon an attack of dysentery. The acute disease may or may not have been associated with dropsy. The dropsy, if present, usually passes away, and for a time the only evidence of incomplete recovery is to be found in the condition of the urine. In some few cases, the disease comes on as an insidious chronic malady, and it is impossible to determine either the date of its commencement or its probable cause.

It is not unfrequently a result of, or, to say the least, it is often associated with, an excessive consumption of food and of alcoholic stimulants, with consequent dyspepsia. In cases thus originating, the approach of the disease is gradual, insidious, and often unsuspected until it has reached an advanced stage. The disease occurs at all ages, from infancy to extreme old age. I have seen it fatal at the age of seventy-five. Beyond childhood, it is more common in males than in females —probably because males are more exposed to cold, and are more intemperate, than the other sex.

This form of disease has, as a rule, a more protracted course than any other form of chronic Bright's disease; and for a period of years it may be unattended by any obvious symptoms apart from the indications afforded by the urine. A patient recovers from the dropsy and other symptoms of acute Bright's disease; he feels and declares himself to be quite well; but the urine shows that recovery has not been complete. The urine may for a long time be normal in quantity, colour, and specific gravity; but it contains albumen,

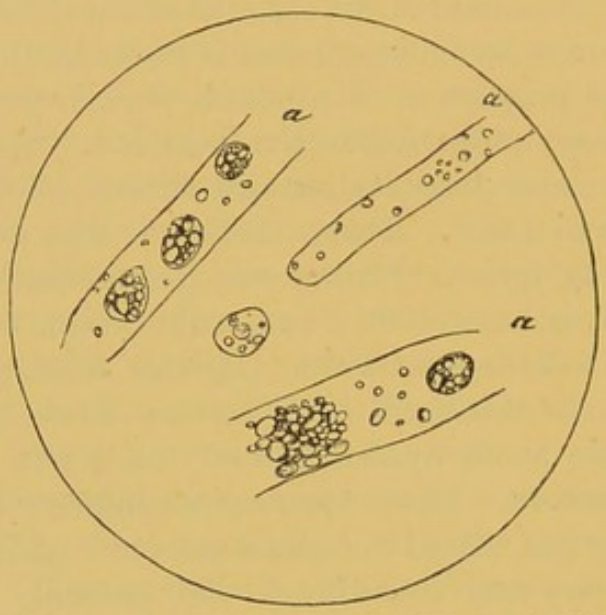
varying in amount from a mere opalescence, with heat and acid, to a dense and copious precipitate; the albumen being usually more abundant after food and exercise. The urine, placed in a conical glass, may remain quite clear, and show no appearance of tube-casts or renal epithelium; or it may either occasionally or constantly deposit a light cloud, in which are found small hyaline casts, in some of which a few oil-globules may perhaps be seen, while others contain an epithelial cell or two, or some fragments of cells. This condition of urine may continue with little or no change for many months, and even for several years, before there is any indication that the general health is suffering from the state of the kidneys. The scarlet fever poison and its products, which originally excited the acute renal disease, have passed away, a certain amount of injury having been inflicted upon the kidney, from which it has not recovered; but there is, up to a certain period, no progressive disease of the gland. The convoluted tubes have been left enlarged; the cortex probably has a somewhat anæmic and mottled appearance, resulting from compression of the intertubular capillary network by swollen tubes. This compression of the intertubular capillaries to some extent impedes the circulation; there is consequent engorgement of the Malpighian capillaries, and hence a transudation of serum into the tubes, which, mingling with the urine, renders it albuminous. This explanation of the albuminuria will readily be understood by a reference to fig. 6 (p. 627). Its mode of production is analogous to that of the albuminuria and the hæmaturia which Dr. George Robinson first, and Frerichs and others since, have produced in rabbits by putting a ligature on the renal vein. In addition to this mechanical hindrance to the passage of blood, it is not improbable that, as a result of acute Bright's disease, the Malpighian capillaries may undergo some physical change, which for a time, or even permanently, favours the transudation of serum through their walls. After a variable period of months or even years, the albuminuria may at length cease, and the cure is complete; but persistent albuminuria, or the state of kidney which gives rise to it, involves, sooner or later, serious structural changes in the kidney, and, as a result of these changes, some of the

secondary results of renal degeneration. The state of things which results from persistent albuminuria following the incomplete cure of acute Bright's disease, is analogous to that which occurs when acute endocarditis has passed away, but has left a thickened and defective valve. For a time, hypertrophy of the heart's walls compensates for the imperfect valve, and the circulation appears to be unimpeded ; but there is a limit to this conservative process. At length, the muscular tissue of the heart ceases to respond to the increasing demands which are made upon it, and it undergoes degenerative changes, the circulation flags, and the serious trouble begins. In like manner, for an indefinite time, the urine, although more or less albuminous, is freely secreted, and contains its due proportion of solids to liquid, there being no indication or symptom of defective secretion. Sooner or later, however, the urine loses its natural sherry colour, and gradually becomes paler. The secretion is less copious. The specific gravity varies. With a scanty secretion it may be as high as 1020, or even 1030 ; but, with a more copious secretion, it may be as low as 1010. By this time, probably, the sediment in the urine will have become more copious ; and it may contain small hyaline and oily casts and cells in considerable numbers. These appearances indicate that, in some of the uriniferous tubes, the gland-cells are undergoing fatty transformation ; and the kidney will present, after death, those small yellow spots of fatty degeneration to which I just now referred.

The kidney has now passed from the first stage of a simple large white kidney to the second stage—namely, that of a granular fat kidney ; and the disease is usually fatal in this stage ; but it may pass on to the third stage of contraction and atrophy, with coarse granulations on the surface, the small yellow fat granulations being still visible. This stage is indicated by the appearance of large granular and large hyaline casts in the urine. The large size of these casts shows that they were moulded in tubes which have been deprived of their lining of gland-cells. The destruction of the gland-cells is followed by atrophy of the gland ; and the amount of urinary sediment, containing the large granular and large hyaline casts,

is an index of the rate at which the disease is making progress. The granular casts in part consist of disintegrated epithelium —in part of the disintegrated fibrinous material of which the hayaline casts are composed.

I have in many instances traced the transition from acute to chronic disease; and in chronic cases I have traced, by the microscopic appearances and other physical changes in the urine, the successive stages of a large white smooth kidney, a granular fat kidney, and, lastly, a contracted and coarsely granular kidney. This sequence of events occurred in a case the later stages of which are illustrated by figs. 33, 34, and 35.



TUBE-CASTS AT THREE SUCCESSIVE PERIODS OF THE SAME CASE.

FIG. 33.—PERIOD OF FATTY ENLARGEMENT OF THE KIDNEY.

a a a. oily casts and cells.

The disease began in a man aged 23, as an acute attack, in October 1846. It passed into a chronic stage with oily casts and cells, which continued for a period of nine years. Then the oily casts and cells were mixed with, and afterwards replaced by, large granular and large hyaline casts. Death occurred from uræmia in October 1856, ten years after the onset of the disease. The kidneys were pale, had many yellow fat granulations in the cortex, and were much atrophied, their combined weight being only $7\frac{3}{4}$ ounces. Their appearance is represented by a chromo-lithograph, illustrating a paper of mine in the forty-second volume of the *Medico-Chirurgical*

Transactions. The microscopic specimens from which figs. 33, 34, and 35 are taken, retain their characteristic appearances after an interval of seventeen years; and they are placed for your inspection beneath the microscopes on the table.

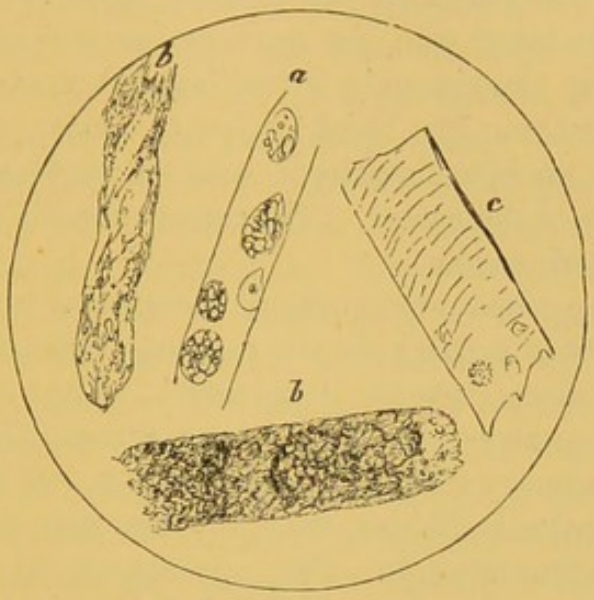


FIG. 34.—COMMENCING ATROPHY AND CONTRACTION.
a. oily cast. *b b*. granular casts. *c*. large hyaline cast.

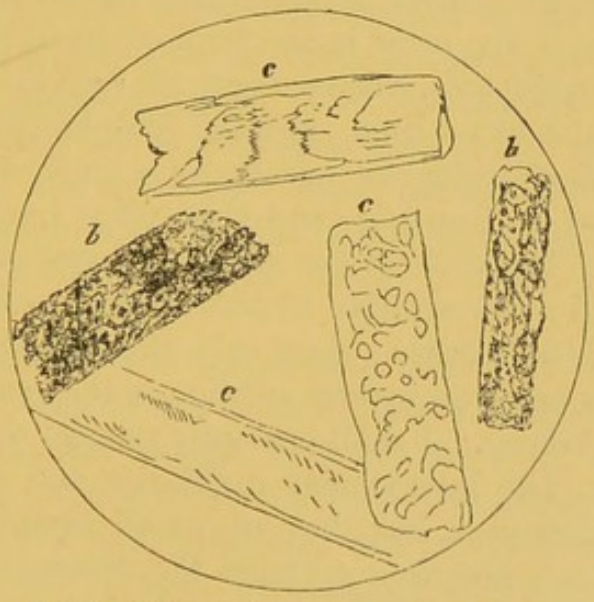


FIG. 35.—ADVANCED ATROPHY AND CONTRACTION.
b b. granular casts. *c c c*. large hyaline casts.—× 200.

Morbid Anatomy and Pathology of the Kidney.—When death has occurred before oily casts and cells have appeared in the urine, the kidneys will be found in the first stage of degenera-

tion—that is, large, white, and smooth on the surface. The weight of each kidney may be from seven to ten ounces. The cortical portion is increased in thickness, and appears more or less anæmic ; while the medullary cones are pink and vascular. The lobular markings on the surface, which are in fact the radicles of the renal vein, are more or less obliterated, so that perhaps, there remain only a few isolated vascular patches, as represented in Dr. Bright's fourth plate. Rarely some hæmorrhagic spots are scattered over the surface or through the substance of the cortex. The capsule readily peels off, and leaves a smooth surface.

On a microscopic examination, the greater number of the convoluted tubes present no other change than that of being larger and more opaque than usual. The epithelium is unusually granular and opaque, and apparently contains a more than ordinary amount of solid matter ; while the central axis of the tube is lighter than the margins, and free from deposit or accumulation. The appearance of the tubes is precisely the same as that which I described in cases of acute Bright's disease unassociated with epithelial desquamation.[1] In some tubes, the central canal contains the fibrinous material which appears in the urine in the form of small hyaline casts. The hæmorrhagic spots, when present, are seen to be convoluted tubes filled with blood from ruptured Malpighian capillaries.[2] The Malpighian capillaries usually have their walls more or less thickened and opaque, and they often have a wax-like appearance. The walls of the minute arteries are sometimes hypertrophied ; but arterial hypertrophy is not constant in this stage of the disease. As a rule, the hypertrophy of the arterial walls bears an inverse relation to the general enlargement of the kidney. I shall presently suggest an explanation of this fact. The intertubular capillaries and veins present no structural change, but they are much compressed by the enlarged tubes ; and this explains the disappearance of the lobular markings from the surface of the kidney.

The enlargement of the cortical portion of the kidney is mainly a result of a kind of hypertrophy of the gland. Many of the tubes are certainly enlarged, and their epithelium is

[1] See *ante*, fig. 17, p. 665. [2] See *ante*, fig. 16, p. 661.

unusually opaque and bulky. The transverse sections of many tubes have twice the normal diameter, and these large tubes must contain an increased number of secreting cells. This increase in the diameter of the secreting tubes is analogous to the increased thickness of fibre in a growing and hypertrophied muscle. When one kidney has been destroyed by disease or accident, as by the impaction of a calculus in the ureter, the other does double work, and, in so doing, doubles its size, without undergoing structural change; the gland is simply hypertrophied. The pathological enlargement of the kidney is like this, but with a difference. When acute Bright's disease leaves such an amount of swelling of tubes and consequent impediment of the circulation as interferes with the prompt and complete excretion of urine, there will continually be some excess of retained urinary products in the blood; and this accumulation of urinary excreta will act as a stimulus to increased growth and development of glandular tissue. For the tissues in question may be said to feed upon those materials for which they have a special affinity; and the growth of a gland is in proportion to the amount of the materials for its proper secretion with which it is supplied by the blood.

The hypertrophy goes on up to a certain point, and then a process of atrophy begins. The commencement of this is indicated during life by the appearance of oily casts and cells in the urine; and then, after death, the kidneys are found in the second stage—namely, that of fatty granulation, as before described, and as represented in fig. 3 of Dr. Bright's third plate.

These yellow specks are usually numerous in the cortex, but are never seen in the cones. Place a section of a yellow spot under the microscope, and you see that, as a hæmorrhagic spot is a convoluted tube filled with extravasated blood, this yellow speck is a tube filled with oil, mostly within cells, but partly loose. It is evident that, in this stage, the epithelium in certain sets of tubes has undergone fatty degeneration (see fig. 36, p. 726). The fatty nature of the material is proved by its solubility in ether, and by the smaller particles fusing and forming larger globules when gently warmed. The probable explanation of these spots of degeneration is, that the

swollen tubes so compress the intertubular capillaries, and thus impede the circulation, that the nutrition of the tubes is impaired, and their cells undergo fatty degeneration. The phenomena are analogous to the softening, with fatty transformation of the brain-tissue, as a result of embolism in a cerebral artery, and like the circumscribed patches of fatty degeneration in the kidney which follow upon and are caused by embolic obstruction in a branch of the renal artery. They also resemble the circumscribed patches of fatty degeneration of the muscular walls of the heart consequent on obstruction of branches of the coronary artery distributed to the diseased parts.[1]

But the degenerative changes may proceed further, and lead to a rapid disintegration of the gland-cells, and their re-

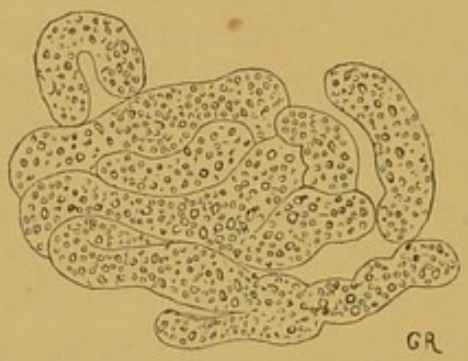


FIG. 36.—A YELLOW SPECK OR GRANULATION SHOWN TO BE A CONVOLUTED TUBE, WITH ITS CONTENTS IN A STATE OF FATTY DEGENERATION.—× 100.

placement by unorganised fibrine. This change is indicated during life by the appearance of large-sized granular and hyaline casts in the urine (see fig. 35), and after death by some of the tubes being filled with the same materials. These destructive changes in the gland-cells explain the atrophy and the granular contraction of the kidney in the third stage of the disease. In the stage of granular contraction cysts sometimes form on the surface or in the substance of the cortex; and in such kidneys the microscopic appearances, including the uni-nucleated cell-lining of some tubes (fig. 26, p. 689), which we have described as occurring in the red granular kidney, are present. I may mention here that a large white

[1] See, upon this point, Dr. Quain's paper 'On Fatty Degeneration of the Heart,' *Med.-Chir. Trans.*, vol. xxxiii. p. 147.

kidney, the result of acute Bright's disease, sometimes passes on into the stage of atrophy with a coarsely granular surface, without going through the intermediate stage of fatty degeneration. This I know to be a fact, from close observation of the urine during life, and a comparison of its microscopic characters with the appearances in the kidney after death. In such cases you may find here and there microscopic evidence of fatty degeneration within a tube; but the change is so slight as to be invisible by the unaided eye. No fatty granulations appear upon the surface or on a section of the gland. The atrophic stage of a kidney which had been enlarged and had undergone localised fatty degeneration is represented by Plate III. in a paper of mine, *On the Forms and Stages of Bright's Disease of the Kidneys.*[1] The atrophy and coarse granulation of a previously enlarged kidney without the fatty granulations are shown in Plate IV. of the same paper, and in figs. 1 and 2 of Dr. Bright's third plate. In the more advanced stages of this chronic disease, more especially in the stage of atrophy, the muscular walls of the arteries are almost always more or less hypertrophied. The explanation of the fact which I just now mentioned—that, as a rule, there is an inverse relation between enlargement of the kidney and hypertrophy of the arterial walls—is, that up to a certain point the increased growth of the glandular tissues obviates the necessity for that stop-cock action of the minute arteries which occurs when the glandular tissue is wasting, and which results in hypertrophy of the arterial walls, as I have explained in the previous section. The rule is, that the arterial hypertrophy commences when the glandular hypertrophy ceases and is succeeded by atrophic changes in the gland-cells and tubes. The Malpighian capillaries are usually more thickened, glistening, and wax-like than in the earlier stages of the disease. This apparently is a result of the continued transudation of albuminous and fibrinous materials through their walls, and their consequent infiltration with these products.

We occasionally find that the walls of some of the renal arteries have undergone a peculiar change which gives them a homogeneous wax-like appearance. I shall hereafter de-

[1] *Medico.-Chir. Trans.*, vol. xlii. p. 153.

scribe and explain this form of degeneration in connection with lardaceous disease of the kidney. In the stage of atrophy and contraction, some convoluted tubes may be seen denuded, some being contracted and others dilated. These appearances are the same as are found in the small red kidney. In the advanced stages of the disease, too, the basement-membrane of some of the tubes and the capsules of the Malpighian bodies are somewhat thickened, but less decidedly and constantly than in the contracted granular kidney.

Symptoms.—Amongst the symptoms of chronic Bright's disease with large white kidneys, *dropsy* is one of the most frequent and prominent. The cases in which dropsy is absent throughout the whole progress of the disease form a very small minority. Some years since I found, from an analysis of twenty-six fatal cases, that there had been dropsy in twenty-four; the proportion being, in round numbers, 92 per cent. And in the majority of cases the dropsy was great, general, and of long duration.[1] When chronic disease has supervened upon an acute attack, the dropsy which attended the onset of the malady may continue throughout, or it may pass away and return after a variable interval of months or years; or, if the urine become copious, as it sometimes does in the second and third stages, the dropsy may never return. Acute renal dropsy usually becomes general within a short period; but the dropsy which accompanies this chronic form of disease shows itself first in the face and feet, and gradually extends to other parts, including, in the worst cases, the serous membranes of the chest and abdomen. The pale, pasty, and puffed appearance of the face is very characteristic of this form of renal disease. As a rule, the more scanty the secretion of urine, and the larger the proportion of albumen which it contains, the greater is the tendency to dropsy.

When dropsical swelling of the legs becomes excessive, so as to cause great tension of the integuments, the circulation through the skin and the subcutaneous tissues is seriously impeded; and this may result in erysipelatous inflammation, sloughing, and gangrene. In one case, I saw gangrene and

[1] See my paper 'On the Forms and Stages of Bright's Disease,' *Med.-Chir. Trans.*, vol. xlii.

sloughing of the skin over the back of both legs, excited by the pressure of the heavy dropsical limbs upon the bed.

Congestion and œdema of the lungs are often associated with, and may be said to form a part of, the dropsical symptoms. The lungs are not rarely the seat of inflammatory mischief. *Bronchitis* and *pneumonia* are amongst the more frequent and serious complications. *Submucous œdema of the larynx* occasionally occurs, and renders the voice husky; more rarely it causes stridulous breathing and dyspnœa. *Inflammation of the serous membranes* is one of the less frequent complications. According to my experience, the pleura is more frequently inflamed than the pericardium; the peritoneum less frequently than either. *Endocarditis* is sometimes set up, and may result in a chronic valvular disease.

Derangements of the stomach and bowels are of common occurrence in all stages of the disease, but more especially in the advanced stages, and when, from any cause, the secretion of urine becomes scanty. There is loss of appetite; dyspepsia, with flatulent distension after food; nausea; water-brash; and vomiting, especially in the morning, when it is often excited by the attempt to clean the teeth. When the urine is scanty, the vomited matters often have a fœtid ammoniacal odour. In the advanced stages, the vomiting may be almost incessant and quite irrepressible. *Diarrhœa*, too, is not an unfrequent symptom. It may be excited by ill-digested food, or by the vicarious excretion of urinary products; not unfrequently, perhaps, by both these influences combined. In the advanced stages, the skin is usually unperspiring, dry and harsh.

Hypertrophy of the heart is less frequently associated with this form of disease than with the chronic desquamative disease. The more advanced the stage of disease, the more frequent is the occurrence of hypertrophy. Dr. Grainger Stewart found this condition in only 12 per cent. of the cases fatal in the first stage, in 38 per cent. of those fatal in the second stage, and in 100 per cent. of those fatal in the third stage—that is, the stage of contraction.[1] The probable explanation of the comparative infrequency of hypertrophy of the heart in the earlier stages is, that until the gland-cells

[1] *A Practical Treatise on Bright's Diseases*, 2nd ed. p. 90.

have undergone extensive degenerative changes, the uræmic condition which excites the arterial resistance and the resulting cardiac hypertrophy does not occur. There is, as a rule, a direct relation between atrophy of the proper glandular tissues of the kidney, hypertrophy of the systemic arterioles, and the correlated hypertrophy of the left ventricle of the heart. For the same reason, too, *cerebral symptoms of uræmic origin* and *cerebral hæmorrhage* are less frequently associated with the large white kidney than with the contracted granular kidney. It appears that, while dropsy results from hydræmia, arterial resistance, hypertrophy of the heart, toxæmic nervous symptoms, and cerebral hæmorrhage, are more direct results of uræmia.

The two forms of *defect of vision* which I described as of frequent occurrence in connection with the small red kidney, are less frequently associated with the disease which we are now discussing. The difference is one of degree. I have seen several cases of uræmic amaurosis and albuminuric retinitis, with an unquestionable history of an enlarged, white, and fat kidney.

Although cerebral hæmorrhage is a less frequent result of this form of disease than of the chronic desquamative disease, *hæmorrhage from mucous membranes*, and especially from that of the nose, is, in the advanced stages, a frequent and often a formidable symptom. These hæmorrhages are probably in part explained by the blood-deterioration, more especially the deficiency of albumen and hæmoglobin, and in part by the malnutrition and consequent brittleness of the walls of the vessels in the advanced stages of the disease.

Diagnosis.—On the subject of diagnosis, I need not add much to what I have already said. A careful consideration of the general history and the symptoms, together with the character of the urine, will rarely leave you in doubt as to the form and stage of the disease. Renal disease dating from an attack of acute general dropsy, followed by persistent albuminuria, can rarely be other than the particular form of disease which we are now discussing. The stage of the disease is to be determined chiefly by the character of the urine. In the first stage, the urine is usually normal in quantity, in colour, transparency, and specific gravity. There

is either no deposit, or a cloudy sediment containing some small hyaline casts. As the disease advances, the urine gradually loses its sherry tint, and becomes lighter coloured. The amount secreted and the specific gravity usually bear an inverse relation to each other. In the second stage, the small hyaline and oily casts are found; and in the third stage the oily casts are mingled with or replaced by the large granular and large hyaline casts (figs. 33, 34, and 35).

Prognosis.—Your judgment as to the probable result of the malady will obviously be greatly influenced by the opinion which you may form as to the stage of the disease and the rate at which it is making progress. In my lecture on acute Bright's disease, I told you that I have seen cases of complete recovery after oily casts and cells had appeared in the urine continuously for many weeks, and after albuminuria had existed for one, two, three, and, in one case, seven years.

The most favourable condition of urine is that in which it retains its normal colour, deposits no sediment on standing, and contains but little albumen. There can be no question that recovery may take place after the disease has passed into the second stage—namely, that of fatty degeneration. You are not, therefore, to despair of a patient whose urine contains oily casts, even in large numbers. As a general rule, the longer the continuance of albuminuria in spite of careful treatment, and the greater the amount of albumen, the more unfavourable is the prognosis. In estimating the amount of albumen, never omit to compare the urine after food and exercise with that passed after rest and fasting. When the urine is very pale, and of low specific gravity, yet highly albuminous, when it deposits a copious and dense sediment composed in great part of large granular and large hyaline casts, it will be evident that the disease is in the third stage, and that the kidney is contracting. The number of the large-sized casts may be taken as an index of the rate at which the degenerative and atrophic changes are progressing.

As in cases of the small red granular kidney, so in the form of disease which we are now considering, high arterial tension with the signs of cardiac hypertrophy, doubling of the first sound and accentuation of the second sound over the aorta,

are important indications of advanced degeneration of the glandular tissues.

The duration of the disease, in cases which ultimately prove fatal, varies extremely. I have notes of the case of a child aged 7, who had acute renal disease with dropsy after scarlet fever; and, the malady having terminated fatally within five weeks from its onset, the kidneys were already in the second stage, the characteristic yellow spots of fatty degeneration being scattered over the pale and enlarged cortex. In this case, the disease ran an unusually rapid course. I have seen a considerable number of cases in which the symptoms have continued for from five to ten years. My experience does not accord with the statements of some writers, who affirm that this disease is of shorter duration than that which results in the red granular kidney. I have before referred to one case which extended over a period of ten years; but the most prolonged case that I have seen or heard of was that of a medical practitioner whose history is partly given in my book on *Diseases of the Kidney* (p. 374). He had dropsy after scarlet fever in 1836, when he was about seventeen years of age. He recovered from the dropsy, and thought no more of his malady until five years afterwards, when his urine was accidentally discovered to be albuminous by a fellow medical student. It had probably been so since the attack of dropsy; and it certainly remained albuminous from that time until his death, which resulted from dropsy in May 1866. He was then in his forty-seventh year; and, if we assume, as we safely may, that the urine had not ceased to be albuminous between the attack of dropsy and the accidental discovery of the albumen five years later, this gentleman had albuminuria for thirty years before his death; yet during the greater part of that time he was a hard-working general practitioner, and, to all outward appearance, in good health. During the last year or two of his life, his urine was saccharine as well as albuminous. When I first saw him in 1851, his urine was of normal colour and specific gravity, but albuminous; it deposited no sediment, and contained no tube-casts. I believe that for many years it retained the same characters, but I have no note of any subsequent microscopical examination. This case teaches two practical

lessons. The first is, not to take for granted that a patient who has recovered from acute renal dropsy is well until his urine has lost all trace of albumen; and the second is, not to assume that persistent albuminuria of necessity involves early death or the speedy occurrence of formidable symptoms.

It is evident from the history of numerous similar cases, that the continuous filtering of albumen through the Malpighian capillaries, while insufficient to exert any appreciable weakening influence upon the system, does not interfere with the excretory function of the kidney, until in the course of time, extending it may be to many years, the glandular structures become disorganised.

The Simple Fat Kidney or General Fatty Infiltration of the Kidney.—Before passing on to the subject of lardaceous degeneration of the kidney, I wish to direct your attention to a condition of the kidney which may be designated 'the simple fat kidney' or 'general fatty infiltration of the kidney.'

There is a form of fat kidney very different from that which I have described as the granular fat kidney. It consists in a uniform infiltration of the epithelium of the convoluted tubes with oil. This state of kidney is analogous to the fatty infiltration of the cells of the liver which occurs often in cases of phthisis and other wasting diseases. This form of fat kidney I discovered and described in the *Medico-Chirurgical Transactions* (vol. xxix.) in 1846. In that paper, however, written within a few months after I had begun to work with the microscope, I misinterpreted the facts, and greatly overestimated the frequency and the importance of this form of fatty change.

The fatty infiltration of the renal epithelium may be found in various grades. When the fat is very abundant, the kidney is increased in size and weight. The colour of the cortex is either uniformly pale, or more frequently mottled by a blending of pale anæmic with red vascular patches. Occasionally hæmorrhagic spots are scattered through the cortical substance. The medullary cones retain their normal colour and vascularity. The consistence of the kidney is usually softer than natural, and frequently the gland has an œdematous feel and appearance. On a microscopic examination, the convo-

luted tubes are found to be universally distended with oil which has accumulated in their epithelial cells. There is a uniform oily infiltration of the renal gland-cells (see fig. 37). This condition of the kidney is found not unfrequently associated with a similar condition of liver in persons who have an excess of adipose tissue beneath the skin, in the abdomen and about the heart. It is commonly found after death from diabetes and from some other chronic diseases which are attended with great emaciation, such as cancer, phthisis, and dysentery. It is probable that the immediate cause of this fatty infiltration of the gland-cells is an excess of fatty matter in the blood. In the case of very fat persons, who are usually large consumers of fat-making adipose and amyloid food, the materials whence the fat is derived are introduced directly into

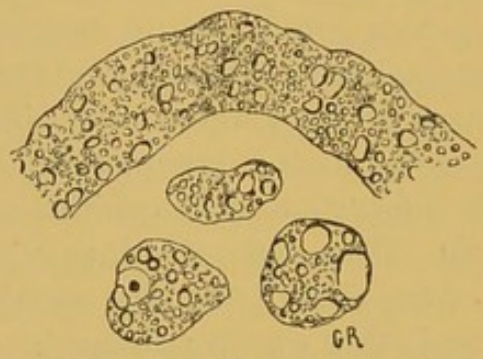


FIG. 37.—A PORTION OF A CONVOLUTED TUBE DISTENDED WITH OIL, FROM A 'SIMPLE FAT KIDNEY.'

Three detached epithelial cells; two are filled and distended with oil, the third contains oil in less quantity, and the cell-nucleus is visible.— × 200.

the blood through the stomach. On the other hand, in cases of wasting disease, it is probable that the fat absorbed from the adipose tissues enters the circulation and infiltrates the gland-cells, more commonly those of the liver, less frequently those of the kidney.

Fat kidneys are common in the domestic dog and cat, probably because these animals lead indolent lives, and consume large quantities of food rich in hydrocarbon. These animals are the counterparts of the human animal when, from eating and drinking to excess, he grows fat and gets fat liver, heart, and kidney. The unhappy Strasbourg geese afford an illustration of fatty infiltration of the liver resulting from a wasting disease. In order to obtain fat livers for patties, the

animals are well fed and fattened; then they are confined in heated cages without food and water. They become feverish, and rapidly waste, while their livers grow large. It seems probable, as Baron Larrey long ago suggested, that the oil absorbed from the adipose tissue enters the circulation and infiltrates the cells of the liver, and probably in a less degree those of the kidney also.

It is a remarkable fact that the liver and kidneys have been found in a state of extreme fatty infiltration in cases of poisoning by phosphorus, death occurring within a week after the poison was taken. Two cases of this kind are recorded in the fiftieth volume of the *Medico-Chirurgical Transactions*—one by Dr. Habershon, the other by the late Dr. Hillier. Fatty infiltration of the liver and kidney appears, as a rule, to have but little influence on the functions of these glands. We sometimes, however, find jaundice and ascites associated with fat liver, and with no other structural change to explain the symptoms; and I have notes of several cases in which albuminuria and the usual symptoms of chronic Bright's disease have occurred in connection with simple fatty infiltration of the convoluted tubes of the kidney. One such case I published in my book on *Diseases of the Kidney*.[1] In that case, the disease followed a second attack of scarlet fever with anasarca. There was dropsy, and the urine contained much albumen and oily cells. She died with uræmic convulsions and coma. The convoluted tubes were greatly and almost uniformly gorged with oil; and my friend Dr. Beale, analysing the cortex of the kidney, found that more than one-fourth of the solid matter was fat. It is reasonable to suppose that so large an accumulation of oil within the gland-cells must impair their secreting power, and also impede the circulation through the intertubular capillaries, which are compressed by the distended and swollen tubes.

I will now briefly recapitulate the chief points of distinction between the 'granular fat kidney,' and what I have here called the 'simple fat kidney,' which is perhaps a better term than 'the mottled fat kidney,' which I formerly employed, but which, I am told, has often been misunderstood. In the

[1] Case of Ann White, p. 414.

granular fat kidney, there are disseminated spots of fatty degeneration in the cortex; and these are secondary results of previous structural changes in the gland. In the simple fat kidney, on the contrary, there is a general fatty infiltration of the gland-cells in the convoluted tubes of the cortex; and this is a primary change. The granular fat kidney is always associated with albuminuria, and often with other signs of serious disturbance of function. The fatty infiltration, although it is sometimes associated with albuminuria and other symptoms of renal disease, is, in the majority of cases, unattended by obvious signs of functional derangement. This condition of kidney, therefore, while it has great interest for the pathologist, has much less clinical importance than the 'granular fat kidney;' but I cannot assent to the statement that, because overfed animals leading unnaturally indolent lives have an excess of oil in their kidneys, this condition is normal or innocuous. By parity of reasoning, it might be maintained that an excessive growth of fat about the heart is a harmless addition of hydrocarbon to the weight of the body, because that state of heart often coexists for a time with apparently good health and great bodily activity.

SECTION VII.

CHRONIC BRIGHT'S DISEASE WITH A LARDACEOUS OR WAXY KIDNEY.

General History—Virchow's Theory—Clinical History and Symptoms—Minute Anatomy and Pathology of the Kidney—Its Relation to Continued and Profuse Suppuration—Hypertrophy of the Heart—Diagnosis—Prognosis—Hæmaturia in Chronic Bright's Disease.

There are cases of chronic Bright's disease associated with kidneys which are usually enlarged, anæmic, pale, and waxlike; thus resembling in some respects the cases which I described in the last section. But the form of disease to which I now invite attention has, for the most part, a peculiar and distinctive clinical history; and the anatomical condition of the kidney is, in some respects, different from that of the ordinary large white kidney.

General History.—The subjects of this form of disease have usually been strumous or otherwise cachectic before the onset of the renal degeneration. In some patients, there has been strumous disease of a joint or of one or more bones, with long-continued suppuration; in others there are symptoms of phthisis. In a large proportion of cases, there is a history of constitutional syphilis, with resulting cachexia. In some instances cancer, in others dysentery, in others habitual intemperance, in others, again, long-contained albuminuria following upon acute Bright's disease, has led to the cachexia out of which this form of renal disease has sprung. One of the earliest symptoms of the disease is a copious flow of urine, at first perhaps not albuminous, but subsequently more or less impregnated with albumen. Another common symptom is profuse and obstinate diarrhœa. Dropsy, more or less general, usually occurs; but it is not so constant or so prominent a symptom as in the class of cases which I described in my last lecture as sequelæ of acute Bright's disease. When at length the patient dies, sometimes from uræmia, but more frequently from exhaustion, the kidneys are found in a state which has been called 'lardaceous' or 'waxy degeneration.' The gland is usually enlarged, sometimes very much so. In one of my own cases, the two kidneys weighed twenty-eight ounces. Dr. Dickinson in one case found their combined weight thirty-three ounces. The surface of the kidney is smooth and pale; the texture of the anæmic and thickened cortex is firm, and has the semi-translucent appearance of white beeswax; while the cones retain their normal colour, vascularity, and size. The cut surface presents numerous glistening points, due to the altered Malpighian capillaries. In some cases, minute yellow fat-granulations are scattered through the cortex. This is the large, smooth, lardaceous kidney; and one of its most remarkable and distinctive features is, that in the majority of cases it is associated with an analogous condition of the liver or spleen, or of both. In a certain proportion of cases, a stage of atrophy follows upon that of enlargement; the cortical substance wastes, and coarse granulations appear on the surface. This is the 'contracted' or 'granular lardaceous kidney.'

Before entering upon a minute description of the kidneys, it will be well to give a brief history of the disease and of the speculative doctrines to which it has given rise. Two theories regarding this disease I believe to be erroneous. The one theory assumes that the blood-vessels are the primary seat of the degenerative changes, and the other that the morbid deposit is of the nature of starch or vegetable cellulose; and in accordance with the latter theory the disease has been called 'amyloid degeneration.' I shall presently show you that the first theory is erroneous; and the second is now universally admitted to be so. The term 'amyloid' was suggested by a supposed chemical resemblance between the morbid deposit and vegetable cellulose or starch, as shown by the staining with a solution of iodine; but careful analyses by various competent chemists have shown that the material has essentially the same composition as the protein compounds, and that it is of an albuminous or fibrinous nature.

It is very desirable that the term amyloid, which is based upon an erroneous chemical theory, should be discontinued, and that the term 'lardaceous,' recommended by a Committee of the Pathological Society,[1] should be adopted. The term lardaceous means no more than that the disease has the appearance of bacon-fat (*lardum*, the fat of bacon); as the term 'waxy' is based upon its resemblance to wax. These names, thus understood, imply no theory as to the chemical composition of the morbid product, and are, therefore, not misleading, as the term amyloid unquestionably is.

Clinical History and Symptoms.—The clinical history of this disease has been carefully investigated, and we are indebted to Dr. Grainger Stewart for insisting upon the fact that a copious flow of urine, of pale colour and of low specific gravity, is one of the earliest and most constant symptoms of this form of degeneration. The urine at first may contain no albumen; but gradually it becomes albuminous and often copiously albuminous. When a patient, whose strength has been reduced by a protracted and exhausting disease, begins to pass urine in large amount and of low density, so that his nights are disturbed by frequent calls to empty the bladder

[1] *Pathological Transactions*, vol. xxii. p. 1.

and to quench his thirst, we may anticipate that his kidneys are about to undergo the degenerative changes which we are now discussing. In the absence of sugar and albumen, it may for a time be a question whether the disease is diabetes insipidus. The appearance of albumen points at once to renal degeneration. The amount of urine secreted daily usually ranges from 50 to 100 ounces or more. The colour is pale, and the specific gravity varies from 1005 to 1015. The urine may be clear and deposit no sediment, so that for days and even weeks together no tube-casts are visible; and, in the earliest stages of the disease, tube-casts are never numerous. In most cases, however, a light cloud collects at the bottom of the conical glass, and in this cloud we may find some small hyaline casts, some casts finely granular, and occasionally some hyaline casts containing oil, either in scattered globules or in cells. In the advanced stage of the disease, when atrophy and contraction of the kidney are in progress, the sediment in the urine may be copious and dense; and it will be found to contain numerous large-sized granular and hyaline casts, exactly similar to those which I described as occurring when the large white kidney has reached the third stage, and is undergoing atrophy and contraction.[1] As the disease makes progress the patient's weakness increases; his breath is short on exertion; his countenance is pallid or sallow; the feet, ankles, and legs become œdematous. In many cases the liver and the spleen are seen and felt to be more or less enlarged, and the abdomen is sometimes much distended by fluid. The disease often has a very chronic course, extending over a period of many months or even years. There may sometimes be a temporary amendment; but in the majority of cases the symptoms gradually become worse, until at length the patient sinks, either from the direct effects of the renal disease, or from one or other of the associated maladies.

The immediate cause of death may be dropsical effusion within the chest—in the pleura or pericardium, or in both. In other cases the patient dies exhausted by diarrhœa, with or without vomiting—the result, probably, of blood-deterioration

[1] See *ante*, figs. 33, 34, and 35, pp. 722, 723.

and the elimination of morbid materials through the mucous membrane of the alimentary canal. In some instances the immediate cause of death is an attack of convulsions or coma. Cerebral symptoms of uræmic origin are, however, less frequent results of this than of other forms of chronic Bright's disease. The retina is rarely if ever affected. Inflammatory complications are of common occurrence. Of these, pneumonia is the most frequent; next to this, inflammation of the serous membranes, especially of the pleura. The pericardium and the peritoneum are more rarely the seat of inflammation.

In many cases death results, not from the direct consequences of the renal degeneration, but from some associated constitutional disease or cachexia. Thus phthisis, or protracted suppuration, with or without disease of the bones or joints, or some form of constitutional syphilis, may be the immediate cause of death.

In the advanced stages of the disease there is extreme anæmia and pallor of the skin; the blood contains much less than its due proportion of hæmoglobin and albumen, and in some cases an excess of urea.

Hæmorrhage from one or more mucous membranes, more especially from that of the nose, is an occasional occurrence in the advanced stages of the disease.

The Minute Anatomy and Pathology of the Lardaceous Kidney.—Most recent writers on renal pathology accept the theory which assumes that the first pathological change consists in thickening and degeneration of the walls of the minute arteries and Malpighian capillaries. In consequence of this degeneration, we are told, albuminous and fibrinous materials transude through the walls of the vessels, and infiltrate the tissues of the kidney; and this is supposed to explain the structural changes in the gland. It has also been suggested that the copious secretion of urine in the early stage of the disease is a result and an indication of paralysis and dilatation of the minute renal arteries consequent on degeneration of their walls. I find this theory inconsistent with anatomical facts, and therefore I reject it entirely. For a number of years I have most carefully studied the condition of the renal blood-vessels in all forms and stages of Bright's disease. My

discovery of hypertrophy of the muscular walls of the arteries was published in the year 1850,[1] some years before the publication of Virchow's theory of amyloid degeneration. I soon learnt to distinguish muscular hypertrophy from lardaceous and fatty degeneration of the arterial walls; and I have carefully noted the microscopic appearances in a large number of diseased kidneys. The result is that, while I have not met with a single case in which thickening of the renal blood-vessels, in any form, was unassociated with extensive changes in the secreting tissue of the kidney, I have examined many kidneys in an advanced stage of lardaceous disease with only incipient degeneration of the blood-vessels. For example, I have before referred to one case in which the two kidneys weighed twenty-eight ounces. The patient at the time of his death was twenty-one years of age. Since the age of three he had suffered from disease of the hip, with purulent discharge from several openings about the joint. For ten or eleven years there had been more or less dropsy, and for several months the dropsy had been general. The clinical history, the character of the urine, and the appearance of the kidneys were those of a typical case of lardaceous degeneration of the kidney; yet the Malpighian capillaries and the arteries in these greatly enlarged, pale, and wax-like kidneys, after the long duration of the symptoms, were only moderately thickened. The gland had increased to nearly three times its normal weight, while the vascular changes were in an incipient stage. The increased size of the gland was not such as could be explained by a mere infiltration of fibrinous material; but the enlargement was the result mainly of an actual hypertrophy of the glandular tissue, analogous to that which I described in the previous section as occurring in cases of the large white kidney. Most of the tubes were enlarged, and their epithelium was opaque from 'cloudy swelling.' In some, the cells were in a state of fatty degeneration; and some tubes contained fibrinous coagula, precisely similar to the large hyaline casts which had appeared in the urine during life. In some cases, more especially when the disease has passed into the stage of atrophy and contraction, the gland-cells have undergone more

[1] *Med.-Chir. Trans.*, vol. xxxiii.

general and extensive degeneration. Numerous small yellow spots visible by the naked eye, when present, indicate the situation of tubes whose contents have undergone fatty degeneration[1]; while other tubes are filled with unorganised fibrine, which may sometimes be squeezed out of them in the form of large hyaline casts. The basement membrane, both in the cortex and in the cones, sometimes appears thickened

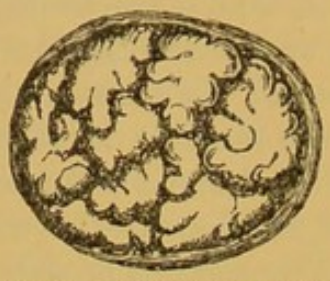


FIG. 38.—MALPIGHIAN CAPILLARIES, WITH OPAQUE GLISTENING WAX-LIKE WALLS. The capsule somewhat thickened.—× 200.

and hyaline; and occasionally the tubular structure is rendered indistinct by an unorganised intertubular effusion. The Malpighian capillaries are thickened, opaque, glistening, and wax-like (fig. 38). Some of the afferent arteries appear quite normal, others are thickened by muscular hypertrophy; but the greater number appear thick, more or less homogeneous, and wax-like; their muscular structure being concealed apparently by an interstitial fibrinous infiltration (see fig. 39).

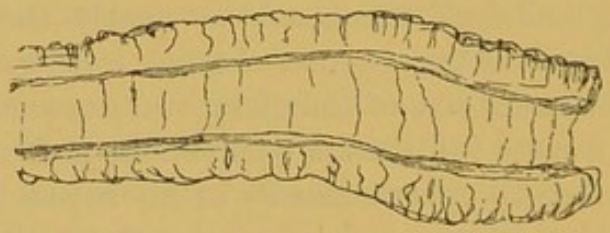


FIG. 39.—A RENAL ARTERY WITH LARDACEOUS INFILTRATION OF ITS WALLS.—× 200.

The straight arteries in the cones sometimes present the same appearance of wax-like degeneration. If we now add to the specimen a drop or two of diluted liquor potassæ (one part of solution of potash, P.B., in ten of water), the walls of the waxy vessels are renderd transparent; so that the red blood-corpuscles become visible through the thickened Malpighian capillaries, and the muscular fibres of the minute arteries are

[1] See *ante*, fig. 36, p. 726.

rendered quite distinct. The weak alkali has a solvent action upon the infiltrated fibrinous material, and thus to some extent brings into view the normal structure of the arterial walls. The canals of the afferent arteries may sometimes be seen irregularly dilated. In some of the arterial canals a collection of oil-globules shows that the circulation has ceased, some time before death (fig. 40). Oil-globules may often be seen in the canals and in the walls of the Malpighian capillaries—less frequently within the intertubular capillaries. I have never seen thickening of the walls of the intertubular capillaries. Thickening of the basement-membrane often gives an appearance of intertubular thickening. When fatty granulations are

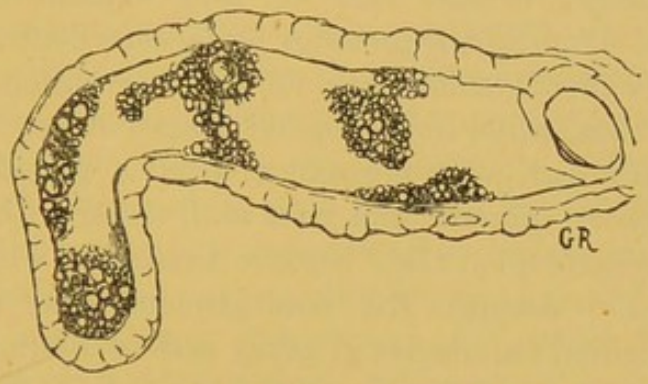


FIG. 40.—A RENAL ARTERY, WITH LARDACEOUS INFILTRATION OF ITS WALLS AND COLLECTIONS OF OIL-GLOBULES IN THE CANAL, WHICH IS SOMEWHAT IRREGULARLY DILATED.—× 200.

visible to the naked eye, their microscopic appearances are identical with those which I have before described in the granular fat kidney.

In the previous section I have shown that the Malpighian capillaries and the walls of the minute arteries in some large white kidneys are thickened, homogeneous, and wax-like. The vessels in these cases have undergone precisely the same change as those in the lardaceous kidney. In fact, the large white kidney, which is found associated with chronic albuminuria, following upon acute Bright's disease, and the lardaceous kidney, which occurs in connection with chronic cachexia, have many points of contact and relationship; and the two forms of disease merge into each other by imperceptible gradations. Chronic albuminuria is generally admitted to be one of the causes of lardaceous degeneration of the kidney.

All writers on the lardaceous kidney agree in stating that the primary cause of the renal degeneration is a morbid condition of the blood. In this opinion I concur; and I believe that some morbid material in the blood, acting upon the secreting tissues of the kidney, is the cause of the copious secretion of urine in the earlier stages of the disease, and of the glandular hypertrophy which gradually supervenes during the progress of the malady. It is probable that the unknown morbid material in the blood has a diuretic influence upon the kidney, analogous to that which grape-sugar is known to have in cases of saccharine diabetes. We do not attempt to explain the diabetic urinary flux by the anatomical condition of the kidney; neither can we thus explain the copious secretion of urine which precedes lardaceous degeneration. I say *precedes* the degeneration; for my observations have convinced me that, while in the earlier stages of the disease the arterial walls are quite normal, at a later period they may become hypertrophied; and later still, either with or without previous hypertrophy, they become infiltrated with fibrinous material, and assume the homogeneous waxy appearance. The Malpighian capillaries in every case of albuminuria have a more or less abnormal appearance. I have shown in section iv. that, after death from acute Bright's disease, the walls of the Malpighian capillaries are opaque and granular. In all cases of chronic albuminuria, these capillaries are thickened; and, when the transudation of albumen has been copious and long continued, the capillary walls always assume an opaque, glistening wax-like appearance. The state of the Malpighian capillaries in the large white kidney is not distinguishable from that of the same vessels in the lardaceous kidney, either by microscopic examination or by any chemical test with which I am acquainted. The test which is usually applied is a weak solution of iodine. The thickened arteries and Malpighian capillaries take the reddish-brown stain much more deeply than the other tissues; and the stained vessels consequently stand out in strong contrast with the pale, waxy, glandular structure. This iodine test, applied to the large white smooth kidney, often stains the vessels as deeply as when applied to the lardaceous kidney, and thus affords addi-

tional evidence of the close relationship between the two forms of disease. I cannot but think that too much importance has been attached to the iodine test, while too little attention has been given to the minute structural changes in the kidney, and their physiological interpretation.

Dr. Dickinson attributes so much influence to *suppuration* as causative of lardaceous degeneration, that he proposed to call the disease 'depurative,' using the word in a sense different from that in which it is commonly understood. Analyses of the morbid material, especially in the liver, have led him to the conclusion that it consists of partially dealkalised fibrine. He supposes that copious suppuration, by lessening the albumen and alkalies in the blood, causes a deposit of dealkalised fibrine in various tissues and organs. It seems not improbable that the diminished alkalescence of the waxy liver may be explained by the comparatively small proportion of alkaline blood which it contains. The liver-cells are infiltrated with, and in part replaced by, fibrinous material; while the vessels are compressed and anæmic. There is no apparent difference between the fibrinous exudation within the tubes of a lardaceous kidney and that which is found in the tubes of a large white kidney, the result of acute Bright's disease passing into a chronic form. Our knowledge of animal chemistry is as yet too elementary to enable us to give a chemical explanation of pathological changes so complex as those which we are now discussing; and, although the lardaceous disease is very frequently associated with suppuration, yet this is far from being constant. The renal degeneration occurs in only a small proportion of cases in which there has been profuse and protracted suppuration; and, on the other hand, the lardaceous form of renal degeneration not unfrequently occurs unassociated with a history of purulent discharges. Dr. Grainger Stewart states that in only six out of eighteen cases which he had himself carefully investigated, was there a history of suppuration. Obviously there is not that constant and close relationship between suppuration and lardaceous degeneration which justifies the application of the ambiguous term 'depurative' to this form of renal disease; and in the second edition of his work, Dr. Dickinson adopts the

term 'lardaceous.' It is probable that the deterioration of blood which results from long-continued profuse suppuration is due rather to the drain of albumen than to the loss of alkaline salts; and so it is intelligible that chronic suppuration and chronic albuminuria may bring about a similar condition of cachexia and malnutrition.

Hypertrophy of the heart, unassociated with disease of the valves or of the large arteries, rarely occurs, except in the cases which have passed on into the stage of atrophy. In explanation of this, we have to take into consideration the fact that the walls of the minute arteries in various tissues and organs usually undergo degenerative changes, whereby their contractile power is impaired. The degeneration of the blood-vessels is of common occurrence in the mucous membrane of the intestines, and the change is rendered conspicuous by the iodine test. The absence of abnormal contraction in the terminal arteries explains the absence of the cardiac hypertrophy which ordinarily results from long-continued and excessive arterial resistance.

Diagnosis.—You will probably have inferred, from what I have said of the close relationship between the 'large white kidney' and the 'lardaceous kidney,' that it is often difficult to distinguish one from the other, and that the distinction has but little practical value. Even when you have the diseased organ before you, you may sometimes be in doubt whether to call it simply a 'large white kidney,' or to designate it 'lardaceous.' Obviously, then, it must sometimes be impossible to make the distinction during the lifetime of the patient. When there has been a copious secretion of urine, which for a time was free from albumen, but later has become copiously albuminous; when a copious secretion of pale albuminous urine, of low specific gravity, is associated with more or less general dropsy; when these symptoms have come on gradually and insidiously in a subject who has been suffering from an exhausting disease such as phthisis, disease of the bones or joints, or cachexia resulting from cancer or constitutional syphilis, we may expect to find lardaceous degeneration. The probability of this will be much increased if the liver or spleen, or both these organs, be found enlarged and indurated. A larda-

ceous kidney may sometimes attain a sufficient size to be palpable in the lumbar region. The tube-casts, when present, are essentially the same in the two forms of disease; and in particular the large hyaline and granular casts (figs. 34 and 35, p. 723), which appear in the advanced stages of both classes of cases, indicate that atrophic changes are in progress, while the amount of sediment having these microscopic characters indicates the rate at which the destructive changes are proceeding.

Prognosis.—Although the history of this form of disease not unfrequently extends over a period of several years, as in the case to which I just now referred, yet the prognosis is, as a rule, very unfavourable—for the obvious reason, that not only is the renal disease often associated with serious structural change in other organs, but, resulting as it does, from a grave constitutional cachexia, its causes are continually operating, and, as a rule, they are but little amenable to treatment. There may be occasional pauses in the progress of the disease, and even periods of temporary amendment, but the usual course of the malady is one of steady progress towards a fatal termination. The end is often hastened by an exhausting diarrhœa, by a copious dropsical or inflammatory effusion into the chest or abdomen, or by inflammation and sloughing of the dropsical legs.

Hæmaturia in Chronic Bright's Disease.—In conclusion, I wish to direct your attention to a possible source of fallacy resulting from the occasional appearance of blood-tinged urine in the advanced stage of this and of other forms of chronic Bright's disease. Dark-coloured, smoky, more or less blood-coloured urine, is of frequent occurrence in cases of acute Bright's disease; it is rare in the advanced stages of any of the three forms of chronic disease which I have described, but it does occasionally happen; and the appearance of hæmaturia might possibly mislead you in your estimate of the stage and gravity of the renal disease.

During the progress of the various forms of chronic Bright's disease, the walls of the Malpighian capillaries become thickened, and therefore probably less liable to be ruptured. In many cases, too, the muscular walls of the minute arteries

are more or less hypertrophied; and the effect of this is to lessen the pressure upon the Malpighian capillaries, and the risk of their rupture. This appears to be the explanation of the undoubted fact that the pale urine of low specific gravity which is secreted by kidneys in an advanced stage of degeneration, is rarely tinged with blood. This rule, however, is not without exceptions. In the advanced stage of all forms of chronic Bright's disease, the blood becomes much deteriorated—partly, as we have seen, by the loss of its normal constituents, partly by the retention of urinary impurities. In some tissues, too, the minute arteries and capillaries may undergo degenerative changes which increase their liability to rupture. There is consequently a tendency to hæmorrhage from various mucous surfaces—from the nose, the lungs, the stomach and intestines, from the uterus, and sometimes from the mucous membrane of the bladder and the pelvis of the kidney. Hæmorrhage from the bladder, or from the pelvis of the kidney, may give the urine the dark colour and the blood-tinged appearance which it often has in cases of acute Bright's disease, when blood escapes from the substance of the kidney. You may come to a right judgment in these cases by a careful consideration of the past history, together with a close inspection of the urine. You will probably find that there are no blood-casts of the tubes, as there usually are when the substance of the kidney is the source of the bleeding. You may find some of those forms of tube-cast which point to the existence of chronic rather than recent acute disease: for instance, oily casts or large granular and large hyaline casts (figs. 33, 34, and 35, pp. 722, 723). You may also find that the urine, when, after standing for a time, it has deposited the blood, presents the pale colour which is indicative of chronic disease in an advanced stage.

Many years ago my friend Mr. James Salter sent me the notes of a case of *purpura* in which there had been profuse hæmaturia. The kidneys had been the seat of chronic Bright's disease; they were enlarged, anæmic, and had some cysts on their surface. The mucous membrane of the calyces, infundibula, and pelvis, was intensely congested and chocolate-coloured with ecchymosis. There was a striking contrast and

a sharp line of demarcation between the pale mamillæ and the dark ecchymosed calyces. It is probable that the absence of hæmorrhage into the substance of the kidney was due to the fact that the walls of the minute arteries and those of the Malpighian capillaries had become thickened during the progress of the chronic degeneration of the kidney which had long preceded the appearance of the purpura. This case affords a good illustration of the fact that hæmaturia, the result of blood-deterioration, may have its source in the pelvis, and not in the substance of the kidney. A microscopic examination of this patient's urine had discovered no tube-casts. None of the blood had been moulded within the uriniferous tubes, because none had escaped from the Malpighian capillaries.

SECTION VIII.

ALBUMINURIA NOT ASSOCIATED WITH BRIGHT'S DISEASE.

Albuminuria not associated with what is commonly understood as Bright's Disease—1. Passive Congestion of the Kidney with Albuminuria, the Result of Impeded Venous Circulation — 2. Albuminuria and Hæmaturia from Embolic Infarctions in the Kidney — 3. Puerperal Albuminuria — Four Classes of Cases — 4. Atrophy and Suppurative Inflammation of the Kidney from Retention of Urine — Symptoms and Diagnosis — 5. Acute Cystitis resembling acute Bright's Disease—Symptoms and Diagnosis.

Before I go on to discuss the treatment of Bright's disease, I wish to direct your attention to certain cases of albuminuria resulting from various causes, but not associated with what is commonly understood as Bright's disease. My object in referring to these cases now, is to give you some hints which may assist you to distinguish them from each other and from cases of actual Bright's disease.

I. *Passive Congestion of the Kidney with Albuminuria, the Result of Impeded Venous Circulation.*—There is a class of cases in which albuminuria results from passive congestion of the kidney consequent on some impediment to the return of blood through the systemic veins. The causes of this impediment are diverse. Valvular disease of the heart is one of the

most frequent of them. Degeneration and consequent weakness of the muscular walls of the heart is a not uncommon cause. The impediment may originate in the lungs, as a result of emphysema with bronchitis, of extensive pneumonic consolidation, or of compression of one or both lungs by a copious liquid effusion into the pleura. Again, a dropsical effusion in the cavity of the peritoneum, the result of cirrhosis or other obstructive disease of the liver, may so compress the vena cava and impede the return of venous blood, as to cause passive renal congestion and albuminuria. In the advanced stages of pregnancy, the pressure of the uterus on the veins may cause passive congestion of the kidney and albuminuria; but I shall presently refer more particularly to albuminuria in connection with pregnancy. In general, the diagnosis of each of these causes of impeded circulation is not difficult. Then, as to the effect upon the urine and the kidney: the urine becomes scanty, in proportion to the degree of venous congestion and the consequent tardiness of the blood-stream through the kidney. The secretion is usually high-coloured, of high specific gravity, often turbid with urates, and more or less impregnated with albumen.

The mechanism of albuminuria, consequent on the passive engorgement of the kidney which results from an impeded return of blood through the veins, may be illustrated by reference to fig. 6 (p. 627). When, in consequence of an obstruction at the heart, the systemic veins become overfull, the distension of the renal vein, acting backwards through the intertubular capillaries, causes engorgement of the Malpighian capillaries, and a consequent transudation of serum through their walls. This serous transudation, mingling with the urine, renders it albuminous. Small hyaline and granular casts may be seen when the turbid urine has been cleared by warmth or by dilution with water, so as to keep the urates in solution. The Malpighian capillaries are sometimes ruptured by over-distension; the urine is then blood-tinged, and blood-casts are visible. The secondary character of the renal complication is usually apparent from the history of these cases; and the diagnosis may sometimes be confirmed by the fact that, when the circulation has been relieved by rest in bed, by hydra-

gogues, by puncturing the legs, or by tapping the abdomen, the albumen disappears from the urine; to return, perhaps, when the circulation again becomes more embarrassed within the chest, or by the reaccumulation of liquid in the peritoneum. I have seen this happen again and again during the progress of the same case.

The first effect of passive congestion upon the kidney is to cause more or less enlargement with some induration of the gland. The ultimate result of long-continued congestion is atrophy and contraction; the surface of the kidney becoming uneven and finely granular, as the wasting process goes on. The explanation of the phenomena is not difficult. For the due performance of its secretory function, and for the maintenance of its nutrition, it is essential that the blood move freely through the gland. An impeded return of blood through the veins involves as a necessary consequence a partial blood stasis, and, as a result of this, a scanty secretion of urine, with impaired nutrition and atrophy of the gland. On microscopic examination of the kidneys, some of the tubes may be seen to be opaque with disintegrated epithelium and fibrine, some denuded, and in various stages of atrophy and contraction. When atrophy of the kidney has been a result of passive congestion consequent on a mechanical hindrance to the circulation, I have never found the walls of the minute renal arteries hypertrophied.

II. *Albuminuria and Hæmaturia from Embolism in the Minute Blood-vessels of the Kidney.*—There is yet another mode in which valvular disease of the heart may, for a time, render the urine albuminous and even bloody. You are aware that, when one of the valves of the heart has its surface roughened by inflammation or by senile degenerative changes, a very common result is a deposit of fibrine upon the roughened surface; and further, that these fibrinous deposits, having no organic union with the valve beneath, are very liable to become detached by the current of blood, and then to obstruct the vessels in any organ to which they may chance to be conveyed.[1] One result of this mechanical plugging of blood-vessels is the formation of so-called 'fibrinous deposits'

[1] See Chapter XXXVII. on Thrombosis and Embolism.

or 'infarctions' in the kidney. The portion of kidney which is the seat of recent obstruction is raised above the level of the surrounding renal tissue; it is firm, anæmic, and of a yellowish white colour, with an intensely red injected margin. The older deposits are softer than the surrounding tissue, appear shrunk and depressed, and have not the red margin. In a still more advanced stage, the appearance of a deposit entirely passes away, and a depressed cicatrix is left.

On a microscopic examination of a recent fibrinous patch, the tubes in the seat of the deposit appear opaque from containing fibrinous coagula. Some tubes contain oil. Many of the intertubular capillaries contain fibrinous coagula; while others contain oil-globules, which are clustered in the form of rings surrounding the tubes. Granular coagula and oil-globules may also be seen in some of the Malpighian capillaries and the afferent arteries. The coagula in the vessels are more clearly seen after the tissues have been rendered transparent by dilute acetic acid. In the red vascular zone which surrounds the recent deposit, the Malpighian and the intertubular capillaries are seen to be injected and gorged with blood.

The probable explanation of the phenomena is, that a soft fibrinous mass from a cardiac valve is arrested in a small branch of a renal artery. The onward movement of blood through the Malpighian and intertubular capillaries to which the obstructed artery leads is consequently arrested, and these vessels become injected with blood from the laterally communicating intertubular capillaries. The blood flowing in the direction of least resistance is driven in a backward direction through the intertubular and Malpighian capillaries towards the *cul-de-sac* of the obstructed arterial branch. Then albumen and sometimes blood escape from the distended or ruptured Malpighian capillaries into the uriniferous tubes; the urine consequently is albuminous and even blood-tinged. In the next stage, the blood stagnating in the capillaries, coagulates, and subsequently the exudation into the tubes, the epithelium of the tubes, and the fibrinous coagula within the obstructed blood-vessels, undergo a fatty transformation, and all trace of the normal structure disappears. The fatty matter at length

becomes absorbed, and a depressed cicatrix remains on the surface of the kidney. Two or more pale fibrinous patches of different dates may sometimes be found in the same kidney. In some instances, the fatty matter, which results from the transformation of the tissues and the fibrinous coagula, does not become absorbed, but remains encysted. This is the explanation of the cysts which are sometimes found filled with a thick dark liquid, composed of oil, free and in cells, with which, often, plates of cholesterine are mingled.

The diagnosis of embolism in the renal vessels is usually more or less uncertain. We may suspect the occurrence, when, with the physical signs of aortic or mitral disease, without great impediment of the general circulation, the urine suddenly becomes albuminous or bloody. In some cases, extensive embolism in one or both kidneys has been attended with severe lumbar pains, a scanty secretion of urine, and vomiting; but, when the obstructed portions of kidney are small, there may be no symptoms to indicate the occurrence of renal embolism.

III. *Puerperal Albuminuria.*—Since the time when Dr. Lever, in the *Guy's Hospital Reports* (1843), published the fact that puerperal convulsions are, in a large proportion of cases, associated with albuminuria, the subject of albuminuria in connection with pregnancy has excited much interest. Later observations have established the essential accuracy of Dr. Lever's observations; but they have also shown that the connection between puerperal convulsions and albuminuria is not constant. Convulsions may occur without albuminuria, and, on the other hand, albuminuria in pregnant women may be unassociated with convulsions. The few remarks which I propose to address to you on this subject will have reference mainly to the subject of puerperal albuminuria, and only incidentally to the association of puerperal convulsions with albuminuria. I have seen more or less of a considerable number of cases of albuminuria associated with pregnancy; and, looking over my notes of these cases, I find that they arrange themselves in four classes, each having in some respects a different history and pathology.

1. Women, known to be suffering from chronic Bright's

disease, may become pregnant, pass through all the stages of pregnancy and parturition, and even suckle their infants, without accident or complication. A lady whom I saw some years since with my friend Dr. S. H. Steel, of Abergavenny, while suffering from chronic Bright's disease supervening upon an acute attack which resulted from exposure to cold, twice became pregnant, each time had an uncomplicated labour, and gave birth to a healthy child which she suckled for about a year, not only without detriment, but apparently with benefit to her health. In 1849 I first saw with the late Dr. Tanner a woman who, during her tenth pregnancy, had general dropsy with albuminuria. The labour was natural, the child was healthy, and was nursed for a time. After that, while the urine continued to be albuminous, the woman twice became pregnant, and had uncomplicated labours. She ultimately died with contracted granular fat kidneys. It is probable that the existence of albuminuria from any cause increases the risk of puerperal convulsions at the time when, to the exalted reflex excitability of the nervous system in the parturient woman, there is superadded the disturbing element of violent uterine and abdominal muscular contraction; but cases like those which I have cited show that, when there is a free secretion of urine, with absence of uræmic symptoms, labour may be unattended with any serious complication.

2. In a second class of cases, during the later months of pregnancy, there is more or less general œdema, with headache and other nervous symptoms, not unfrequently culminating in convulsions, which may recur again and again. The urine is scanty, high-coloured, often turbid with urates, of high specific gravity, and contains a large amount of albumen. On microscopic examination it is found to contain small hyaline casts, with a few granular casts, but few or no epithelial casts. After delivery the urine quickly becomes copious, of pale colour, of low specific gravity, and within forty-eight hours the albumen may have entirely disappeared. Such a case I saw in the year 1857 with Dr. Greenhalgh and Mr. Peter Marshall. The most probable explanation of this class of cases is, that the pressure of the gravid uterus on the

vena cava causes gradually increasing passive engorgement of the kidney, albuminuria, a scanty secretion of urine, dropsy, and at length uræmic convulsions. The rapid disappearance of the albuminuria and the other symptoms, after the emptying of the uterus, is explicable on no other theory than that of passive renal congestion consequent on mechanical pressure. Cases of this class are more common in primiparæ, probably for the reason that in first pregnancies the abdominal walls are less yielding; there is, therefore, greater tension and greater pressure on the large venous trunks than during subsequent pregnancies, when the abdominal walls are more flaccid.

3. There is a third class of cases, in which the theory of mechanical pressure is not admissible. I allude to those cases in which albuminuria comes on at an early period of pregnancy, before the uterus has attained sufficient size and weight to interfere mechanically with the circulation through the kidney. In these cases there is sometimes evidence of acute desquamative nephritis. The urine is not only scanty and highly albuminous, but often blood-tinged, and contains epithelial and blood casts. When albuminuria sets in during the progress of pregnancy, it is very apt to lead on to convulsions, to retinal hæmorrhage and albuminuric retinitis, with serious defect of vision. The renal symptoms may gradually pass away after delivery; if so, they may or may not return with the next pregnancy. In other cases the albuminuria is persistent; the urine is of pale colour, of low specific gravity, and deposits small hyaline and granular casts. Ultimately, uræmic symptoms occur; and, after death, the kidneys are found either contracted and granular, or large and pale. A painful case of this kind I saw some years since with Mr. Cadge, of Norwich. The most probable explanation of this class of cases is that which refers the renal disease to some previous blood-change. Obviously, pregnant women are exposed to the ordinary exciting causes of renal disease; and acute Bright's disease originating during pregnancy may result from exposure to cold and wet, from excessive eating and drinking, or from some zymotic blood-poison. But, in addition to these more common causes of albuminuria, it is

probable that, connected with the evolution of the uterus and the development and growth of the fœtus, there may sometimes be associated abnormal blood-changes, resulting in renal disease with albuminuria. In addition to other indications of the occasional occurrence of morbid states of blood in pregnant women, I may refer to those cases in which puerperal chorea is associated with acute endocarditis and fibrinous deposits in the mitral or aortic valves; the chorea being, probably, a result of capillary embolism in the region of one or both corpora striata.

4. There is yet a fourth class of cases, of which I have seen and noted several examples. I refer now to cases in which albuminuria and other symptoms of renal disease appear for the first time soon *after delivery*. Within a day or two after delivery, or after an interval of several days, sometimes after imprudent exposure to cold, a rigor occurs, and is followed by febrile symptoms. The urine is soon found to be scanty, with all the characters indicative of acute desquamative nephritis. There may be general dropsy, with or without uræmic nervous symptoms, such as headache and convulsions. The renal symptoms, after a period varying from a few weeks to several months, may gradually and entirely pass away; or the disease may become chronic and result in a large white kidney.

In such cases as this, the renal symptoms may with confidence be referred to the blood-contamination, consequent on absorption of morbid materials from the interior of the uterus after parturition. These cases are pathologically allied to, and sometimes associated with, a form of septicæmic puerperal fever. An interesting case of this kind, about which I was consulted, was published by Messrs. Melland and Windsor, of Manchester, in the *British Medical Journal*, September 12, 1847. Here, too, I would suggest the probability that, when the fœtus dies and is retained *in utero* until decomposition has commenced, there may sometimes be an absorption of foul gases and liquids, which in one case may give rise to the phenomena of *ante-partum* phlegmasia dolens, as occurred in one of my hospital patients; while, in other

instances, acute desquamative nephritis may result from this source of blood-infection.

It will be obvious that the distinction between the four classes of cases of albuminuria in connection with pregnancy, which I have here briefly indicated, is of considerable practical importance; inasmuch as upon an exact diagnosis depends, not only the prognosis, but the treatment of each case of puerperal albuminuria.

One hint I may give you with reference to the expediency of allowing a woman suffering from albuminuria to nurse her infant. The case of Dr. Steel's patient, to which I just now referred, shows that, when there are no symptoms of blood-poisoning, the mere fact of albuminuria does not prevent a woman from being a good nurse; but, on the other hand, when albuminuria is a result of recent blood-infection, the mother's milk may become contaminated, and act as a poison to her infant. In one case about which I was consulted, a lady had acute renal disease, resulting probably from *post-partum* absorption from the interior of the uterus. She recovered after a long illness, complicated with pelvic cellulitis and abscess; but the child, after taking the breast for five weeks, became feverish, and died with symptoms of septicæmia, at the age of six weeks. It seemed probable that the infant's illness was a result of infection through the milk; the infection being not uræmic, but septicæmic—a consequence of the absorption of noxious uterine discharges. In cases similar to this, the mother should not be permitted to nurse her child.

IV. *Atrophy and Suppurative Inflammation of the Kidney from Retention of Urine.*—The effect upon the kidney of retention of urine varies according to the nature and seat of the impediment. It differs, too, according as the obstruction occurs gradually or suddenly. One of the most frequent causes of renal disease, consequent upon retention of urine, is stricture of the urethra. The urinary organs behind the stricture undergo changes of structure, in proportion to the degree and the duration of the obstruction. The canal of the urethra on the vesical side of the stricture becomes dilated; its mucous membrane is frequently inflamed, and secretes

pus. The muscular coats of the bladder become thickened by hypertrophy, and its mucous membrane often inflamed and sacculated. The obstruction then affects the ureters, one or both of which may have their canals dilated and their walls thickened; and at length the natural cavities of the kidney—the pelvis, infundibula, and calyces—undergo the same process of dilatation. The medullary cones become flattened out by the pressure of the retained urine. The cortical substance of the gland is expanded, and presents lobed bulgings on its surface, which correspond with the original lobes of the embryonic kidney. The glandular tissue is squeezed between the distended interior cavity and the fibrous investing capsule; and the intertubular capillaries are compressed by dilated tubes. Thus the circulation is impeded, and the result is atrophy of the gland, which may, by degrees, be converted into a membranous cyst, all traces of glandular structure being lost.

It is but seldom that the kidney undergoes much dilatation without the occurrence of other structural changes. The mucous membrane of the dilated pelvis often presents irregular inflamed patches, and secretes a purulent liquid; and the apices of the medullary cones are frequently ulcerated. Then, as the mischief extends, inflammatory deposits occur in the substance of the kidney, and numerous small abscesses are scattered through the cortex. One or more of the abscesses on the surface may burst through the capsule, and then the kidney may be found imbedded in pus.

When retention of urine is the result of stricture, or enlarged prostate, or calculus with thickening of the walls of the bladder, or of atony of its muscular coats, both kidneys are usually affected simultaneously, but in different degrees; but when one ureter is obstructed by a calculus, or by a cancerous growth in the bladder, the structural changes are limited to the corresponding kidney.

The explanation of these changes is not difficult. The secreted urine is unable to escape, in consequence of the obstruction in front; there is, therefore, an accumulation, first in the ureter and pelvis of the kidney, and later within the uriniferous tubes. The tubes become distended by the retention of their own secretion, just as some tubes in the small red

granular kidney, having lost their lining of gland-cells, but continuing to secrete a serous liquid, become distended and dilated into cysts. The epithelial lining of the straight tubes is disintegrated and destroyed by the pressure of the retained urine; and at length some of the tubes in the cortex, whose basement-membrane is more delicate than that of the cones, give way, and allow their contents to become infiltrated amongst the intertubular capillaries. The infiltration of acid urine may cause the immediate formation of coagula within the capillaries, and, as a consequence of localised capillary and venous obstruction, irregular atrophic puckerings of the gland occur, somewhat similar to those which result from arterial embolism, to which I have before referred. But the escape of urine through the broken walls of the tubes may excite suppurative inflammation and abscess. A rapid cell-formation takes place between the tubes; and soon the glandular structure is disintegrated, and replaced by inflammatory products. The changes within and between the uriniferous tubes are a miniature representation of what happens on a larger scale, when a distended urethra gives way behind a stricture, and a perineal abscess results from the infiltration of urine into the submucous tissues.

Bear in mind that the changes within the substance of the kidneys are due to the retention and accumulation of the newly secreted acid urine within the tubes, and not to the regurgitation of fœtid ammoniacal urine, as has sometimes been suggested. Without doubt, the urine in the bladder in case of old stricture, vesical calculus, and cystitis, is often fœtid from decomposition; but the regurgitation of such urine into the uriniferous tubes is a physical impossibility. If you have ever attempted to inject the tubes from the pelvis of the kidney you will have found the task a very difficult one, in consequence of the resistance offered by the liquid and solid contents of the closed tubes. Obviously, then, during life, while streams of secreted urine are perpetually flowing through the tubes, it is impossible that urine from without can regurgitate into them. The tubes are dilated, and some of them ultimately ruptured, by the retention and accumulation of their contents, and not by the regurgitation of urine from the pelvis of the

kidney. The intertubular coagula and suppuration result from the infiltration of urine amongst the intertubular capillaries and veins.

The *symptoms* of renal disease, consequent upon an impeded escape of urine, are usually more or less masked by the diseased condition of other parts of the urinary organs. The mucous membrane of the bladder in cases of stricture, vesical calculus, or enlarged prostate, usually secretes pus; and there are no means by which this can be distinguished from matter derived from a suppurating kidney. For the suppurative process in the kidney rapidly destroys the tubular structure of the organ; the pus, therefore, is not moulded within the tubes, and there is no microscopic evidence of the renal origin of the pus. Chemistry, again, affords no more assistance than the microscope. The urine, which contains pus, is always albuminous. The coagulability of the urine by heat and other tests is, therefore, no indication that the kidneys are implicated, except when the degree of coagulability is out of proportion to the amount of pus and, it may be, blood, mingled with the urine. Chemical analysis affords little practical aid in estimating the efficiency of the kidney. The urine is usually fœtid and alkaline, and much of the urea is decomposed into carbonate of ammonia while the urine is still in the bladder. A low specific gravity of the urine with a scanty secretion would be a suspicious condition, and especially so when associated with indications of uræmia, such as drowsiness, headache, vomiting, and a brown dry tongue, with an excess of urea in the blood. Pain and tenderness in the region of one or both kidneys may be severe when, with sudden retention of urine, there is a great distension of the cavity of the kidney; but, in cases of long-continued and slowly-increasing obstruction, these symptoms bear no proportion to the amount of structural change in the kidneys; and it sometimes happens that the first indication of serious renal disease is afforded by the occurrence of alarming symptoms of uræmic poisoning, quickly passing on to fatal typhoid collapse and coma, with a low temperature. The cases are few in which the kidneys are sufficiently enlarged by distension and expansion, to form a palpable tumour in the lumbar region.

V. *Acute Cystitis simulating Acute Bright's Disease.*—It has happened to me to meet with a considerable number of cases of acute inflammation of the mucous membrane of the bladder, unconnected with stone, stricture, or gonorrhœa, which, in consequence of the urine being blood-tinged and albuminous, have been mistaken for cases of acute Bright's disease. Therefore, before I proceed to describe the treatment of Bright's disease, I think it well to point out to you the distinctive features of acute cystitis. Remember that I exclude from our present consideration such obvious and common cases as cystitis from stone, stricture, retention of urine, and gonorrhœa; and I refer to cystitis not excited by any obvious mechanical cause. In a large proportion of cases which I have seen, the disease directly followed, and was probably caused by, a chill. In some cases, dyspepsia, with rheumatic or gouty symptoms, had preceded the cystitis. In one, the disease came on after feasting, with excess of wine. In one case—that of a physician—the symptoms commenced within a few hours after he had been impressed by a peculiar odour from the throat of a boy whom he was attending with a low form of scarlet fever. It is probable that, in all these patients, the immediate cause of the cystitis was some irritating material in the urine. In the case last mentioned, some poisonous product may have entered the circulation, and passed out through the kidneys, exciting no disease in them, but setting up inflammation in the bladder. It is difficult to explain or to understand how it happens that exposure to cold should in one person excite acute cystitis, and in another acute desquamative nephritis. The disease, according to my experience, is about twice as frequent in males as in females. The ages of my patients ranged from seventeen in a female to sixty-nine in a male. In some cases there has been more or less of vesical irritation for a few days before the acute attack, but in most instances the onset has been sudden and severe.

The chief symptoms are frequent micturition, with more or less of uneasiness or pain in the region of the bladder. The calls to pass urine may occur every half-hour, or even oftener; and micturition is usually attended with an increase of pain and a sense of scalding in the neck of the bladder. The

vesical irritation is increased by exercise, by exposure to cold, and by alcoholic liquors. The urine quickly becomes turbid with puriform mucus, and it is often blood-tinged. In one case there was a puriform discharge from the urethra as well as from the bladder, though the disease was certainly not the result of gonorrhœa. Usually the urine has an acid reaction; but, if there be much admixture of blood, the acidity is lessened by the alkali of the blood. It contains an abundance of albumen, partly derived from the puriform secretion, partly from the blood. On a microscopic examination, pus-cells and blood-corpuscles are seen in abundance, but no tube-casts. Although the local symptoms are distressing, there is little or no constitutional disturbance. The nights are disturbed by frequent calls to micturate, and the broken rest is attended with a sense of fatigue and nervous exhaustion; but there is little fever, and no vomiting. If, within a few days from the onset of the symptoms, the patient be subjected to appropriate treatment, the disease usually subsides as rapidly as it came on. If, on the other hand, the symptoms be negligently or erroneously treated, the disease may become chronic, and cause prolonged and severe suffering. The urine becomes alkaline, ammoniacal, and fœtid; there are perpetual pain and annoyance; and ultimately the disease may extend backwards through the ureters to the kidneys, and so set up a fatal pyelo-nephritis.

Diagnosis.—The distinction between acute cystitis and acute Bright's disease is sufficiently obvious, if you bear in mind that the local symptoms are all referable to the bladder, while dropsy, vomiting, and other renal symptoms are absent. The urine is usually secreted in normal quantity, and of normal specific gravity. It is albuminous only in direct proportion to the amount of blood and pus which it contains, and the most careful microscopic examination discovers no tube-casts.

I shall have something to say of the treatment of acute cystitis in a future chapter.

SECTION IX.

THE TREATMENT OF ACUTE AND CHRONIC BRIGHT'S DISEASE.

Acute Bright's Disease—Rest in Bed—Uniform Temperature—Milk Diet—Case in which an Exclusive Milk Diet was continued for Five Years—Caution as to Use of Diuretics—Warm Baths—Warm Wet Pack—Pilocarpine—Objections to its Use—Purgatives—Rules for their Use—Local Bleeding—Hot Fomentations—Dry Cupping—Venesection—Case of Recovery after Seven Years' persistent Albuminuria—Chalybeates—Change of Air. *Chronic Bright's Disease*—Ascertain and avoid the Cause—Cold, Alcoholic Stimulants, and Over-feeding to be avoided—For Dropsy, Hot Baths—Diuretics—Hydragogue Purgatives—Incisions in the Legs—For Anæmia, Preparations of Iron—For Syphilis, Iodide of Potassium—Cod-liver Oil—For Dyspnœa, various Remedies to meet the various Causes—For Renal Asthma, Ether—Brandy—Chloral—Nitrite of Amyl and Nitro-glycerine to be given with Caution—For Muscular Twitchings and Cramps, Bromides with Chloral, Opium in Exceptional Cases—Hydriodate of Hyoscin—For Dyspepsia, Strychnine with Hydrochloric Acid—For Vomiting, Ice, Chloroform Inhalation, Food and Chloral per Rectum—For Albuminuric Retinitis, Leeches, Diaphoretics, &c.

I have endeavoured to prove to you that the various forms of Bright's disease are results of the physiological excretory function of the kidney. The kidney is one of the main channels by which effete and noxious materials are cast out of the circulation. During the process of excreting abnormal products, the tissues of the kidney—primarily the gland-cells, secondarily the blood-vessels and the connective tissue—undergo structural changes. It follows from this interpretation of the pathological changes in the kidney, that a leading principle of treatment is to lessen as much as possible the excretory work of the kidney, by instructing the patient to avoid the exciting causes of his malady, by a carefully regulated diet, and by such remedial agencies as experience has proved to be beneficial.

In all cases of *acute Bright's disease*, whatever may have been the exciting cause, rest in bed and in a room of moderate uniform temperature, well ventilated, but without chilling currents of cold air, is an essential part of the treatment. In a large proportion of cases, rest in bed, with a scanty diet and a liberal use of diluent drinks, will suffice for the cure. A

convincing proof and illustration of the effect of exercise, food, and cold, upon the amount of albumen in the urine, is afforded by the fact that, in most cases of albuminuria, the urine passed after rest in bed and before breakfast, contains much less albumen than that which is secreted after exercise in the open air, and after an ordinary meal.

The diet may consist of milk alone, if milk do not disagree, as it does with some patients. Milk is especially suitable for children; and it serves both for meat and drink, so that no other food or liquid need be taken. It may be taken cold or tepid, from half a pint to a pint at a time. An adult will take sometimes as much as a gallon in the twenty-four hours. Children will take less, in proportion to their ages. If the cream disagree, causing heartburn, diarrhœa, headache, or other symptoms of dyspepsia, the milk may be given skimmed. One reason, amongst others, for giving the milk as a rule unskimmed—that is, with the cream—is, that constipation, which is one of the most troublesome results of an exclusively milk diet, is to some extent obviated by the cream in the unskimmed milk. The advantage of milk as a main article of diet is that, as a rule, it is easy of digestion; and that, taken freely, it supplies an abundance of liquid, which, by its diluent action, has a diuretic influence, and so favours the removal of the dropsy. There are some patients with whom, unfortunately, milk in any form, even in small quantities, so decidedly disagrees, that we have to find a substitute in beef-tea, chicken-, veal-, or mutton-broth, with an egg or two, and some farinaceous addition, such as barley-water, arrowroot, rice, or sago, or a small quantity of bread. Under this regimen, adopted and rigidly carried out at the very commencement of acute Bright's disease, the urine soon becomes copious, while the albumen diminishes and gradually disappears, and the dropsy is quickly removed.

In some cases, under the influence of an exclusive milk diet, albuminuria, even of long duration, speedily passes away.

Some years since a lady from Scotland had had albuminuria for fifteen months, commencing after an attack of scarlet fever. She had been under medical care all the time, and was treated very judiciously, except that her diet had not been

regulated, and she had been allowed to take wine. There was a good deal of albumen in the urine, with a considerable amount of œdema, and shortness of breath. I advised her to try a milk diet alone; and in the course of a very few days the albumen almost entirely disappeared. Her medical attendant in Scotland had thought it better for her to spend the rest of the winter in a southern climate; and she had made arrangements to go with her husband and other relatives to Malta. I therefore lost sight of her after a few days, and the milk treatment was necessarily interrupted on board the steamer; but I taught her to test her urine with nitric acid; all that is required being to drop a few drops of nitric acid into the cold urine in a test-tube. It is sometimes important that the patients should be able to test their own urine, and thus to ascertain the influence of diet upon it. When she got to Malta, she immediately resumed the exclusive milk diet, and found in less than a week that the albumen had disappeared, though there had been some albumen when she landed. As she and her husband were not quite satisfied with her testing, they asked an Italian doctor to see her; and he sent me a note to the effect that he found the urine entirely free from albumen. This was a very rapid recovery after the continuance of albumen for fifteen months.

In illustration of the length of time during which a patient may be well nourished on an exclusive milk diet, I may briefly mention the case of a gentleman who was 55 years of age in June, 1873, when he first consulted me for chronic gouty albuminuria, the result of a too generous mode of living. In October of that year I advised him to try an exclusive milk diet. This he continued for more than five years, taking about a gallon of skimmed milk daily. The milk yielded by his own cows in the country was so rich that, unless some of the cream was taken off, he became too stout. On this simple diet his health greatly improved, and he frequently reported himself as feeling quite well, though a trace of albumen still remained. After five years on milk diet he gradually went back to solid food without alcoholic stimulants, and I have heard of him quite recently as remaining in good health.

I frequently quote this case to patients or to their friends,

who appear to think that an exclusive milk diet, even for a short time, is little better than starvation.

In section iv. p. 664 I have given the physiological explanation of the copious flow of urine which usually occurs during convalescence from acute Bright's disease, and especially when there has been a copious dropsical effusion. This abundant flow of urine usually occurs without aid from diuretic drugs, or, indeed, from drugs of any kind. Stimulating diuretics, such as squills, cantharides, or turpentine, would be injurious by increasing congestion of the kidney. The best means of promoting diuresis in cases of acute Bright's disease are those which tend to lessen congestion of the kidney, such as dry cupping or hot poultices or fomentations over the loins, warm baths, and a free use of diluent drinks, one of the pleasantest and most efficacious being the 'imperial drink,' made with cream of tartar and lemon.

In the later stages of an acute attack, when the dropsy has disappeared, the urine being normal in colour, quantity, and specific gravity, but still more or less albuminous, a too free use of diluent drinks may be injurious by diluting the gastric secretions and so impeding digestion; with the result of increasing the excretory work of the kidney, and so retarding convalescence.

Warm baths are particularly useful in the early stage of an acute attack, and more especially when exposure to cold has been the exciting cause of the renal disease. A warm-water bath, at a temperature of 100° F., may be given every night during the first few days of an acute attack; or a hot-air lamp-bath; or, what I believe in most cases to be still more efficacious, a wet sheet and blanket bath. A sheet is wrung out of warm water, and the patient, either naked or covered only by his shirt, is enveloped in the wet sheet up to the neck. Then three or four dry blankets are closely folded over the wet sheet. He may remain thus packed from two to four or six hours, or even longer. Some time since a boy in the hospital with acute renal disease and almost complete suppression of urine, consequent on scarlet fever, was kept packed incessantly for four days without serious discomfort, and with great relief from very distressing and alarming symptoms.

When he left the hospital all traces of his malady had disappeared. If the packing be long continued the sheet has to be re-wetted as soon as it becomes dry. The evaporation and consequent drying of the sheet will be slow in proportion to the closeness of the blanket packing. If the blanket next to the wet sheet be covered by a macintosh cloth, the sheet remains wet for a much longer time than when no waterproof covering is used; but patients often complain of a feeling of oppression when surrounded by the impervious macintosh. The advantage of the blanket bath over a warm-water or hot-air bath is that it requires no special apparatus, that the diaphoretic action may be more prolonged, and that in most cases it is more agreeable to the patient. The hot-air bath not unfrequently causes an unpleasant throbbing in the head, or a feeling of exhaustion and even faintness. When the wet pack is removed the patient should be quickly rubbed dry, and enveloped in dry blankets.

The diaphoretic action of any form of warm bath is assisted by copious libations of simple diluent drinks, and it may also be aided by the internal administration of the solution of acetate of ammonia. It has been objected, on theoretical grounds, that to promote perspiration in these cases is injurious, by diverting to the skin the water which is required to wash out the uriniferous tubes. To meet this objection you have only to bear in mind that the dropsical patient is oppressed by an excess of water, which has been poured into the areolar tissue and the serous cavities, in consequence of defective urinary secretion, resulting from the inflammatory engorgement and obstruction of the kidneys. If, therefore, by the relaxing effect of external warmth you divert a large amount of blood to the surface, you thereby lessen the congestion of the kidney, increase the freedom of the renal circulation, and so favour the occurrence of that copious secretion of urine which is one of the surest signs of satisfactory progress, and by which the uriniferous tubes may be effectually flushed and cleansed. Whatever fluid is lost by perspiration may be quickly restored by the liberal use of diluent drinks, which again assist the secretory activity of both the skin and the kidneys. In fact, one of the main objects of treatment is to increase the freedom

of the circulation, more especially through the kidneys, and thus to get rid of the excess of stagnant water which has accumulated in consequence of defective action of the skin and kidneys.

Pilocarpine has been recommended as a diaphoretic in the treatment of Bright's disease. In the few cases in which I have tried this medicine, the salivation and depression have been so distressing that the patient has been unwilling and, indeed, unable to endure the continuance of the treatment.

Purgatives may be usefully combined with other means for lessening dropsical effusion. In ordinary cases of acute Bright's disease I do not, as a rule, advise the frequent employment of drastic purgatives. I reserve this method of treatment for cases in which there is an excessive and increasing dropsical effusion which does not yield to other means of cure, but more especially for cases in which cerebral symptoms, the result of uræmia, are either present or apparently impending. Dr. Abercrombie and others, who wrote on brain-disease before Dr. Bright's discovery had led on to our present knowledge of uræmic nervous symptoms, published cases of cerebral disease which they took to be of an inflammatory or an apoplectic character, in which the most strikingly beneficial results were obtained by free purging. Dr. Abercrombie, in discussing the treatment of inflammatory affections of the brain,[1] states that, according to his own experience, 'more recoveries from head affections of the most alarming aspect take place under the use of very strong purgatives than under any other mode of treatment.' Our more recent experience is entirely confirmatory of Dr. Abercrombie's statement; but our improved pathology enables us to add a very important qualification—namely, that most of the cases in which formidable cerebral symptoms have been removed by the action of strong purgatives have been neither inflammatory nor apoplectic in their nature, but cases in which brain symptoms have resulted from blood-poisoning, and in the majority of instances the poison has been uræmic. Amongst the cases recorded

[1] *Pathological and Practical Researches on Diseases of the Brain*, 3rd ed. p. 153.

by Dr. Abercrombie as examples of inflammation of the brain successfully treated, there are two (Cases LXX. and LXXX.) in which the brain symptoms were associated with anasarca, which had followed an attack of scarlet fever. These were unquestionably cases of acute renal disease, with cerebral symptoms of uræmic origin.

I offer for your practical guidance this rule of treatment: when such symptoms as headache, delirium, convulsions, or coma are the result of uræmia, give purgatives freely; and, if the renal disease be acute, and therefore probably curable, your treatment will often be completely successful. On the other hand, when you have reason to believe that similar brain symptoms are consequent on cerebral hæmorrhage, or embolism, or thrombosis, be very cautious in the use of purgatives; which may greatly increase the patient's distress and exhaustion, while they can do little to improve his condition. In inflammatory affections of the brain and its membranes, purgatives are often useful, but less frequently and strikingly so than when cerebral symptoms are the result of uræmia. As to the form of purgative in uræmic cases, croton oil is the most convenient, when there is coma and consequent difficulty in swallowing a more bulky dose. When there is no such difficulty, two pills, composed of three grains of calomel with seven grains of compound colocynth pill, may be followed in four hours by an ounce of the compound senna mixture; or the following powder, which I think an improvement on the compound jalap powder, may be given: ℞ Scammonii resinæ, gr. v. to viij.; potassii tartratis acidi, ℈j.; pulveris zingiberis, gr. iij. The dose to be repeated once, or oftener, according to circumstances.

When, in a case of acute Bright's disease, the renal congestion is excessive, as shown by the scanty secretion of highly albuminous urine, with vomiting, headache, and other threatening nervous symptoms, local bleeding by leeches or cupping on the loins is often extremely useful, and is quickly followed by an increased secretion of urine. If, by the abstraction of a few ounces of blood from the loins, we relieve the renal congestion, we thereby lessen the destruction of blood-constituents which results from contamination of the blood by urinary

products. Moderate and timely local bleeding, therefore, tends to economise blood, and to prevent its waste.

The manner in which local bleeding by cupping or leeching the loins relieves a congested or inflamed kidney has been explained in Chapter V. (p. 62).

As a rule, I prescribe local bleeding only when, the secretion of urine being extremely scanty, there is a consequent threatening of head symptoms or other serious results of uræmia. In ordinary cases, I apply hot fomentations or poultices, covered by macintosh, to the loins. These act by relaxing the superficial arteries. The skin, therefore, receives a larger supply of blood, and thus a portion of the blood is diverted from the renal vessels. Then, too, there is some degree of depletion from the full cutaneous capillaries by the free local sweating which the warmth occasions.

Dry cupping acts in a somewhat similar way to hot fomentation. It draws an abundance of blood through the arteries into the subcutaneous capillaries, which, when the cups are removed, returns through the veins to the heart.[1] In some cases of scanty, almost suppressed, secretion of urine, with high arterial tension and grave cerebral symptoms, such as recurring convulsions or profound uræmic coma, venesection has afforded great and immediate relief.

As a rule, it is well to give no alcoholic stimulants; or, if need be, to give them very sparingly in cases of acute Bright's disease. The imbibition of alcohol imposes extra work on the kidney, and so is opposed to the principle of lessening as much as possible the excretory function of the inflamed gland. Excess of alcohol is, amongst the lower classes, one of the most frequent causes of albuminuria; and a very moderate employment of alcohol may tend to perpetuate and aggravate disease originating from other causes.

When acute Bright's disease is making satisfactory progress towards recovery, the dropsy usually disappears for a variable time before the urine ceases to be albuminous. It is very important to impress upon the patient that, until the urine has regained its normal characters, he should be warmly clothed

[1] See *ante*, p. 66.

with woollen next the skin; and he must be extremely careful to avoid cold, fatigue, and errors of diet.

The duration of albuminuria in cases that ultimately recover is very variable. I have seen many cases of recovery after the disease had continued from three to twelve months, and I have seen some recoveries after the urine had been albuminous for one, two, three, and in one case even seven years.

The most satisfactory case of recovery after a long duration of albuminuria was that of a very distinguished medical graduate of London, who, when he consulted me in November 1877, was twenty-six years of age. His urine had been continuously albuminous after food since an attack of scarlet fever in June 1871. In spite of the anxiety which this symtom had occasioned, his general health had been good, and he had worked hard and obtained the highest honours at the university. I advised him to place his main reliance upon a carefully regulated diet. Under the influence of an exclusive milk diet for five or six weeks, the albumen had much diminished. He then took a small quantity of solid food; and, after a time, a two-ounce glass of Hunyadi water every morning, which, acting rather freely on the bowels, relieved him of a dull pain before felt in the region of the liver, and still further reduced the amount of albumen. During the month of July 1878, the albumen disappeared; and there has been no recurrence of the symptom. In this case, then, albuminuria of seven years' duration has been completely removed, and I have lately seen my former patient in perfect health. The complete recovery of health after so long a continuance of the symptoms may afford encouragement to those who are engaged in the treatment of these troublesome and anxious cases, the ultimate result of which must for a time be more or less doubtful; but the more I have seen of the disease, the more hopeful I have become as to the effect of treatment, when the history and the symptoms, and, above all, the chemical and microscopical characters of the urine, do not indicate extensive and irremediable degeneration of the kidney.

In most of the cases of recovery from long-continued albuminuria, the preparations of iron have entered largely into the

medicinal treatment of the disease, and have apparently contributed much to the favourable result. There are two preparations which I have found especially useful; these are the tincture of the perchloride and the syrup of the phosphate—the former in doses of from ten minims to half a drachm, and the latter in drachm doses twice or thrice daily. Fellows' syrup. hypophosp. co. in drachm doses is also an easily assimilated and useful preparation. The preparations of iron are best taken soon after food. I have frequently combined with each dose of the perchloride of iron ten grains of hydrochlorate of ammonia; and I believe that this ammonio-chloride of iron is a useful combination. The preparations of iron should not generally be given after the use of tea as a beverage, for the reason that the resulting tannate of iron is apt to irritate the stomach, and is less readily absorbed. In some cases it is well to omit the iron every week, for a day or two, the patient being thus enabled to continue the medicine longer, and to utilise it more completely. If the full doses of iron which I have recommended are not well borne, smaller doses should be cautiously tried. It is especially important to bear in mind that any medicine which causes disorder of the digestive organs will be injurious rather than beneficial. And amongst such medicines are to be included drastic purgatives, given too frequently and without sufficient reason. Purgatives are often given with the idea of reducing the work of the kidneys, but the effect often is to impair the digestive powers, with the result that the food which is taken, not being completely digested, adds to the irritation of the kidney. The kidney is one of the channels by which imperfectly digested materials are excreted, and the practical result of drastic purgatives may be, by irritating the stomach and impairing digestion, to increase rather than to lessen the work of the kidney.

Amongst other remedial agencies, when acute renal disease is prolonged and threatens to become chronic, change of air and scene is often highly beneficial. Residence during the winter season in a warm, dry, equable climate, such as may be found at Cannes, Nice, Mentone, and Algiers, has in many instances been attended with highly beneficial results. The bright warm sun and dry invigorating air favour the action

of the skin and of the bronchial mucous membrane; the patient is able to be much in the open air, and thus the respiratory, the digestive, and the secretory functions are all assisted and promoted. I have seen some most remarkable recoveries effected under the influence of *a long voyage* after other means had failed to effect a cure.

The treatment of *chronic* Bright's disease must obviously vary according to the form and stage of the malady and the nature of the secondary complications. In each case, it is practically important to ascertain, if possible, the probable cause of the renal disease. Your inquiries should be directed to determine whether the chronic malady is a sequel of an acute attack, or whether it commenced as an insidious chronic disease. Then inquiry should be made as to the exciting cause, which in most cases may be arrived at with a high degree of probability. Is there a history of gout, or of habits likely to induce a gouty diathesis? Excessive eating and drinking, chronic dyspepsia, frequent exposure to cold and wet, cachexia the result of syphilis or of other constitutional disease, scrofula or other hereditary taint, and chronic lead-poisoning, are amongst the probable determining causes which should be made the subject of inquiry; and then the treatment should be directed to remove, if possible, or, so far as may be, to counteract, the morbific influence.

Whatever may be the form or the stage of chronic Bright's disease, the skin should be protected from cold by warm woollen clothing, care must be taken to avoid over-fatigue, and the diet should be carefully regulated both as to quantity and quality. As a rule, in all cases of chronic renal disease, alcoholic stimulants in any form should be given sparingly, or abstained from entirely, unless, for some special reason, they appear to be indicated. You will find that, when there is extensive degeneration of the kidney, alcoholic liquors usually produce far more decided, and often deleterious effects, than result from equal quantities of the same liquors when the kidneys retain their normal structure and functional activity.

One of the commonest mistakes in the treatment of Bright's disease is that of over-feeding a patient. Some practitioners

appear to have an idea that, inasmuch as a large amount of albumen is being drained off from the kidneys, they must supply this waste of material by prescribing a liberal dietary. This is a very great mistake. You will find in almost all cases of Bright's disease that the albumen is more copious after food than before a meal. You may sometimes find the urine almost, and even quite, free from albumen before breakfast, but highly albuminous after that meal.

I have often met with cases in which the mistake of overfeeding has been made. Some years since a manservant came to me, suffering very seriously from acute renal disease, and having had an epileptiform uræmic convulsion. This man was valet to a rich invalid gentleman, who allowed him anything he chose to take in the way of food and drink; and he had chosen to eat very largely and to imbibe a considerable amount of wine and spirits. This excess of food and alcohol had, no doubt, been the cause of his kidney disease. He went to a medical man, who told him that, as he was losing much albumen, he must take as much solid food and eat as many eggs as he could consume, and that he might go on with his liberal allowance of whisky. Now, that is an illustration of what I consider a great mistake in theory and practice. I persuaded this man to live entirely upon milk, and I stopped his whisky; and the result was that he got very rapidly well. If he had continued the excessive feeding and stimulation, no doubt he would have had incurable degeneration of the kidney. It is an error to suppose that the fatality of kidney disease depends, generally, upon the drain of albumen from the blood. It is the disorganisation of the kidney and the consequent retention of poisonous materials in the system that destroys the patient, and not the loss of albumen.

In one class of cases—cases of large white kidney, with a scanty secretion of highly albuminous urine—*dropsy* is usually a prominent symptom, and requires special treatment. The tendency to dropsy is no doubt increased by the dry and inactive state of the skin, which often resists the relaxing effect of external warmth, so that a hot-air bath, or even the hottest room of a Turkish bath, fails to excite diaphoresis.

Patients who do not perspire under the influence of the hot-air bath, usually complain of painful throbbing in the head, difficult breathing, and other distressing symptoms. On this account, I prefer in most cases the wet sheet and blanket bath, which, as a diaphoretic, is both more agreeable and more efficacious than the hot-air bath.

Diuretics are notoriously uncertain in their operation. In order to assist the action of diuretics, diluents should be freely given; and I have often obtained most satisfactory results by keeping the patient entirely on milk, either skimmed or unskimmed, in accordance with the rules which I have before laid down, with the addition of a mixture containing acetate of potassium and infusion of digitalis, a drachm of each for a dose, to be repeated three times a day. By these means, a copious secretion of urine is often induced, and the dropsy is speedily and completely removed. A strong infusion of fresh broom-tops, taken in sufficient quantity every morning to act as a purgative, often proves a very efficient diuretic. The *succus scoparii* of the Pharmacopœia may be substituted for the fresh infusion. The imperial drink (cream of tartar and lemon), in doses of from two to four pints in the twenty-four hours, is also a pleasant and efficacious diuretic. The late Sir James Simpson was the first to use the vapour of oil of juniper as a diuretic. Thirty or forty drops of the oil may be floated on boiling water in an ordinary jug or in a suitable inhaler; and the mixed vapours of the volatile oil and water may be thus inhaled twice a day. In some cases, the diuretic action is very prompt and decided. Another plan, originally proposed by Dr. Christison, is to apply digitalis freely to the skin. Dr. Christison's plan consists in making a strong infusion by adding an ounce of the dried digitalis leaves to a pint of boiling water. A large piece of spongio-piline, steeped in the infusion, is kept constantly applied to the abdomen. An alternative plan is to pour an ounce of tincture of digitalis on the surface of a large hot linseed poultice, which is then applied over the loins and back; the poultice, with the tincture of digitalis, being renewed two or three times a day. This plan sometimes succeeds when other methods have failed to remove the dropsy.

The free action of a hydragogue purgative, such as elaterium, compound gamboge pill, compound jalap powder, or the powder composed of scammony resin, cream of tartar, and ginger, which I have before mentioned, is often followed by a more copious secretion of urine. The probable explanation of the indirect diuretic action of a hydragogue purgative is this. The purgative excites a copious watery secretion from the blood into the bowel; this is followed by the absorption of a portion of the dropsical fluid which had been effused into the areolar tissue, and perhaps into one or more serous cavities. The partial absorption of the dropsical effusion removes or lessens the pressure on the vessels, more especially on the veins; and so the circulation becomes more free, at the same time that the absorbed liquid exerts a diuretic influence on the kidneys similar to that which we have seen to occur during convalescence from acute renal dropsy, and not unlike that which often results from the introduction of abundant diluents through the stomach.

When other means fail to remove the dropsy, when the anasarcous distension of the legs is increasing and causing pain and incipient erythematous inflammation, or when the breathing is becoming impeded by the accumulation of water within the abdomen or the chest, or by an œdematous condition of the lungs, prompt, decided, and sometimes permanent relief may be afforded by allowing the water to escape through an incision in the skin, about half an inch long, just above either the outer or the inner ankle of each leg. The incision must be deep enough to enter the areolar tissue beneath the skin. The best instrument for making the incision is a cupping scarifier with a single blade, which is made for the purpose by Messrs. Matthews, of Carey Street. The scarifier, driven by a spring, makes a clean cut through the skin with such rapidity that it causes little or no pain. We had lately a good opportunity of testing the comparative painlessness of an incision made by this instrument. A dropsical patient had his legs acupunctured by the house physician, and cried out with the pain caused by the needle punctures. A few days afterwards, the punctures having ceased to discharge, while the dropsical swelling was but little reduced, we made an incision into each

leg with the scarifier. He declared that he scarcely felt the cuts; and the incisions discharged so freely that the dropsy was, for a time, completely removed.

I have seen many cases in which life has been prolonged for a considerable period, and some in which a complete and permanent cure has followed incision of the legs, after other means had failed to afford relief. To refer to one case only out of a number: towards the end of July 1861, I first saw a clerk to the New River Company, aged 22. Since the end of March, he had suffered from general dropsy, the result of exposure to cold. The urine became nearly solid with heat and acid, and it contained numerous oily casts. Purgatives and diuretics failed to lessen the dropsy; and at the beginning of September the swelling of the legs was so great that the skin cracked, and fluid oozed through the fissures. I then advised that the legs should be incised. An abundant discharge of liquid occurred, and the urine then became more copious. From that time he steadily improved, the dropsy passed away, and gradually the urine ceased to be albuminous; but it was not until the end of April 1862, more than a year from the commencement of his illness, that all trace of albumen had disappeared. The chief medicinal treatment after the incision of the legs consisted in giving the tincture of perchloride of iron three times a day, and a dose of strong broom-tea every morning. Since his recovery he has insured his life, he has married, and has several children. I heard of him quite recently—that is, in 1873—as remaining well. I have thought this case worthy of especial mention, as an example of complete recovery, after dropsy to an extreme degree, and albuminuria with numerous oily casts, had continued for the greater part of a year; the first favourable change in the patient's condition following directly upon a copious discharge of water through incisions in the legs.

After the dropsical legs have been punctured, the folded sheet and macintosh, placed beneath to receive the serous discharge, should be frequently renewed and kept clean. The liquid quickly decomposes and becomes ammoniacal, and in this state it may irritate and inflame the skin. Cleanliness is, therefore, essential for safety as well as for comfort. Any

inflammatory redness about the wound may usually be removed quickly by the application of a lead lotion. It is true that severe inflammation and sloughing have sometimes followed incisions or punctures in anasarcous legs; but this may, and often does, occur from over-distension of the skin, or from the mere pressure of the heavy dropsical limbs upon the bed. The result of my experience is that inflammation of anasarcous legs has been as often subdued as provoked by acupuncture or incision; that inflammation is much less likely to follow incisions in cases of renal than of cardiac dropsy, when the circulation is much impeded by valvular disease; and that an incision made with the spring scarifier is as safe as acupuncture, and much less painful.

The copious secretion of urine which usually follows a discharge of dropsical fluid through incisions in the skin, admits of nearly the same explanation as that which I have already given of the like phenomenon, after the action of a hydragogue purgative. The escape of the dropsical effusion through one or more incisions in the skin removes pressure from the veins, and permits the blood to move more freely through the vessels. This greater freedom of the circulation is attended by a quickened absorption; and some of the absorbed dropsical liquid, more or less charged with urea, enters the circulation, and exerts a diuretic influence on the kidney. In the treatment of a copious dropsical effusion, the main object, and the chief difficulty, is to overcome the *vis inertiæ* of the stagnant liquid. If once we can set the liquid in motion, whether by a primary diuretic action upon the kidneys, by first exciting a free discharge of liquid through the bowels, or by giving exit to a portion of the liquid through incisions in the skin—in whichever way the current is started—the outward movement of liquid often continues until the whole of the dropsical effusion has been swept away, and, in each case, a free secretion of urine constitutes a part of the eliminative process.

The *anæmia* of chronic Bright's disease is to be counteracted by a carefully regulated diet, and by the persevering use of one or other of the preparations of iron. When with

anæmia there is a scanty secretion of urine, and a tendency to dropsy, a very useful combination is twenty minims of the tincture of perchloride of iron, with a drachm of spirit of nitrous ether, and a drachm of infusion of digitalis, or from ten to twenty minims of the tincture of digitalis in an ounce of water, given three times a day, soon after food. If the mixture be found to constipate, from half a drachm to a drachm of sulphate of magnesium may be added to each dose, or the bowels may be acted upon by an occasional dose of the compound colocynth pill. Mercury in any form often acts powerfully and injuriously in cases of Bright's disease. The chief use of mercurials in these cases is to assist the operation of saline or vegetable purgatives.

Syphilitic symptoms, when present, are best treated by gradually increasing doses of iodide of potassium, with bark, or quinine. The calomel vapour bath has been found a useful remedy in some syphilitic cases.

When chronic renal disease is associated with a *strumous diathesis*, cod-liver oil may often be given with advantage.

Dyspnœa is one of the most frequent and distressing symptoms associated with advanced Bright's disease. It has various causes, and requires various remedies. When it results from œdema of the lungs, or dropsical hydrothorax, it is best treated by the remedies for dropsy. In some cases, anæmia appears to be the chief cause of dyspnœa. The red blood-corpuscles are the oxygen-carriers. When the blood—whether in cases of chlorosis or of Bright's disease—contains an excess of water with a corresponding deficiency of red corpuscles, the defective oxidation of the tissues and the demand for air are manifested by hurried and laborious breathing. The remedy for this form of dyspnœa is to be sought for in the elimination of water, a carefully regulated nutritious diet, and iron as a restorative tonic.

When dyspnœa results from pulmonary congestion and bronchial catarrh, it is best treated by warm baths, fomentations, or poultices to the chest, and mild counter-irritants.

Paroxysmal dyspnœa, in many cases of advanced Bright's disease, appears to result from the influence of deteriorated and poisoned blood upon the nerves and the nervous centres.

It is sometimes spoken of as 'renal asthma.' The heart's action is rapid and feeble, the breathing is distressed and hurried, while the respiratory murmur is loud, puerile, and unattended by wheezing or crepitating sounds. This form of dyspnœa is usually more common and more distressing at night. It is not improbable that, in some cases, cardiac weakness, with palpitation and dyspnœa, may result from excessive contraction of the minute branches of the coronary arteries, excited by the stimulant action of morbid blood, and causing anæmia and malnutrition of the muscular walls of the heart. These symptoms are most effectually warded off by a carefully regulated diet, by promoting the action of the skin and bowels, and, so far as possible, of the kidneys; the object being to free the blood from accumulated impurities. Temporary relief is often afforded by ether, or by brandy. In some cases, a small dose of chloral hydrate, not more than ten grains, repeated at intervals of six or eight hours, is very beneficial. Chloral has this advantage over every preparation of opium, that it has no astringent action on the bowels, and that it rather increases than checks the secretion of the kidneys. It therefore never excites the distressing sickness which often results from the astringent influence of opiates in the advanced stages of renal degeneration.

The inhalation of nitrite of amyl sometimes affords temporary relief from the distress of uræmic dyspnœa; but generally the relief is of but short duration; and the same may be said of nitro-glycerine, which, in doses of one-hundredth of a minim, often relieves the intense headache which is a common result of uræmia.

That the temporary benefit resulting from the use of these remedies is due to their physiological action of relaxing the arterioles is possible, but by no means certain. It is well, however, to bear in mind that in the advanced stage of granular kidney, when there is high arterial tension and a corresponding greatly hypertrophied left ventricle, sudden and extreme dilatation of the arterioles might expose the *capillaries* of the brain and other organs to the risk of being ruptured by the forcible impulse from the heart.

The immediate effect of nitro-glycerine and nitrite of

amyl in lessening the high arterial tension in cases of advanced renal degeneration is one amongst the numerous facts confirmatory of the doctrine which explains the high tension by the contraction of the hypertrophied arterioles, but which are inconsistent with the theory of 'arterio-capillary fibrosis,' which assumes that the impeded circulation is the result of a form of senile degeneration of the coats of the blood-vessels, upon which the drugs in question could exert no influence.

The *muscular twitchings* and the *painful cramps* which are common results of uræmia, and not unfrequent precursors of convulsions, may sometimes be subdued, or much mitigated, by twenty-grain doses of bromide of potassium or ammonium, given twice or three times in the twenty-four hours. A combination of chloral hydrate with the bromides appears sometimes to have a powerful influence in warding off uræmic convulsions.

Although the use of *opium* in all forms and stages of Bright's disease requires extreme care, on account of its tendency to check all the secretions except that of the skin, yet you will occasionally meet with cases in which the distressing nervous symptoms resulting from uræmia are relieved by opiates more effectually than by any other means. There are cases in which the carefully observed result of a cautiously conducted experiment is a better guide than theory.

In the February number of the *Practitioner*, 1887, Dr. Tirard has given some particulars of a case of chronic Bright's disease, in which the subcutaneous injection of the hydriodate of hyoscin procured more refreshing sleep than had been before obtained by chloral. The solution was prepared by Mr. Martindale according to the formula of Dr. Mitchell Bruce[1]—℞ Hyoscinæ hydriodatis, gr. j.; aquæ destillatæ, ♏200. Of this at first two minims were injected subcutaneously, and afterwards one minim was found to give satisfactory results. This drug seems to deserve a careful trial in cases of uræmic insomnia.

The sufferers from Bright's disease are always *dyspeptics*, and the *gastric symptoms* are often very obstinate and distress-

[1] *Practitioner*, November 1886.

ing. When, in consequence of renal degeneration, the blood is contaminated by retained urinary products, there is often a vicarious excretion of these impurities by the mucous membrane of the stomach and bowels. The gastric secretions are mingled with the ammoniacal products of decomposing urea; digestion is consequently impaired; there are flatulent distension of the stomach and bowels, nausea, vomiting, and diarrhœa. Relief is to be sought by a carefully regulated diet, and by giving with the food from ten to twenty drops of dilute hydrochloric acid with a vegetable bitter. The liquor strychninæ in doses of three minims, or the tincture of nux vomica in ten-minim doses, with a mineral acid, is sometimes especially efficacious. Pepsine may sometimes be given with advantage.

In some cases of advanced renal degeneration, the *vomiting* is so incessant, that the patient has to be sustained by nutritive enemata, while iced water only, or iced milk in small quantity, is taken by the stomach. In some instances that have come under my observation, the straining and exhausting efforts of vomiting have been checked only by frequent whiffs of chloroform vapour.

When the stomach is very irritable, chloral can rarely be retained; it acts at once as an emetic; but, injected into the rectum, its soothing influence is very similar to that of chloroform vapour, while it has the advantage of producing a more durable impression on the nervous system, and therefore requires to be less frequently repeated.

When the retina is the seat of hæmorrhage, or of albuminuric retinitis, the eyes should be allowed to rest, and they should be carefully shaded from excess of light. This condition is always very grave and distressing, but recovery of sight, more or less complete, may occur, especially when the retinitis is a result of the albuminuria of pregnancy. In the early stage of engorgement with 'choked disc,' a leech or two applied to the temples may be beneficial.

In the case of an unmarried lady, aged 28, suffering from chronic albuminuria with what is probably a large white kidney, whom I saw last year with Dr. Sherrard, of Brighton, the sight was entirely lost from the rapid onset of bilateral albuminuric

retinitis with hæmorrhage; but very great improvement resulted from the persevering use of the wet pack and a combination of quinine and sulphate of iron, with sulphate of magnesium in sufficient doses to promote free action of the bowels. Linimentum iodi was also applied behind the ears.

In that case, before recourse was had to the wet pack, the hypodermic injection of pilocarpine was tried, but it caused so much distress that it had to be discontinued.

CHAPTER XLVI.

CLINICAL LECTURE ON THE CURATIVE INFLUENCE OF AN EXCLUSIVE MILK DIET IN SOME CASES OF INFLAMMATION OF THE BLADDER.[1]

Utility of an Exclusive Milk Diet in various Forms of Disease—Case of Chronic Diarrhœa and Dysentery—Acute Bright's Disease—Painful Cystitis of Nine Months' Duration—Recovery—Cystitis of Two Years' Duration—Recovery in a Fortnight—Cystitis of Three Months' Duration—Recovery in Three Weeks—Directions as to the Method of giving Milk—Copaiba for Chronic Catarrh of the Bladder.

GENTLEMEN,—In the wards of the hospital you have frequent opportunities of observing the beneficial influence of an exclusive milk diet in various forms of disease. You have seen some cases of chronic diarrhœa and dysentery rapidly and completely cured by this diet, without the aid of medicines. There is one such case now in Craven ward. E. J——, a sailor, aged 25, who got acute dysentery at Calcutta in June last, was admitted November 1, suffering from abdominal pain and frequent watery, slimy, blood-tinged stools. He was placed on milk alone, at first with no medicine. Improvement began at once, and he is now, in less than a month, nearly convalescent. You have seen that the diarrhœa of typhoid fever is often promptly checked by omitting beef-tea and eggs from the dietary, and feeding the patient for a few days upon milk alone. You have seen in numerous cases of acute Bright's disease (acute albuminuria) the speedy disappearance of the albuminuria under the influence of rest in bed, a few warm baths, and copious libations of milk. And not unfrequently you have seen that a too early addition of solid food to the dietary has been followed by the reappearance of

[1] *Lancet*, December 16, 1876.

albumen in the urine. A child now in Twining ward has afforded an instructive illustration of this principle:—

Caroline D——, aged 5, was admitted on Sept. 14, with acute general dropsy, following scarlet fever, her urine being blood-tinged and nearly solid with albumen. She was ordered to have a wet sheet and blanket pack daily, as much cold or tepid milk as she would drink, no other food, and at first no medicine. She improved rapidly, the urine became copious, the dropsy disappeared, and at the end of a fortnight there was a mere trace of albumen in the urine. On October 16 the urine was free from albumen. She was then allowed to have fish in addition to milk, and on the following day there was again a trace of albumen, which continued until the 19th, when the fish was omitted and milk alone allowed. On the 23rd the urine was again free from albumen. Then she was ordered a slice of mutton daily; and a few days afterwards, on Oct. 30, the urine was once more found to contain a trace of albumen. Again, therefore, she was restricted to a diet of milk alone; and the smallest possible trace of albumen is present even now (Nov. 27); until this has entirely disappeared we shall restrict her to the exclusive milk diet.

It is impossible to overestimate the supreme importance of strict attention to diet in the treatment of all cases of albuminuria.

But I desire now to direct your attention especially to the beneficial influence of an exclusively milk diet in the treatment of some cases of *cystitis*. No case of this kind has occurred recently in my hospital practice, but within the last two years I have seen elsewhere, several cases in which the curative influence of a milk diet has been very remarkable; and I propose now to give you a few particulars of three illustrative cases, taking them in the order in which they occurred.

Miss D——, aged 17, consulted me first on January 22, 1875. The following history of the case was given in a letter from the patient's mother. 'It was in March, 1874, that my daughter first noticed an irritability of the bladder, disturbing her many times in the day, and three or four times every night. In June she began to suffer pain, especially at

night. She was treated by various medical men, but became daily worse, the water containing blood, pus, and albumen. On October 14 she was sounded for stone, and its absence proved, but all the symptoms became much aggravated. During the succeeding three months she suffered, at intervals varying from two to three days, severe attacks of pain over the bladder, lasting from two to five hours, during which she passed water every two, three, or five minutes. Very hot hip-baths and opium pills afforded temporary relief. I remarked that fish, and more especially oysters, as food, always brought on a severe attack of pain and irritation. On Jan. 22, 1875, being then seventeen, she came under your care.'

She was then suffering from severe pain over the bladder, and from very frequent and painful micturition, so that her life was rendered miserable. The urine was acid, and contained a considerable amount of pus. All the symptoms appeared to be the result of inflammation of the bladder, but her mother had been positively assured that there was disease of the kidneys. She had taken a great number and variety of drugs, but opiates alone had afforded any relief. I advised a trial of an exclusive milk diet, warm hip-baths at night, and an occasional opiate draught, which she had been in the habit of taking for the relief of pain. She also took occasionally some pills containing camphor and extract of henbane. I saw the patient five times during the months of January, February, and March. During that time the milk diet was continued, and there was a gradual improvement. After March 22 I did not see her or hear of her again until Sept. 12, when I received a letter from her mother, in which she says: 'I am sure you will be glad to hear that your young patient is now quite restored to health, and if on any day she does not feel quite so well, a return to *exclusive* milk diet for twenty-four hours makes all right again. She began your treatment in January, and although the improvement was very slow it was continuous. She spent the month of August at the seaside, bathed and swam without any ill-effects, and she is able now to resume all her old occupations. Her food is still principally milk and biscuit, plain meat once a day, very seldom some green vegetables, never tea,

coffee, beer, or wine. I give you these details, believing they will interest you, and because we have had to feel our way so cautiously in the matter of food.' After receiving this letter I requested the writer to call on me with her daughter, and to bring me a specimen of my quondam patient's urine; which I found to be in every respect quite normal, while the young lady reported herself, and looked, in perfect health, and free from all local irritation.

Mr. C. B.——, a country gentleman, aged 48, consulted me October 13, 1876, on account of frequent and painful micturition, which had troubled him for two years. He stated that two years ago he contracted a gonorrhœa, for which, at the commencement of the malady, he used an injection of zinc and morphine. This quickly stopped the discharge, but he then began to suffer from pain in the bladder and frequent calls to pass water, the urine being turbid with mucus, and for several weeks tinged with blood. The frequent painful micturition and turbid urine had continued to the present time. He generally had to pass water every hour both day and night. The pain and irritation were much increased by walking and driving; so that, in going about, he had to wear an india-rubber urinal. The urine brought to me was turbid with puriform mucus, of normal density, of acid reaction, and deposited some uric acid. It was slightly coagulable by heat and acid, the albumen being probably derived from the pus; no tube-casts were present. He had consulted two eminent London surgeons, one of whom had sounded him for stone with a negative result; but the prostrate was said to be enlarged. The symptoms had continued unabated, notwithstanding the various remedies which had been employed; temporary relief only having been obtained by the use of opium suppositories. Up to the time of his coming to me he had continued to take wine at his meals rather liberally.

I advised him to take cold or tepid milk exclusively—no other food or drink,—and I prescribed no medicine. He returned to his home in Yorkshire on the day after his visit to me, and a few days afterwards I received from him the following report, dated October 20:—

'I consulted you on Friday, October 13. I returned home

on Saturday, the 14th, and since the middle of that day I have taken nothing but milk (neither solid nor liquid), as you prescribed. From the very beginning of this diet—that is, from the morning of Sunday, the 15th—I have felt no traces of the inflammation. It has been difficult for me to realise its sudden (and for the time) total disappearance. I twice yesterday drove three miles at a time in a common cab without pain or disturbance. Up to the 14th the irritability had been as bad as ever, and on the afternoon of the 13th I had been in very severe pain while driving a short distance in London. I write to ask how long I ought to continue the milk diet if the irritability does not return. The water is clear, but still leaves a slight cloud after standing in a glass bottle. I am in no hurry, and will follow your instructions strictly. I shall be glad to be liberated from the milk diet, but I prefer it to the inflammation.' In a letter dated October 22 he says: 'The necessity for making water very frequently (about once an hour) ceased along with the inflammation or irritability. I have since made water in the daytime at irregular intervals, but scarcely, if at all, more frequently than when in good health. Yesterday I walked three miles, and sat down for an hour at the end of the walk, without having to make water, and to-day I have also taken a fair amount of walking exercise without any uneasiness. After the length of time that I have been practically a cripple, this seems only too good to last.' On the 23rd I received by rail a sample of his urine, which deposited only a scanty sediment of mucus, and contained no albumen. I then advised him by letter, while still abstaining from alcoholic liquors, to take in addition to his milk, some plain solid food—fish, game, chicken, or mutton,—with some cooked vegetables, and for breakfast an egg with bread-and-butter. On Oct. 27, exactly a fortnight after his first visit, he called and reported himself quite well. He had been four hours and a half in the train the day before, without having once to pass water; he could hardly realise the fact that he was free from all discomfort. The urine was as clear as sherry, and entirely free from mucus. He returned home, and I received from him a note, dated Nov. 6, in which he says: 'Until last Friday, the

4th, I continued to abstain from all alcoholic liquors, as you advised. On and since that day I have drunk wine at dinner only, as usual. This has had no bad effect to my knowledge, and I find myself at present perfectly well.' He ends with an expression of thankfulness for the benefit derived from the treatment.

I have seen a considerable number of cases of rapid recovery from *recent acute cystitis*, when the disease has been promptly and judiciously treated, but I never before saw so rapid and complete a cure of severe cystitis of *two years' duration* as occurred in this case. The result surprised and gratified me almost as much as it did the patient. The *modus operandi* of the milk is sufficiently obvious. The urine is largely diluted with water, and rendered mild and unirritating by the digestible nature of the food; the bladder, therefore, being comparatively undisturbed by its contents, reverts to its normal condition, the inflammation of the mucous membrane subsides, and the morbid secretion of puriform mucus ceases.

The Rev. J. W——, aged 29, a curate in a manufacturing town, consulted me on November 2 on account of frequent micturition, and a painful feeling of irritation extending from the bladder to the end of the penis. He passes water almost every hour. These symptoms have troubled him for the last three months. He has a weak stomach, and he believes that his bladder trouble was excited by his having drunk, one night, two glasses of beer at supper. The urine was acid, turbid with pus, and slightly coagulable by heat and acid. I advised him to try a diet of milk exclusively, and to take no medicine.

Four days afterwards, on November 6, he called and said that he had been taking a gallon of milk daily, and that since the second day the feeling of irritation and the frequent calls to pass urine had ceased.

November 10.—He has taken, in addition to milk, mutton, chicken, and rice. There has been no return of irritation, and he feels quite well. The urine is much less turbid, but it still deposits a slight cloud of mucus. He was to return to his work on the following day.

On November 23 he sent me a specimen of urine, which I found entirely free from mucus and in every respect quite normal; and in a letter dated November 24 he says, 'My health is, I think, better now than it has been for many months past, and my bladder does not seem to be affected in the slightest degree.'

Perhaps a few hints as to the mode of giving milk in these cases may be useful. The milk may be taken cold or tepid, and not more than a pint at a time, lest a large mass of curd, difficult of digestion, form and collect in the stomach. Some adults will take as much as a gallon in the twenty-four hours. With some persons the milk is found to agree better after it has been boiled, and then taken either cold or tepid. If the milk be rich in cream, and if the cream disagree, causing heartburn, headache, diarrhœa, or other symptoms of dyspepsia, the cream may be partially removed by skimming. One reason, amongst others, for giving the milk, as a rule, unskimmed—that is, with the cream—is that constipation, which is one of the most frequent and troublesome results of an exclusively milk diet, is to some extent obviated by the cream in the unskimmed milk. As a rule, it is unnecessary, and, therefore, undesirable, to add bread or any other form of farinaceous food to the milk, which in itself contains all the elements required for the nutrition of the body. When the vesical irritation and catarrh have passed away and the urine has regained its natural character, solid food may be combined with the milk, and thus a gradual return may be made to the ordinary diet, while the effect upon the urine and the bladder is carefully watched.

There are some patients with whom, unfortunately, milk in any form, and even in small quantities, so decidedly disagrees that it is for them as unsuitable a diet as any other form of indigestible food would be.

If a chronic catarrhal condition of bladder remain after the acute symptoms have subsided, copaiba balsam often effects a rapid and complete cure. One capsule may be given an hour or two after food three times a day; and, if the stomach will bear it, the dose may be increased until six, and even nine capsules are taken, in three doses, in the twenty-

four hours. In one case which had been of a year's duration, the urine being turbid with blood and pus, and smaller doses of copaiba having failed to cure, at the suggestion of Sir William Fergusson, who saw the patient with me, the dose was increased to three capsules three times a day, and the result was a complete and permanent cure within six weeks; the urine at the end of the treatment being of a natural sherry colour, transparent, and without a trace of mucus. Quite recently I have seen a young lady in whom acute cystitis from cold had left a vesical catarrh after a period of six weeks. I prescribed one capsule three times a day, and, in less than a week, the urine was entirely free from mucus, and the cure was complete. I have seen equally good results in other cases. It is probable that the remedy, being excreted by the kidneys, has a local curative action on the mucous membrane of the bladder. The copaiba sometimes brings out a transient erythematous rash on the skin. When this occurs, the dose of the copaiba must be lessened, or it may be necessary to discontinue the medicine for a time, and then perhaps to resume it in smaller doses.

If I might venture to give a hint to my surgical colleagues and friends, I should say that an exclusively milk diet would probably be found very suitable, for most patients, during the first few days after the operation of lithotrity; the object being of course to lessen, as much as possible, the inflammation and catarrh resulting from the mechanical irritation of the mucous membrane of the bladder.

I have recently seen two cases in which the vesical irritation and catarrh resulting from a stone in the bladder, were much mitigated by the milk diet; the patients being thereby brought into a more favourable condition to undergo successfully, one the operation of lithotomy, the other that of lithotrity.

CHAPTER XLVII.

ON THE VARIOUS MODES OF TESTING FOR SUGAR IN THE URINE.

Moore's Test—Fermentation Test—Trommer's Test—Fehling's Solution—Dr. Pavy's Ammonio-Cupric Process—Picric Acid and Potash Test—Formula for the Ferric Acetate Colour Standard—The Picro-Saccharometer—Proportions of Picric Acid required—Reduction of Grains per Ounce to a Percentage Proportion—Distilled or Soft Water for Dilution—Various Modes of Diluting—The Picric Acid must be in Proportion to the Amount of Sugar—The Influence of Albumen on the Test very slight—Unoxidised Sulphur Compounds in the Urine have no Influence on the Test—Comparison with Pavy's Ammonio-Cupric Method in Solutions of pure Glucose and in Urine—Reduction in Normal Urine estimated by Picric Acid and by Pavy's Method—Nature of the Normal Reducing Agent and Mode of Determining the Amount—Picric Acid and Potash give a Red Colour to Normal Urine when Cold—Some Constituents in Normal Urine prevent the Precipitation of Suboxide of Copper in Testing with Fehling's Solution—Reduction due to the Administration of Certain Drugs—All Urines should be Tested for both Sugar and Albumen—Ready Method of doing this—Pocket Test Case for Bedside Albumen and Sugar Testing—Polarising Saccharometers—The Practical Importance of Testing for Albumen and Sugar in every Case of Disease—Glycosuria not to be Confounded with Diabetes—Typical Case of Saccharine Dyspepsia—Final Comparison of the Chief Tests for Sugar in the Urine.

GENTLEMEN,—In a former lecture (p. 636) I described and demonstrated the various modes of testing for albumen in the urine; on the present occasion I propose to show you the chief tests for sugar, and to estimate the relative value of each. I will first show you what is called Moore's test. I pour about a drachm of saccharine urine into a test-tube, and add half its bulk of liquor potassæ, then I heat the mixture over the lamp; and after boiling it for a minute or two, you see it gradually assume a brandy-brown colour. This test is easily applied, but it is not very delicate, since it will not indicate a proportion of sugar less than about two grains to the ounce. I will presently show you far more delicate tests.

Then Moore's test has sometimes misled inexperienced observers in this way:—Liquor potassæ often contains lead from the bottles in which it has been kept; and when lead-contaminated liquor potassæ is boiled with albuminous urine, the sulphur of the albumen combines with the lead to form a dark sulphide, which unpractised experimenters might mistake for the brown colour produced in saccharine urine.

The Fermentation Test.—When saccharine urine is mixed with yeast and kept warm, fermentation takes place with the evolution of carbonic acid and the formation of alcohol. Sir William Roberts (on *Urinary and Renal Diseases*) has shown that this test may be made use of for a quantitative analysis. As the sugar is decomposed, the specific gravity of the urine falls. Each degree of specific gravity lost indicates one grain of sugar to the ounce. The great objection to this method is the length of time, at least twenty-four hours, required for its completion. The urine is capable of absorbing about its own bulk of carbonic acid, and, according to Sir William Roberts, urine containing two and a half grains to the ounce or less gives no visible sign of fermentation. It is, therefore, less sensitive than even Moore's test.

Trommer's Test.—To this saccharine urine in a test-tube, I add a drop or two of a solution of sulphate of copper, and to this again an excess of liquor potassæ. The oxide of copper, which is first thrown down, is redissolved, and forms a clear blue liquid. Now, on applying heat the oxide of copper is reduced to a suboxide, which forms a dense red or yellow deposit. An excess of copper, not being dissolved, may cause confusion, and the dark brown colour, from the action of the potash on the sugar, may interfere with the result.

Fehling's Solution.—A more satisfactory mode of applying the copper-test is in the form of Fehling's solution, which contains the following ingredients:—sulphate of copper 90½ grains, neutral tartrate of potassium 364 grains, solution of caustic soda, specific gravity 1·12, four fluid ounces, cold water to make up six fluid ounces. To use this solution, a column of about three-quarters of an inch is poured into a test-tube, and heated until it boils, and then a drop or two of the urine to be tested is added. In a few seconds, if the urine contain

much sugar, the liquid becomes of an opaque yellow colour, and a copious red or yellow precipitate falls. If the quantity of sugar be small, the urine is added more freely, but not beyond the volume of the copper-solution. Fehling's solution soon undergoes change by keeping, a portion of the oxide of copper becomes precipitated, and then, of course, the strength of the test is changed. When the test has been kept for some time, it will deposit suboxide on boiling without the presence of sugar. This is one reason for boiling the test before adding the suspected urine. If boiling the liquid render the test turbid, it must be filtered, or a fresh solution prepared. When the solution is used for a quantitative analysis, it must be freshly prepared, and its strength is such that 100 minims are reduced by half a grain of sugar, the complete reduction being shown by the disappearance of the blue colour, while the suboxide of copper is precipitated. The process must be rapidly completed; otherwise by exposure to the air, a portion of the suboxide is reconverted into cupric oxide, and the amount of sugar will thus be over-estimated. When the urine is highly saccharine, it must be diluted to a definite proportion, five, ten, or more times, before the analysis is made; and then the result is to be multiplied by the number of dilutions.

Dr. Pavy has modified and improved upon Fehling's method of analysis.[1] The essential difference between the method of Dr. Pavy and that of Fehling consists in the addition of a sufficient quantity of *ammonia* to the copper-solution to prevent the precipitation of the cuprous oxide, after its production by the reducing action of the glucose. Rochelle salt (potassio-tartrate of sodium), though it effectually dissolves cupric oxide, is incapable of dissolving cuprous oxide; and some difficulty is often experienced in ascertaining, by Fehling's process, the exact moment of disappearance of the blue colour due to cupric copper, on account of the turbidity and red tint imparted by the precipitated cuprous oxide. This difficulty is removed by Dr. Pavy's process, since the ammonia altogether prevents the precipitation of cuprous oxide, and in a clear solution the exact amount of sugar required to completely de-

[1] *Lancet*, March 1, 1884, p. 376.

colourise the cupric blue tint may be more easily determined. The chief precaution necessary is to completely exclude air during the determination; because a colourless ammoniacal cuprous solution is rapidly rendered blue by exposure to atmospheric oxygen, the cupric hydrate being thereby reproduced.

The Picric Acid and Potash Test for Sugar.—In a letter which I published in the *Lancet*, November 18, 1882, I stated that I had accidentally stumbled upon the fact that picric acid, when boiled with caustic potash, forms a most delicate test for glucose. I added some picric acid solution to a boiling specimen of diabetic urine, which had been previously mixed with liquor potassæ, and found, to my surprise, that the liquid at once assumed an intensely dark colour. I was not then aware of the fact that this reaction of picric acid with grape-sugar had been published by Braun, a German chemist, nearly twenty years before.[1] In that paper, it is shown that grape-sugar, when boiled with picric acid and potash, reduces the yellow picric acid to the deep red picramic acid, the depth of colour depending on the amount of sugar present.

I am not aware that, hitherto, any attempt has been made to utilise this as a qualitative clinical test for sugar in the urine, or as a means of accurately estimating, by the depth of colour, the amount of grape-sugar in a saccharine solution. But now, after having, for a time, been the subject of much hostile criticism, the value of the test for both purposes has been completely proved and established.

I take a fluid drachm of a solution of grape-sugar, in the proportion of a grain to the fluid ounce, mix it with half a drachm of liquor potassæ (*P. B.*), and forty minims of a saturated solution of picric acid, and make up the mixture to four drachms with distilled water.[2] The mixture is conveniently

[1] *Ueber die Umwandlung der Pikrinsäure in Pikramminsäure, und über die Nachweisung der Trauben-Zucker.* C. D. Braun, *Zeitschrift für Chemie*, 1865.

[2] It has been ascertained by careful experiment that, while half a drachm of liquor potassæ may be used as a constant quantity for the analysis of saccharine solutions of any strength, the proportion of picric acid solution should not be less than forty minims, even for the analysis of weak solutions

made in a boiling-tube ten inches long and three-fourths of an inch in diameter, which should be marked at the height of four drachms. With a long boiling-tube, there is little risk of the liquid boiling over; and the steam, condensing in the upper cooler part of the tube, flows back as water, so that there is little loss by evaporation. The liquid is now raised to the boiling-point, and kept boiling for sixty seconds, so as to ensure complete reaction between the sugar and the picric acid. During the process of boiling, the pale yellow colour of the liquid is changed to a beautiful claret-red.

The liquid having been cooled by cautiously immersing the tube in cold water, we ascertain that its level is that of the four-drachm mark on the tube; and if found below the mark, it is brought up to it by the addition of distilled water. The colour now is that which results from the decomposition of picric acid by a grain of sugar to the ounce, four times diluted by the reagents; and this colour is a convenient standard for comparison in making a quantitative analysis. The picramic acid solution, on exposure to the light even for a few hours, becomes paler; but the colour may be exactly imitated by a mixed solution of ferric perchloride, acetic acid, and ammonia. The following is the formula for the standard colour solution, for which, and for much other valuable aid in working out the details of the analytical process, I am indebted to my son, G. Stillingfleet Johnson, F.C.S.

of glucose. If a grain to the ounce solution be boiled with a less proportion of picric acid, the resulting colour is slightly, though appreciably, paler than when the full amount of acid is used. When the full proportion of picric acid is added, a yellowish precipitate of potassium picrate falls in the cold liquid. This is redissolved before the liquid reaches the boiling-point. That the deeper tint obtained by boiling a drachm of solution of glucose, containing *one grain* of glucose per fluid ounce with thirty minims of liq. potassæ and forty minims of picric acid solution, and water up to four drachms, is not due to any red coloration produced by the action of the potash upon the excess of picric acid present, is proved by the fact that the colour thus obtained is identical with that which is observed when one drachm of solution of glucose, containing *four grains* of glucose per one fluid ounce, is boiled with thirty minims of liq. potassæ, forty minims of picric acid solution and water up to four drachms, and the resulting dark liquid is subsequently *diluted four times*. In this latter case, the excess of picric acid present over and above the quantity necessary for complete interaction between it and the total sugar in solution is very small. Either of the above processes therefore might be employed for fixing the grain standard.

Liq. ferri perchlor. fort. (sp. gr. 1·42), ʒj.; acid. acet. glacial. (sp. gr. 1·058), ʒiv.; liq. ammoniæ (sp. gr. 0·959), ɱ100; aqua destil. ad ℥iv.[1]

Mix thoroughly the iron solution with the acetic acid, then add the liq. ammoniæ, and dilute up to four ounces. The ingredients are all of the strength prescribed by the British Pharmacopœia.

The colour of this standard is identical with that which results from boiling with picric acid and potash a grain to the ounce solution of glucose, four times diluted by the reagents and water in the boiling-tube; and as all saccharine solutions, containing not less than a grain of sugar to the ounce, are similarly diluted in the process of analysis, the standard is, in fact, one of a grain to the ounce. When a fresh iron solution is made, it should be checked by comparison with a grain-solution of sugar, boiled with picric acid and potash, and four times diluted as above described.

I have here a standard iron solution, which was made a year ago, and which, having been kept in a closely stoppered bottle, retains its colour unchanged. I have also a solution of grape-sugar, one grain to the ounce, in eighty per cent. of water and twenty per cent. of rectified spirit, which has kept without change in an accurately stoppered bottle for the same period. The alcohol prevents the spontaneous decomposition of the sugar, but has no reducing action on picric acid.

If, now, a drachm of a solution of grape-sugar, containing *two* grains to the ounce, be mixed with the same quantity of liquor potassæ and picric acid, and made up to four drachms in the boiling-tube, the result of boiling the mixture as before for sixty seconds will be the production of a much darker colour than when the one-grain solution was acted upon; but, if now the dark liquid be diluted with its own volume of water, the colour will be the same as that of the one-grain solution.

The dilution, after boiling, is accurately done in a stoppered tube, twelve inches long and three-quarters of an inch in

[1] This standard solution may be obtained from Messrs. Bell & Co., 225 Oxford Street, from Messrs. Savory & Moore, 143 New Bond Street, and probably from other pharmaceutical chemists.

diameter, graduated into 10 and 100 equal divisions. By the side of this tube, and held in position by an S-shaped band of metal, is a stoppered tube of equal diameter, and about six inches long, containing the standard iron solution.[1] (See fig. 41.)

Sufficient of the dark saccharine liquid to be analysed is poured in to occupy exactly ten divisions of the graduated tube. Distilled or rain water is then added cautiously, until the colour approaches that of the standard. The level of the liquid is then read off and noted. A more exact comparison of the saccharine liquid with the standard is made by pouring into a flat-bottomed colourless tube, about six inches long and an inch in diameter, as much of the standard as will form a column of liquid about an inch in height, and an exactly equal column of the saccharine liquid in a precisely similar tube. The operator then looks down through both tubes at once, one being held in each hand, upon the surface of a white porcelain slab, or a piece of white paper. In this way, a slight difference of tint is readily recognised; and, if the liquid to be analysed be found to be darker than the standard, it is returned to the saccharometer and diluted until the two liquids are found to be identical in colour; when the final reading is taken.[2] If

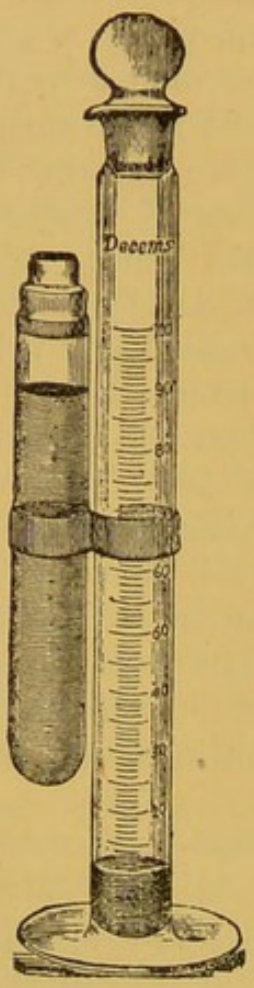


FIG. 41.—THE PICRO-SACCHAROMETER AS DESCRIBED IN THE TEXT.

The shading of the side tube indicates the ferric acetate standard. The darker shading at the bottom of the graduated tube shows the saccharine fluid, darkened by boiling with picric acid and potash, and occupying ten divisions before dilution.

[1] This picro-saccharometer and the other apparatus required for a quantitative analysis are made by E. Cetti, 36 Brooke Street, Holborn, E.C.

[2] For clinical purposes, it is quite unnecessary to use the flat-bottomed

we are in doubt whether the saccharine liquid is darker than the standard, a few drops may be poured back into the saccharometer, so as slightly to shorten the column of liquid in the test-tube; and if now the tints appear more nearly equal, it is evident that the liquid requires rather more dilution. The saccharine liquid having been diluted four times by the reagents, a colour equal to that of the standard would indicate one grain of sugar per fluid ounce. If further dilution were required—say from ten to twenty divisions—the proportion of sugar would be two grains per ounce, and so on to thirty or forty or upwards, or to intermediate divisions. Thus, dilution from ten to thirty-five divisions would indicate 3·5 grains of sugar per ounce.

We have found, by experiment, that ten minims of a cold saturated solution of picric acid are rather more than sufficient for decomposition by one drachm of a solution of grape-sugar, in the proportion of one grain to the ounce. A drachm of the solution would of course contain one-eighth of a grain of sugar. In making an analysis of *urine*, the various ingredients should be mixed in the following proportion: A drachm of the urine, a drachm of a saturated solution of picric acid, half a drachm of liquor potassæ, and a drachm and a half of water. This amount of picric acid is sufficient for the analysis of a drachm of a solution of sugar in the proportion of six grains to the fluid ounce. If the amount of sugar be less than this the excess of picric acid does not interfere with the result.[1] But if the proportion of sugar be higher than six grains to the ounce, the saccharine fluid should be diluted with distilled or rain water, in a definite proportion, before commencing

tubes. A sufficiently accurate result is obtained by comparing the depth of colour in the two tubes of the saccharometer.

[1] Although some impure samples of picric acid give a reddish colour when boiled with caustic potash, pure picric acid, such as may be easily and cheaply obtained, forms a pale yellow mixture with boiling liquor potassæ. I once met with an impure sample which not only gave a red colour when boiled with liquor potassæ, but the liquid was rendered turbid by fine granules. The impurity was removed by solution and recrystallisation. A simple test of the purity of picric acid for chemical purposes is to boil a mixture of one volume of a saturated solution with half its volume of liquor potassæ. The resulting liquid should be quite pellucid, and of a pale lemon-yellow colour, without the slightest tinge of red.

the analysis, and the product of the analysis of the diluted fluid is then to be multiplied by the degree of dilution—two, five, or ten, as the case may be, to which the original fluid has been subjected.

When the urine has been diluted ten times, the figures on the saccharometer indicate the number of grains per ounce. Thus, when the ten times diluted urine, after boiling with picric acid and potash, is further diluted in the saccharometer, from 10 divisions to 35, to obtain the standard colour, the amount of sugar is 35 grains to the ounce; while the same figures, with a sample five times diluted, would indicate half that amount—that is, 17·5 grains.

To reduce the amount of sugar per ounce to the proportion per cent., we have to remember that an ounce of water at 62° F. weighs 437·5 grains. If now the proportion of sugar is 40 grains per ounce, then as 437·5 : 100 : : 40 : 9·142 per cent.

Distilled water, or clear rain-water, should be used for diluting. Hard water, containing salts of lime, is rendered somewhat turbid by the carbonate of lime precipitated by mixture with caustic potash; and any turbidity in the liquid interferes with the exact estimation of the depth of colour. In testing undiluted urine, a slight turbidity often results from separation of phosphates by the potash. This turbidity may be removed by allowing the phosphates to form a sediment, or more speedily by filtration. When a highly saccharine liquid is diluted five or ten times before mixture with the testing materials, no phosphatic turbidity occurs. In making a volumetric analysis, care must, of course, be taken that the measurements and dilutions are accurately made.

The preliminary dilution of a strongly saccharine specimen may be made in the graduated saccharometer tube; or, into a flask graduated to contain fifty cubic centimetres, five or ten cubic centimetres of the saccharine liquid may be delivered from a graduated measure or pipette;[1] then, the flask being

[1] If a graduated *measure* is used it should be rinsed out after pouring the urine from it into the diluting flask with some of the water used for diluting, but a *pipette* is graduated to deliver a certain measure of liquid without subsequent rinsing.

filled up to the graduation with distilled or soft water, the dilution will be ten times with five cubic centimetres, and five times with ten cubic centimetres of the liquid to be analysed. Or, without any special apparatus, an accurately measured drachm of urine may be diluted up to five or ten drachms with distilled water.

Another important point is that, while the amount of potash remains the same, the picric acid must be in proportion to the quantity of sugar in solution. It has already been mentioned that ten minims of the picric acid solution are more than equal to one-eighth of a grain of glucose, which is the amount contained in one drachm of a solution of glucose, in the proportion of a grain to a fluid ounce. A considerable excess of picric acid does not appreciably affect the colour of the picramic acid, while a deficiency would, of course, lead to an under-estimate of the amount of sugar. If an analysis with sixty minims of picric acid solution indicates from six to seven grains of sugar per ounce, it is probable that some sugar has been left undecomposed; and a second analysis of a diluted specimen, might therefore give a higher and a more correct result. If, on the other hand, a second analysis, with a larger proportion of picric acid, gives an identical result, we may feel certain that the whole of the sugar has been decomposed, and the amount correctly indicated by the resulting picramic coloration. In any case, when the amount of sugar indicated is less than would suffice to react upon the amount of picric acid employed, the result may be relied upon as correct.

The presence of albumen, even in large amount, has but little influence on the picric acid test for sugar. In illustration of this the following experiments will suffice. A specimen of urine, normal as regards its reducing influence on picric acid, but containing a large amount of albumen, was boiled with picric acid and potash, with sufficient water to dilute the urine by its own volume of liquid. A second portion was treated in the same way after the separation of the albumen by boiling and filtration, and the first specimen gave a darker tint than the second, to a degree that might be considered to indicate one-tenth of a grain of sugar per ounce. Another portion of the urine was decolorised by repeated filtering

through charcoal; and of this, one specimen was tested while it retained its albumen, another after the separation of the albumen, the result being that both yielded identical tints of colour; and this was very slightly paler than that of the specimen which was tested after having been deprived of its albumen without previous decolorisation by charcoal. The explanation is that pure albumen has no reducing influence on picric acid when boiled with dilute potash, such as is used in testing for sugar; but with seralbumen, as with white of egg, there is associated a colouring matter which is partly separated by filtering off the coagulated albumen, and entirely removed by repeated filtering through charcoal. The colouring matter in question has a slight reducing influence on picric acid, although the colouring matter of normal urine has been found to have none. The coagulated albumen collected on the filter, after being thoroughly washed, gives no red reaction when boiled with picric acid and potash diluted in the same proportion as that employed in testing for sugar. This has been proved by repeated experiments.[1]

For practical purposes the very slight influence of even the largest amount of albumen on the colour resulting from the picric acid test may be disregarded. The fact that an exact quantitative analysis of saccharine urine may be made without the previous removal of albumen is one amongst other advantages which this method possesses over the copper process, for which the separation of the albumen is an essential preliminary.

The accuracy of the picric acid method of volumetric sugar-analysis has been fully and fairly tested by my son and

[1] When I first published to the profession my observations on picric acid and potash as a test for sugar, it was suggested that unoxidised sulphur-compounds in the urine, and especially in albuminous urine, would form an alkaline sulphide when boiled with potash, and this would decompose the picric acid and render the test fallacious. Experience has proved that these theoretical objections were quite groundless. My son proved conclusively that, when pure albumen is boiled with diluted solutions of potash, such as are used in testing for sugar, no alkaline sulphide is formed. The final communication of my son, in which he demonstrated that the apparently contradictory results obtained by different observers are explained by the varying proportions of the caustic potash employed, was published in the *Chemical News*, February 23, 1883, p. 87.

myself. Our plan has been to compare the results of this process with those obtained by Dr. Pavy's ammonio-cupric method. We have analysed the same specimens, many of them albuminous as well as saccharine, by the two processes; and our results are found to be practically identical, the differences being only such as are due to unavoidable slight errors in conducting an experiment. Both methods, in fact, are based upon the same chemical principle—namely, that glucose, when heated with potash in the presence of an oxidising agent, has a tendency to rob it of its oxygen. In the one process the reducing action of the sugar is exerted upon an oxide of copper, in the other on picric acid. A definite weight of sugar reduces, in the one case, a proportional amount of cupric oxide, and in the other an equivalent proportion of picric acid, with resulting picramic acid and a corresponding measurable intensity of colour. In analysing pure solutions of glucose in water, the two methods give identical results, but in the majority of cases of urinary analysis the ammonio-cupric process gives results slightly in excess of the picric acid method. This excess is due to uric acid and urates, which reduce cupric oxide but not picric acid.

I claim for the picric acid and potash method of analysis that it is as accurate as any other, and that for the estimation of sugar in the urine it is even more accurate than either Fehling's or Dr. Pavy's process, because the picric acid is not acted on by uric acid or urates, which reduce the oxide of copper. The method of analysis by the picro-saccharometer is more speedy and more easily learnt and practised than any other. The materials and apparatus required are easily prepared, inexpensive, and not, like Fehling's copper solution, liable to undergo rapid changes by keeping. For exact results with the picric acid process, the main requisite is that the standard should be accurate. This is as important as the exact proportion of copper in Fehling's or Pavy's volumetric solution. The standard iron solution, as I have shown, may be kept for many months without the slightest change of colour; and a solution of grape sugar in water, one grain to the ounce, with twenty per cent. by volume of rectified spirit, may be kept unchanged for an indefinite period, and used occasionally for

comparison with the ferric acetate standard. For obtaining a solution of glucose of known strength we have relied entirely upon Dr. Pavy's ammonio-cupric process.

The picric process of analysis is very useful for ascertaining the amount of sugar in wines and other liquors, and so enabling the medical attendant to advise his diabetic patient as to the safety or otherwise of his beverages. Dark-coloured liquids, such as porter or port wine, must be decolorised by animal charcoal before commencing the analysis, but for light-coloured liquors, such as ale, champagne, and sherry, no preliminary process is required.

During the last three years I have tested with the picric acid and potash a large number of specimens of *normal* urine, with the almost uniform result of a depth of colour indicating that if sugar were the reducing agent the amount would be 0·6 grain in the fluid ounce, the indication usually varying from 0·5 to 0·7 grain; but in exceptional cases, in urines of high density, amounting to 1 grain in the fluid ounce. In a considerable number of cases I have tested the same specimens by the ammonio-cupric method, with the indication usually of from 0·7 to 0·9 grain in the fluid ounce, *i.e.* an excess of that obtained by picric acid of 0·2 grain in the fluid ounce.

The following have been the proportions of the various liquids: 2 drachms of urine, ½ a drachm of liquor potassæ, 40 minims of picric acid solution, made up to 4 drachms with distilled water. The mixture is kept boiling for a minute, and, when cooled, is compared with the standard. The urine having been diluted only by its own volume, a depth of colour equal to that of the standard would indicate 0·5 grain of sugar; but, in nearly every case, I have found it so much darker than the standard as to require further dilution equal to an additional 0·1 grain before the standard colour is reached, thus giving an indication of 0·6 grain.

When the mixture is rendered turbid by phosphates, these must be removed by filtration before the colour can be quite accurately estimated.

So constant is this degree of coloration with normal urine that if, instead of diluting up to 4 drachms, the dilution be

carried further by 50 minims, the resulting colour might be taken as an approximation to an accurate grain standard, and, in the absence of a more exact standard, might be used for making a quantitative analysis. The question arises, Does normal urine contain as much as 0·6 to 0·7 grain of glucose in the fluid ounce?

At one time I thought that this question required an affirmative answer, but I am now convinced to the contrary. My son has long been engaged in an elaborate investigation of the reducing agent which is always present in normal urine, and an abstract of his results has recently been published.[1] He has obtained conclusive evidence (1) that not a trace of glucose is to be found in normal urine; (2) that the chief, if not the sole, substance in normal urine that reduces picric acid, under the influence of caustic potash, is kreatinin.

The following process is employed by my son for estimating with accuracy the quantity of normal reducing agent—*i.e.* kreatinin—in any specimen of urine:—

A measured volume of the secretion, having its normal acidity, or, if alkaline, acidulated with acetic acid, is mixed with one-twentieth of its volume of a cold saturated solution of sodium acetate, and one-fifth of its bulk of cold saturated solution of mercuric chloride.

Filtration is performed at once, the precipitate being rejected, whilst the filtrate is left to itself for forty-eight hours. The precipitate which has formed is then collected upon a weighed filter-paper, washed with cold distilled water and allowed to dry by exposure to the air at the ordinary temperature. When dry, the precipitate and filter-paper are weighed together, then the weight of the former is ascertained by deducting that of the latter from the total weight.

This precipitate contains the whole of the kreatinin in the volume of urine taken, and it contains 20 per cent. by weight of that substance. Therefore, if we take one-fifth of the weight of the mercury precipitate, we know the weight of kreatinin in the volume of urine taken.

It has been found that *twelve* parts by weight of the urinary kreatinin reduce cupric oxide to the same extent as

[1] *Proceedings of the Royal Society*, vol. xlii. pp. 365–368.

ten parts by weight of glucose. It is easy, therefore, to deduct from the total reduction exerted by the urine (whether saccharine or not) that due to the kreatinin which it contains. For clinical purposes the preceding process is of course quite unnecessary.

One difference between normal urine and a weak solution of glucose consists in the fact that while the former gives some red coloration with picric acid and potash *while cold*, this coloration being increased by boiling, a pure solution of glucose gives no red colour until the mixture of the urine with the reagents has nearly reached the boiling point. In this respect normal urine resembles a solution of kreatinin, which has so powerful a reducing influence on picric acid, when mixed with potash, that it gives a red colour at the ordinary temperature.

Fehling's solution is decolorised by boiling with normal urine, but a precipitate of suboxide is prevented by some constituents of the urine which keep it in solution. With reference to this subject Dr. Beale relates the following instructive experiment. A precipitate of suboxide of copper was obtained by boiling a solution of grape-sugar with alkaline copper solution. To a portion of subo ide produced in this way about a drachm of warm healthy urine was added, and the reddish precipitate was instantly dissolved, forming a perfectly clear solution.[1] I have repeated this experiment with the same result.

Since using the picric acid and potash test for sugar I have so often found sugar in urine when, from the comparatively low specific gravity, I should not have suspected it, that I now test every specimen of urine, first for albumen and then for sugar, by the following simple and trustworthy method, which may be completed in about two minutes. To about a drachm of acid urine I add its own volume of picric acid solution. If the liquid remain clear no albumen is present. If a precipitate occurs, not dissolved by boiling, there is albumen in proportion to the amount of precipitate. I now add half a drachm of liquor potassæ and boil for a few seconds; the coagulated albumen if present is dissolved by the alkali and a

[1] *Kidney Diseases, Urinary Deposits, &c.*, p. 246.

red or black coloration occurs. If, when an ordinary test-tube half an inch in diameter is held up to the light, a bright red colour is visible through the middle of the column of liquid, no sugar is present. As little as two grains of glucose to the ounce will render the liquid so dark that no light is transmitted through the middle of the tube. The amount of sugar must then be determined by the quantitative method.

I have often found that the urine of patients who are taking salicylate of sodium is darkened by boiling with picric acid and potash to a degree that might indicate from one to two grains of glucose per ounce. The copper solution is also reduced by the same urine as it would be by a small amount of glucose. My son is investigating the nature of the substance which causes this reduction. He has ascertained that it is neither glucose nor an excess of kreatinin. It is probably some product of the decomposition of the salicylate of sodium, since that salt itself has no reducing action on picric acid or cupric oxide.

Dr. Sherwin[1] has found that urine to which chloral hydrate had been added to prevent putrefaction reduced Fehling's solution like glucose. The urine of two patients who were taking medicinal doses of chloral also gave a similar reaction. I have confirmed Dr. Sherwin's observation, and I find that while chloral hydrate has a most powerful reducing action on the copper solution, it has none on picric acid.

One great advantage of picric acid as a test for albumen and sugar is that, in the form of powder, it can be carried in a pocket test-case without risk of injury either to the case or the pocket. After a trial of various forms of pocket case I find the most convenient to be a nickel-plated urine test-case, made by Jahncke, Canonbury Works, Dorset Street, Essex Road, N., and sold by most surgical instrument makers and pharmaceutical chemists for 10*s*. The case, as I have it fitted, contains two test-tubes and a small nipple pipette, but some may prefer to have a small urinometer in place of the second test-tube. The pipette is useful for transferring the portion of urine to be tested from the chamber utensil to the test-tube.

[1] *Boston Medical Journal*, Nov. 1886, p. 487; and *Pharmaceutical Journal*, Dec. 1886, p. 507.

The case also contains a small spirit-lamp, a small bottle with powdered picric acid, another containing grain lumps of caustic potash, and a graduated drachm measure. In testing for albumen as much picric acid powder as may be carried on the end of a penknife (about a grain) is placed in one of the test-tubes; to this is added about a drachm of water. The powder is dissolved by boiling the water over the spirit-lamp. An equal volume of urine is then added to the hot picric acid solution, when the presence of albumen will at once be shown.

To test for sugar a grain lump of potash is added to the mixed urine and picric acid, and the boiling is continued for a few seconds, when the blackening of the liquid would indicate the presence of sugar. First diluting the urine by means of the graduated drachm measure, or in any other convenient way which gives a known degree of dilution, and by subsequent dilution of the blackened liquid in the test-tube until it acquires only the degree of coloration of the standard solution before described (p. 797), an approximate estimate of the amount of the sugar per ounce may be made at the bedside; but for an exact analysis the graduated saccharometer must be employed.

Polarising Saccharometers.—I will not attempt to show you or describe to you the various forms of polarising saccharometers, for they appear to me to be too complicated, and most of them too costly, for ordinary clinical use.

Of the practical importance of testing for *albumen* in every case of disease, I have before said enough,[1] and now I propose to add a few practical remarks on the subject of *glycosuria*. And in the first place you will have to bear in mind that the terms glycosuria and diabetes are by no means synonymous; we not unfrequently meet with glycosuria in proportions varying from three or four grains to fifteen or twenty grains per ounce, the urine being normal in quantity and specific gravity, and there being no complaint of thirst or other symptom of diabetes. In a large proportion of cases these patients have passed middle age; they are more or less troubled by flatulence, irregularity of the bowels, and other symptoms of dyspepsia. Questioned as to their dietary, they confess to a fond-

[1] See Chapter XLV. p. 646.

ness for various forms of saccharine food; and, in short, these are cases in which the taste for such food has survived the power of digesting it. The result is that undigested sugar is eliminated, and a rigid abstinence from saccharine food, including of course all kinds of fruits, is often followed by the speedy and complete disappearance of the glycosuria. These curable cases of saccharine dyspepsia in middle-aged and elderly people differ essentially from the intractable diabetes which is so often met with in young subjects.

The following is a typical example of this form of saccharine dyspepsia. On December 8 I was consulted by an eminent provincial oculist in large practice. He was forty-nine years of age, working hard, living well, and not taking much exercise. He had had gout more than once. Eight days before, he began to be giddy, with confusion of thought, and a physician whom he consulted found a trace of albumen in his urine. I found the urine with a sp. gr. 1021, a trace of albumen, at once shown by picric acid, and slowly by nitric acid. When boiled with picric acid and potash, it became inky black, and the saccharometer indicated six grains of sugar per ounce. There were no diabetic symptoms, but he was manifestly dyspeptic, and confessed to a great liking for sweets. These, of course, I advised him to discontinue, and I prescribed a mixture with quinine, strychnine, hydrochloric acid, and ginger. On January 25, when I again saw him, the albumen had gone, and the sugar was reduced to one grain per ounce. Also an eczematous rash which had troubled him for some months had disappeared. In this case, as in many cases of glycosuria, the albuminuria was probably a result of irritation of the kidneys by the unassimilated sugar, and the skin-eruption was probably due to the same cause.

This is one of many cases which have lately come under my observation, in which the routine practice of applying the picric acid and potash test for sugar has led to the unexpected discovery of variable amounts of glucose, and has thus given a clue to the successful treatment of the associated symptoms.

In conclusion let me briefly recapitulate the main points regarding the tests which have hitherto been commonly

employed for the detection of sugar in urine. These tests are :—

1. Moore's Test.
2. The Fermentation Test.
3. Trommer's Test.
4. Fehling's Test.
5. Pavy's Ammonio-Cupric Method.

1. *Moore's Test* is easy of application, and is not affected by any substances usually present in urine, except glucose. Its disadvantages are its want of delicacy, and the fallacy which may result from the presence of lead in the liquor potassæ (p. 793).

2. *Fermentation Test.*—There are fallacies connected with this test which have been fully described (p. 793), and it is also objectionable on account of the length of time required for its completion.

3. *Trommer's Test.*—This is a good qualitative test, but as it cannot detect less than two grains of glucose in a fluid ounce of urine, the negative evidence which it may afford is of comparatively little value (p. 793).

4 & 5. The chief objections to *Fehling's* and *Pavy's* tests for the quantitative estimation of sugar in urine are, 1st, that they are affected by uric acid, which is a constant ingredient of that secretion. For clinical purposes the difference of a fraction of a grain per ounce is of no practical importance; but, 2nd, the successful working of both these methods requires an amount of manipulative skill which is not easily acquired without special instruction in a laboratory. Dr. Favy's ammonio-cupric process, admirably suited as it is for the laboratory, has the special disadvantage for the consulting-room, that during its performance the air of the room becomes unpleasantly charged with ammoniacal vapour.

Those who use this test will be grateful to Dr. Pavy for the elaborate table which he has published[1] to facilitate the calculation of the amount of sugar in a given solution.

Finally, the picric acid method possesses all the advantages of the before-mentioned tests, while it is free from their attendant drawbacks. Thus as a qualitative test it is

[1] *Lancet*, March 1, 1884.

more rapidly completed and more delicate than Moore's and Trommer's tests, and indicates the smallest amount of saccharine matter in the urine. And as a quantitative method, it is free from the objection which applies to all the copper tests, of being affected by uric acid or urates; neither does the presence of albumen interfere with the action of the test, as it does with all the forms of copper testing. Then as regards accuracy and facility of operation, having worked diligently at all the methods, I am in a position to make a comparison, and I have found it easier to distinguish between slight shades of red colour in working the picric acid process than to be assured of the exact period of complete disappearance of the blue colour in the copper solution, even when using Dr. Pavy's improved ammonio-cupric test.

Lastly, with reference to rapidity of operation, the picric acid process surpasses all the others. In fact it is so quickly and easily worked that any student or practitioner will find, after a few experimental trials of the method, that he can, in the course of a few minutes, and while talking to his patient, complete an exact quantitative analysis of saccharine urine; and thus obtain the data for estimating the influence of dietetic and other remedial measures on the amount of sugar secreted.

CHAPTER XLVIII.

ON MOVABLE KIDNEY.

General Account of the Condition—Importance of a Correct Diagnosis illustrated by Cases—Three Forms of Misplaced Kidney—Movable Kidney more frequent in Women than in Men, and especially in Women who have borne Children—Not confined to the Working Classes—The Right Kidney much more frequently affected than the Left—Case of Movable Left Kidney with Scybala in the Descending Colon—Causes of Movable Kidney—Symptoms—Diagnosis—Treatment—Abdominal Band and Pad—Examples of Great and Immediate Relief—Surgical Treatment—Nephroraphy—Nephrectomy.

The subject of misplaced and movable kidney is one of considerable interest and practical importance. Rayer, writing in 1841,[1] remarked upon the fact that, although the mobility and displacement of the kidneys had been, from ancient times, cursorily mentioned by anatomists, it had not hitherto attracted the notice of physicians. Since the publication of Rayer's great work, in which he gave the history of several examples of misplaced kidneys which had come under his own observation, this class of cases has been the subject of a copious literature.

The practical importance of the subject may be illustrated by a reference to the history of cases in which a kidney, out of its normal position, has been mistaken for a morbid growth; a mistake which has frequently been the source of great and groundless anxiety, and which has often led to the adoption of painful and mischievous methods of treatment. Thus Rayer[2] states that two medical men had been rendered most anxious as to the cause of a constant pain in the right loin, where each of them had discovered a movable tumour, as to the nature of which the most diverse opinions had been

[1] *Traité des Maladies des Reins*, tome 3, p. 783.

[2] *Op. cit.*, pp. 783 and 801.

given. One of these men had been so alarmed by the discovery of the supposed tumour in his belly that he had, for the time, given up the practice of his profession. Rayer succeeded in convincing both his anxious confrères that they were suffering from nothing more serious than a misplaced kidney.

My attention was first specially directed to this subject by reading a series of instructive cases which were published by Dr. Hare.[1] One of the first cases that subsequently came under my observation was that of Mrs. J. M——, aged 32, who was admitted under my care in the hospital in November 1859.[2] She was suffering from bronchitis; in addition to which she presented in a marked degree, the physical signs of emphysema of the lungs. She was married, but had borne no children. She stated that, for about seven years, she had been aware of the existence of a tumour in the right side of her abdomen; she could feel the tumour move within the belly, and it was attended with a sense of weight and dragging. She was of spare habit, and the abdominal walls were flaccid, so that the tumour could be readily felt. It had the form, size, and consistence of the kidney. Its usual position was midway between the umbilicus and the anterior superior spine of the ilium, but it could readily be pushed beneath the ribs on the right side. When the fingers of the left hand were placed in the right flank, and those of the right hand in front, near the navel, the tumour could readily be pushed to and fro between them. When it was firmly compressed between the two hands, the patient complained of some degree of tenderness. The left kidney could not be felt below the ribs. The liver extended somewhat below the margin of the ribs, in consequence, probably, of that organ having been depressed below its normal position by the enlarged emphysematous lungs; but when the movable kidney was in its lowest position, there was a considerable interval between it and the under surface of the liver. She complained of a burning pain in passing water, the urine being alkaline and depositing a copious sediment of phosphates, but not being otherwise abnormal.

[1] *Medical Times and Gazette*, 1858, vol. i.

[2] The case was published in the *Medical Times and Gazette*, Jan. 7, 1860.

The surface of the patient's abdomen presented numerous large scars caused by tartar emetic ointment, which, by the advice of a London surgeon, had been rubbed in as a remedy for the tumour.

At the present day, after the publication of numerous cases of misplaced kidney during the last twenty years, an error of diagnosis and a resort to a painful and useless mode of treatment, in a case so plain and palpable as this, would be inexcusable.

An exact diagnosis of these cases will not only have the negative result of preventing needless anxiety as to the nature of a supposed tumour, and the consequent resort to mischievous methods of treatment, but it has the yet further and greater advantage of suggesting remedial measures which, in a large proportion of cases, entirely remove or greatly mitigate the suffering so often caused by a misplaced kidney.

And here it may be well to mention, that under the general term misplacement of the kidney are included three classes of cases :—

1. Misplacement without mobility of the organ.

2. Misplacement with perceptible mobility behind the peritoneum—the so-called 'movable kidney.'

3. Misplacement with mobility, the kidney lying between two folds of peritoneum, constituting a mesonephron. This is the so-called 'floating kidney.'

The first and third class of cases are of comparatively rare occurrence, and the condition is usually congenital. The second class, the 'movable kidney,' has by far the greatest clinical interest and importance.

A simply misplaced kidney is not only a congenital abnormality, but it has rarely been discovered except by a *post-mortem* examination,[1] and it has little or no clinical importance. In rare cases, however, it has happened that a kidney in the iliac fossa, or on the promontory of the sacrum, has seriously impeded parturition.

Although several cases of 'floating kidney,' with a com-

[1] See an interesting paper on *Misplacement and Mobility of the Kidney*, by Mr. Durham. *Guy's Hospital Reports*, 3rd series, vol. vi. 1860, and the report of a committee of the Pathological Society, *Path. Trans.* vol. xxvi.

plete investment of peritoneum and a mesonephron, have been ascertained to be such by *post-mortem* examination, the cases are very rare; and it is impossible to distinguish such a case from a freely movable kidney behind the peritoneum, except by making an exploratory incision in the loin or by opening the abdomen. To the surgeon who contemplates the operation of nephroraphy or nephrectomy, the anatomical difference between a kidney with and without a mesonephron is one of great practical importance; since in the former case the kidney is not accessible without opening the peritoneum.[1] My future remarks will have reference solely to cases of post-peritoneal movable kidney.

Amongst the numerous contributions to the literature of this subject, one of the most interesting and instructive is Landau's *Wanderniere der Frauen* (Berlin, 1881), which has been admirably translated and annotated by Dr. Champneys.[2]

All observers agree in stating that the disease is by far more frequent in women than in men. Landau's collected statistics give the proportion of 87 women to 10 men.

Amongst women it is more frequent in those who are married and have borne children than in the unmarried and the barren. Out of 42 cases observed by Landau 2 only occurred in women who had never borne children.

Amongst 178 cases of movable kidney collected by the same author, 151 were on the right side, 24 on the left, and 14 bilateral. In contrast with this accidental misplacement of the kidney, statistics show that congenital malposition of the kidney is far more frequently found on the *left* side.

Most of the recorded cases of movable kidney have occurred amongst hospital patients, but the condition is of frequent occurrence amongst women in the middle and upper classes.

Out of the 15 cases which have come under my own observation, and of which I have preserved a record, there were 14 women and 1 man. The only one of these cases that occurred amongst my hospital patients was that of the woman before referred to (p. 813). The others were private patients in comfortable circumstances.

[1] See Mr. Henry Morris's *Surgical Diseases of the Kidney*, p. 47.

[2] *New Sydenham Society's Selected Monographs*, vol. cx. 1884.

Of the 14 women, one only was single, and of the 13 married women, 10 had borne one or more children, 2 had not borne children, and of one I have no record of her having borne children or not.

In all but one of my 15 cases the right kidney was the one misplaced. The case of left-side movable kidney is recorded in the *Medical Times and Gazette*, October 1859. This case was that of Mrs. M——, 34 years of age, married, but without children. There was a history of attacks of pain in the left loin, which had been supposed to be the result of a renal calculus; but no gravel or blood had ever appeared in the urine. I found a tumour in the left loin, and I could distinctly feel a scybalous mass in the descending colon; but after the removal of the scybala by enemata and a dose of castor oil, a tumour still remained in the loin. This at first excited the suspicion of a malignant growth or an inflammatory thickening of the colon, a result, perhaps, of the irritation caused by hardened fæces.

Further examination, however, convinced me that the case was one of a movable left kidney. In form, size, and consistence it exactly resembled the kidney. The lowest point to which it could be made to descend was the umbilicus, from the left side of which it was freely movable upwards, and it could readily be pushed beneath the ribs. This movement was effected without pain to the patient, who complained of some degree of tenderness, only when the mass was firmly compressed. She stated that, when lying on her back, she had often felt the lump with her hand, and that she had frequently had a sensation of something solid moving within the abdomen, as well as a rather painful sense of dragging when standing or walking. I was then able to assure her that the tumour in question, although a source of inconvenience, need cause her no anxiety, and I advised her, in addition to adopting means for promoting a regular action of the bowels, to wear an elastic abdominal support.

The Causes of Movable Kidney.—A kidney which has become enlarged by strumous or cancerous disease, or by calculous nephritis, may be misplaced downwards by its own weight. Such cases are of common occurrence, but they have no

relation to our present subject. Amongst the causes of a misplaced and movable kidney, the absorption of the adipose capsule in persons who, having been corpulent, have undergone great and rapid emaciation, is supposed to be influential. In one of my patients, the symptoms and signs of a movable kidney followed upon a severe attack of typhoid fever. Landau refers to the fact that in four out of nine of Dietl's cases, severe ague and typhoid fever had preceded the movable kidneys.

The vast majority of women affected with movable kidney have borne children, and it is manifest that frequent and difficult labours are amongst the most influential causes of this condition. Frequent pregnancies may contribute to the production of movable kidney by causing a flabby condition of the abdominal walls with *pendulous belly*; and so depriving the kidney, in common with other abdominal viscera, of its normal support. On the other hand, the excessive intra-abdominal pressure during pregnancy and parturition may directly thrust the kidney out of its place.

In seven out of my fourteen cases of women, the kidney trouble was attributed to either frequent or difficult labours.

The strain of violent or repeated coughing is stated by several authors to have misplaced the kidney. One of my patients, who had suffered from a movable kidney for fourteen years, stated that, while coughing violently during her pregnancy, she felt something suddenly give way in her right side, and from that accident she dated the commencement of her troubles.

In like manner, lifting heavy weights and straining at stool are amongst the assigned causes of movable kidney. One of my patients, when twenty-two years of age, while making a great effort to raise her husband, who had fallen on the floor in a fit, was seized with sudden pain in her right side, which continued until, a few days after, she felt a swelling beneath the ribs on that side. This proved to be a misplaced kidney, which has since caused much and prolonged suffering.

Another of my patients, Miss C——, 31 years of age, while lifting a heavy two-year-old child, was seized with a sudden pain in the right loin, so severe that she dropped the

child. For some days the pain compelled her to lie down. In the course of a few weeks she began to suffer from the usual symptoms of a movable kidney, and when I saw her, a year afterwards, the right kidney was manifestly misplaced.

Cases are recorded in which the kidney has been dislocated by a blow or a fall. The only instance of movable kidney in the male that has come under my own observation was believed to have been caused by a fall from a tree when the patient was fifteen years of age. Two years after the fall Mr. M—— began to suffer from pain in the right loin, where he felt a lump beneath the ribs. The lump had continued, and there had been periodical attacks of pain in the loin. When I saw him, twenty-two years after the accident, the right kidney was misplaced and apparently enlarged.

Tight lacing of the chest by stays has been supposed by some authors to have contributed to the misplacement of the kidney; and it is not improbable that by restraining the movement of the ribs, and so compelling a greater compensatory descent of the diaphragm in inspiration, the subjacent organs, especially the liver and the kidneys, may be unduly depressed. Even though the influence of tight lacing may be insufficient to push the kidney from its normal position, it would tend to perpetuate and increase a misplacement which may have resulted from other causes.

Various conditions have been supposed to contribute to the greater frequency of misplacement of the *right* kidney. Amongst these, one of the most influential appears to be the downward pressure of the solid liver, when the abdominal organs are compressed during the violent straining efforts of the abdominal muscles. The left renal artery, being shorter than the right, is supposed to lessen the mobility of the kidney on that side. In addition, the ascending colon is less firmly bound to the right kidney than is the descending colon to the left, which, therefore, is more firmly fixed than the right.

Symptoms.—Although, in some recorded cases, a movable kidney has caused little or no inconvenience, its malposition in the loin having been only accidentally discovered; on the other hand, in the great majority of instances, symptoms of greater or less severity result from the misplacement. Amongst

the most frequent subjective symptoms are a constant feeling of weight and dragging in the loin, increased by the erect posture and by exercise; and a sensation of something moving within the abdomen, especially on turning, when in bed, from the affected side to the other. Pain often extends to the thigh and to other parts. Thus Landau states that two of his patients had lumbar and intercostal neuralgia on the left side with a movable right kidney; and on pressing firmly against the misplaced kidney, both patients complained of circumscribed pain in the region of the opposite kidney. These pains are doubtless due to the dragging of the kidney upon its attachments, but more especially upon its nerves. The pains are in most women increased during the menstrual period. In addition to the more or less constant aching pains, these patients are liable, after unusual exertion, to paroxysmal attacks of more severe pain, with vomiting and a scanty secretion of urine; the urine sometimes being blood-tinged. During these attacks, which may continue for many hours or even for several days, the affected kidney is found to be enlarged and exquisitely tender. The attacks usually pass away with a copious secretion of urine. I have myself had no opportunity of examining a patient during one of these paroxysms, but most of my patients have described the symptoms in almost identical terms, and in strict agreement with the account here given.

It is not surprising that symptoms which bear so striking a resemblance to those of renal calculus, should often have been misinterpreted.

Various explanations of these painful attacks have been suggested, but the most probable interpretation of the symptoms is that given by Landau—namely, that there is '*an intense local disturbance of the circulation in the movable kidney, caused by twisting, or kinking, or acute angular insertion of the renal vessels, especially the vein, in consequence of the change of position and rotation of the kidney.*'

The diagrams by which Landau's paper is illustrated render his explanation very intelligible and convincing.

The intense congestion of the kidney which would result from a twist of the renal vein explains, not only the pain and

swelling of the kidney, but also the scanty secretion of urine and the occasional presence of albumen and blood in the secretion.

One of my patients, Mr. M—— (p. 818), had his urine blood-tinged at the time of his visit to me, and, of the fourteen others, four had a trace of albumen without tube casts. The escape of albumen is probably due to the strain upon the Malpighian capillaries consequent on the occasional obstruction of the twisted renal vein (see fig. 6, p. 627).

Another result of the descent of the kidney from its normal position is that the *ureter*, which is normally placed at the lowest part of the renal pelvis, and therefore at the point most favourable for the escape of the urine, moves to a higher position, and if the kidney is very low, to the highest point of the pelvis. But if the kidney rotates (as often happens), the ureter will also become twisted on its axis. In the former case kinking of the ureter occurs, and in the latter, torsion; either of which would impede the escape of urine.

Landau adduces much evidence in support of his contention that *hydronephrosis* sometimes results from the frequently recurring obstruction of the ureter in a movable kidney.

On the other hand, hydronephrosis from other causes, might result in the downward displacement of the kidney by its own weight.

Diagnosis.—The diagnosis of a movable kidney is, as a rule, not difficult, if only the possibility of a tumour in the abdomen being of that nature be borne in mind. Palpation affords the most conclusive evidence of the nature of the tumour. The renal form and size of the mass, its smooth surface, the readiness with which it can be pressed upwards beneath the ribs, the sickening pain on pressure, together with the flattening in the loins, and the want of resistance there when the patient is resting on the hands and knees, are the most characteristic features of the tumour.

Mistakes have been made in opposite directions. A movable kidney has been mistaken for a tumour of the ovary or mesentery or omentum, and, on the other hand, a tumour not renal, has been supposed to be such.

It would seem to be *à priori* improbable that a distended

gall-bladder could become so movable as to be mistaken for a misplaced kidney, yet more than one such case has been recorded. Landau gives the history of a case in which a tumour beneath the right lobe of the liver was so freely movable downwards that it was at first supposed to be a misplaced kidney. At length, however, with rapidly increasing emaciation, jaundice occurred, and then the diagnosis of cancer of the gall-bladder was confirmed by the autopsy. One peculiarity of the tumour was that, when the downward pressure which removed it from beneath the liver was relaxed, it at once returned to its original position, which, on the supposition of its being renal, could have been accounted for only by its having formed adhesions to the under-surface of the liver.

Treatment.—The main object of treatment is to replace the kidney in its normal position, and to retain it there. This can, in most cases, be effectually accomplished by wearing a properly made and fitted elastic abdominal belt, with a pad placed beneath the ribs, so as to prevent, as far as possible, the descent of the movable kidney. I am in the habit of sending my patients to Mr. Heather Bigg, with instructions to him to make a suitable band and pad; and in most cases, when I have had the opportunity of ascertaining the result, this plan has been immediately and completely successful. For instance, Mrs. B——, æt. 32, a tall lady, who consulted me in May 1885, had suffered severely from the usual results of a movable kidney, since the birth of her only child three years before. I prescribed an abdominal belt, and when she called on me after wearing it for a fortnight she was greatly relieved, and 'felt quite a different woman.' Her husband, who came to consult me in November 1886, reported that she had remained quite well since wearing the abdominal support. In the case of Miss C——, before referred to (p. 817), the belt gave immediate relief, and she could, at once, walk more comfortably.

Mrs. G——, æt. 23, when I saw her, November 4, 1884, had had eleven attacks of severe pain from a movable kidney in fifteen months. Her health had greatly suffered, and she had lost a stone in weight. The pains had begun soon after her marriage, but she had not been pregnant; and

the probable cause of the misplaced kidney was extreme constipation, with resulting straining at stool. In addition to the means for obviating constipation, I prescribed the usual abdominal support. She wrote to me on December 30 that she was, as she said, 'a cure;' but, as she had found the original belt 'very uncomfortable,' she had replaced it by a flannel one of her own making, which she thus described: 'The belt I wear is simply two thicknesses of flannel shaped exactly like the lower half of one's stays, so that it goes under them without wrinkling. I button it behind, and it is nothing more than a sloping band about ten inches deep in front.' She had had no pain since wearing the support, and she added: 'I feel quite strong, and my friends say that I am looking my old self.' As I have not since heard from this lady, I conclude that she is free from her former malady.

Of the eleven patients for whom I have recommended the wearing of an abdominal support, six obtained immediate and, so far as I know, durable relief from great and prolonged suffering. In one case, that of Mrs. L——, sent to me by Dr. William Budd, of Exeter, in March 1885, Dr. Budd reports that 'she wore the belt for a time, and it afforded her a certain amount of support, but her attacks of pain and sickness are just the same.' One lady declared that she could not wear the belt after it was made for her. In the other three cases I have not heard the result, nor do I even know if the belt was obtained. One lady whom I saw with a movable right kidney, was in the early period of pregnancy, when, of course, a belt could not have been adjusted.

It is of importance that the abdominal belt and pad should be applied while the patient is in the recumbent posture, and that they should be so adjusted as to ensure that the kidney is supported and pressed upwards. A belt so applied as to cause downward pressure upon the misplaced kidney, would tend to aggravate the patient's suffering.

In addition to wearing a properly fitted abdominal support, these patients should avoid violent exercise, long standing, and all straining efforts, whether at stool or in any other way.

During the paroxysm of pain which results from twisting and obstruction of the renal vessels and the ureter, attempts

may be made, by changing the patient's position and by gentle manipulation, to restore the kidney to its normal position.

Although a large proportion of these patients are greatly relieved by the habitual use of an abdominal belt—as to which all writers on movable kidney are agreed—there will still remain a minority of cases, in which relief cannot be obtained by such means. The question will then arise whether surgical aid should be sought.

Two operations have been proposed and practised for the relief of the suffering caused by a movable kidney—namely, nephroraphy and nephrectomy.

Dr. Newman[1] has collected eight cases, including one operated on by himself, in which nephroraphy was completely successful. In all these cases, the kidney having been fixed against the lumbar parietes by sutures, the distressing symptoms which had long previously existed disappeared. It would appear, therefore, that when the symptoms are very severe, and when all other means have failed to afford relief, the operation of nephroraphy is advisable.

But nephrectomy is a much more formidable proceeding. I do not hesitate to advise a recourse to the surgical treatment of serious renal diseases, when the circumstances appear to justify, and even to demand, such treatment; in illustration of which I may state that, within the last three years, five of my patients have, by my advice, undergone the operation of nephrolithotomy, and in each case with complete success and relief from great and prolonged suffering.

The extirpation of a disorganised kidney is, under certain conditions, a justifiable proceeding. In one of my five cases before referred to, the kidney was so far destroyed by calculous nephritis, that it was deemed right to remove it, together with the stone; but I doubt if I shall ever be induced to advise or to sanction the extirpation of a kidney which, though movable and misplaced, is not otherwise abnormal. No case is recorded in which death has resulted from a movable kidney, but the operation of nephrectomy for that condition has, in several instances, been fatal.

[1] *Glasgow Medical Journal*, August 1883.

CHAPTER XLIX.

CASE OF ENCEPHALOID CANCER AFFECTING A TESTICLE WHICH HAD BEEN RETAINED WITHIN THE CAVITY OF THE ABDOMEN.[1]

Pain in Right Groin, exciting Suspicion of Renal Calculus—Right Testicle not descended—Loss of Flesh—Tumour in Abdomen increasing to an enormous size—The Testicle found after Death converted into a Medullary Tumour weighing Sixteen Pounds.

The subject of the following history was C. D——, æt. 27, a well-developed, muscular man, of active habits, a zealous boater, cricketer, and sportsman. When a child he was delicate, but after the age of eleven he enjoyed robust health, until he overworked himself in preparing for a scholarship examination at Cambridge, when he became dyspeptic and nervous. He first consulted me for these symptoms in the year 1853. He gradually recovered, worked for his degree, and graduated in honours. He then continued to reside at the University as a private tutor. The second occasion on which I heard of him as a patient was in the month of April in the present year (1858), when he wrote to me from Devonshire. His letter is dated April 9, 1858; and the following is his own account of the commencement and progress of the illness for which he then consulted me:—

'Last September, when out shooting on a very fatiguing beat on a hot day, an uneasiness in my *right* side,[2] which I had felt for a short time before, grew into such intolerable pain, that I had to knock up for a couple of hours. It went off, and I finished the day's sport without suffering much more pain. For a day or two afterwards the pain was severe. A

[1] *Medico-Chirurgical Transactions*, vol. xlii.

[2] The seat of pain was just above Poupart's ligament on the right side.

country medical man assured me it was muscular, and applied hot fomentations. The sensation was that of an aching of the *bone*, and did not appear to be near the surface. Up to the middle of January this uneasiness continued incessantly; sometimes, but not often, becoming positive pain. I rowed during the whole of November, but this did not cause any increase of uneasiness; on the contrary, I think it was less than at any other time.

'In January I went to two balls, and after the last the muscle above the hip was much swollen, and the pain increased so much that I consulted a surgeon in Cambridge. He, too, called it muscular; but by the middle of February the pain became so bad that, for four or five hours at a time, I could only find ease by sitting in a crouching posture. This would be the case two or three times a week, and I do not exaggerate in calling the pain at such times absolute agony. I also had frequently a severe contraction of the rectum, often expelling its contents involuntarily. The medical man changed his mind and thought the *caput coli* was at fault, and gave me mild aloetic pills.

'The urine is slightly high in its specific gravity, but clear and of good colour. The pain of passing water is very great at times. *Once*, while the pain was very intense, I passed some urine which left a thick deposit of mucus, and was quite turbid while warm.

'Just before I left Cambridge the surgeon told me that he had ultimately made up his mind that the pain was attributable to the passing of renal calculi along the ureters. But I have never had any pain in the kidneys.'

My first impression, on reading this account of the patient's symptoms, was that he had renal calculus; and I thought this the more probable from my knowledge of the fact, that, when he was under my care five years before, his urine contained oxalate of lime in great abundance. I now wrote and asked some questions which were suggested by this view of the case. What these questions were will be evident from his reply, which is dated April 11, 1858, and which I give in his own words:—

'As you have asked me questions regarding the retraction

of the testicle, I will at once make to you the confession, distressing to me to make, that I have no *right* testicle, nor do I remember ever possessing one. The left is of the ordinary size, and so far as I can judge, the proper and healthy operation of the sexual organs is not impaired by this defect; at least I have not experienced any inconvenience on this score. It is painful to me to impart this secret, which I have hitherto guarded with the greatest care, nor is there a soul besides yourself who is at all aware of it. True it is, that we have all of us "a skeleton in the cupboard" of some nature or another. There has been no retraction of the testicle of any consequence, nor has there been pain in the right thigh. My general health is pretty good; but having led a very sedentary life during the past winter, I am not in a *rude* state of health. Muscles rather relaxed and flabby. The pain has never produced a feeling of nausea. To-day I am upright again, but there is an aching towards the extremity of the glans penis, which I have not felt till within the last few days. I have now, I believe, answered all your questions.'

On April 13, he again wrote as follows: 'I enclose some sediment that I passed last night. The pain was worse yesterday than I have felt it for some time. I drank three hot glasses of weak gin and water last evening, and passed only about three-fourths of a pint, after an interval of at least ten hours since the last time of passing any urine. It has given me pain for some days past, as I told you, to pass any urine at all. To-day, with the exception of a slight uneasy feeling at the point of the hip, I feel as well as I ever did in my life. Yesterday, not able to walk one hundred yards, and doubled up. To-day, able to walk a couple of miles, which I am just going to put into practice, and perfectly upright. Surely I am not wrong in attributing this change to the passing of the sediment.'

The sediment here referred to was composed of the ordinary urate of ammonia, with numerous small octohedra of oxalate of lime. There was no trace of albumen in the urine, nor did the microscope reveal the presence of pus or mucus.

Up to this time I had not seen the patient, but on April 17 he came to me on his way from Devonshire to Cam-

bridge. His general appearance did not indicate any serious organic disease; there was no emaciation, and his limbs were firm and muscular. The pain of which he complained appeared to be near the bladder on the right side, but there was no irritability of the bladder, and there seemed no reason to suspect the existence of a calculus either in the kidney, ureter, or bladder.

The recti muscles appeared to be unusually firm and rigid; but with this exception I detected nothing abnormal in the state of the abdomen after a most careful examination, and I could form no opinion as to the nature of the disease.

May 31.—I saw him again in London. In the meantime he had been suffering more or less constantly from the pain low down in the right side of the abdomen. On May 21 he had joined in a cricket match, and while engaged in the game he was suddenly seized with unusually severe pain, which compelled him to lie down for some time. He was then carried home and placed in a warm bath. From that time the pain had been more severe, and his condition had very much changed for the worse. He now walked in a bent position with much pain and difficulty; he had lost flesh considerably, had a pale, anxious, haggard look, and was convinced that his disease would, as he said, 'end in the churchyard.'

He returned to Cambridge, where he continued to lose flesh and strength, and to suffer from severe and almost constant pain. The nature of the disease being still doubtful, I gladly acceded to the patient's wish to have another opinion upon his case; and I arranged a consultation with Dr. Bright on June 12, the patient again coming up from Cambridge for the purpose.

In giving the history of the case to Dr. Bright, I told him that I had ventured to make a guess at a diagnosis. Connecting all the facts which have already been mentioned with one not before alluded to—namely, that the patient's mother had died of cancer of the stomach—it seemed to me not improbable that the right testicle, retained in the abdomen, had become the seat of malignant disease, and that in this might be found the explanation of the long-continued pain, and of the recent rapid emaciation. We then proceeded to examine

the patient's abdomen, and Dr. Bright pointed out, what was now sufficiently evident, that just above Poupart's ligament on the right side, there was a growth or deposit either in the abdominal wall or immediately behind it; this portion of the abdomen being hard and resisting under pressure, and yielding a dull sound on percussion. There was, besides, considerable fulness of the whole lower part of the abdomen. Dr. Bright's opinion was that there was a deposit in the abdomen, perhaps cancerous, and he thought it possible that the retained testicle might be, as I had suggested, the primary seat of the disease. From this time the progress of the case was very rapid. After the consultation with Dr. Bright the patient returned to Cambridge, where he remained about a week; he then went to stay at the house of a relative about ten miles from London, where I was asked to see him on June 29.

I found him wonderfully altered, very pale, and much emaciated. He was in bed, though he had been down-stairs the day before my visit. The tumour in the abdomen had greatly increased, extending now beyond the median line and considerably above the umbilicus. It was quite immovable under pressure, and appeared to be closely adherent to the abdominal wall. The whole surface of the tumour was dull on percussion, but there was resonance in the region of the ascending colon. The greatly distended abdomen had very much the appearance observed in a case of large ovarian cyst. During the previous day or two, there had been occasional bilious vomiting, which he had encouraged by copious draughts of warm liquids.

I did not see him again alive. He became rapidly weaker, and died on July 7, retaining his consciousness to the very last.

Forty-seven hours after death I examined the body with my friend Mr. Spurrell, who had been in daily attendance upon the patient after he left Cambridge.

The body was much emaciated. The tumour had increased considerably since my visit on June 29; it now extended far over to the left side, and as high as the epigastrium. It felt firm and hard through the wall of the belly.

The whole anterior surface of the tumour adhered to the wall of the abdomen, and there were some adhesions about the right iliac fossa. The adhesions were broken down without much difficulty, and the tumour was removed. When *in situ*, it occupied a large part of the abdominal cavity. The small intestines were pushed upwards and to the left, and the liver was pushed up beneath the ribs; the tumour formed no adhesions to any of the viscera. In removing the tumour, about four pints of dirty grumous liquid escaped from some of the large cysts contained in its substance. The weight of the mass after the escape of this liquid was sixteen pounds; its original weight could not have been much under twenty pounds. Its dimensions were as follows: Length, fourteen inches; breadth, twelve inches; thickness, from three to six inches. At the under surface of the tumour was a projecting body, about the size of a duck's egg; its surface was smooth, covered by a bluish-white capsule, which above was spread out and gradually lost over the tumour. The vas deferens, quite normal, passed to the back of this body, and some fibrous tissue went from its anterior surface to the internal abdominal ring. This body was evidently the right testicle, which above was spread out into the large tumour already described. On section the testicle was found degenerated into a cancerous mass. My friend Dr. (now Sir) Andrew Clark examined it, and found in it distinct traces of its original tubular structure. The testicle and the whole of the diseased mass into which it had grown had the usual characters, both to the naked eye and under the microscope, of fungus hæmatodes. The tumour was, for the most part, a soft, solid mass, but it contained many cysts, varying in size from that of a hazel-nut to that of an orange. One cyst was as large as a good-sized cocoa-nut. Many parts of the diseased mass were infiltrated with blood. All the other viscera in the abdomen were quite healthy. The chest was not examined.

[The chief reason for the republication of this case has been the consideration that a knowledge of its remarkable history may assist some practitioner in the diagnosis of any similar case that may occur hereafter.]

CHAPTER L.

SOME PRACTICAL HINTS RELATIVE TO LARYNGOSCOPY, AUTO-LARYNGOSCOPY, AND RHINOSCOPY.[1]

The Convenience of having the Frontal Reflector above the Eyes rather than with a Perforation in front of the Right Eye—The Use of a Shade below the Reflector—The Author's Simple Method of Auto-laryngoscopy and Laryngoscopic Demonstration—Mode of practising Rhinoscopy—Case illustrating its Practical Value—Dr. Fränkel's Mouth-dilator.

I HAD the advantage of commencing the study and practice of laryngoscopy under the teaching and guidance of the late Professor Czermak, for whom I got together a class of physicians and surgeons, who met for a course of practical instruction at King's College Hospital. We were all taught to use a perforated concave reflector, held in front of the right eye; and I continued to work with this for some time before I adopted the plan of placing the reflector on the forehead above the eyes. I found this so much more convenient than the other method, that I have ever since continued to use it. It is quite unnecessary, and therefore undesirable, to look through a perforated reflector placed in front of one eye. With the reflector on the forehead, we avoid the discomfort and inconvenience resulting from the effort required to keep one eye applied to the opening in the mirror; we have the free and unimpeded use of both eyes; and we consequently find it much easier to direct a steady light into the throat, to introduce the laryngeal mirror, and to practise any other manipulation which may be required, either for diagnosis or for treatment.

Another great advantage resulting from this position of the reflector is that, by means of a shade attached to the

[1] This chapter should properly have preceded Chapter XIX.

frontal band, we completely protect the eyes from the direct light of the lamp, and thus avoid the fatigue and the confusion of sight which result from exposure to a bewildering glare of light (fig. 42). When the direct rays of the sun are used for the illumination of the throat, it is especially important to be able to protect the eyes from the otherwise blinding glare.

The practice of using a perforated reflector was borrowed from the opthalmoscope, in using which instrument it is necessary to look through the centre of the luminous beam; but the conditions which attend the exploration of the interior of the eye through the small opening of the pupil, are very different from those which exist when we are looking through the wide-open mouth, at an image reflected from a mirror of

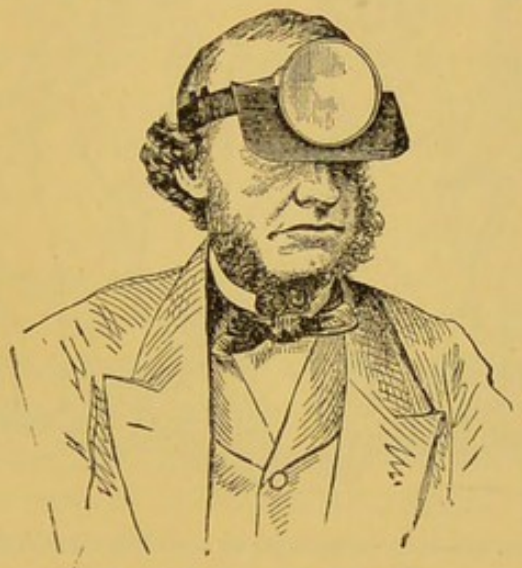


FIG. 42.—FRONTAL REFLECTOR AND SHADE.

considerable size. In the latter case, nothing is gained by looking through a perforated reflector, and the disadvantages I have already pointed out.

I have no doubt that many students have been discouraged by the difficulties attending the attempt to see the larynx through a hole in the reflector.

One of the most useful means of acquiring skill and confidence in the examination of the larynx, is the practice of auto-laryngoscopy—that is, the examination of one's own larynx. The simplest, and, on the whole, the most satisfactory method of auto-laryngoscopy was devised by myself

many years ago.[1] This method of auto-laryngoscopy requires no special apparatus. Sitting at a table of convenient height, I place a common looking-glass in front of me, and a moderator or gas lamp on one side of the glass, but two or three inches further back, so that the light may not pass directly from the lamp on to the face of the glass. Now, with the reflector on my forehead, I direct the light from the lamp, as it were, into the open mouth of my own image in the looking-glass; then, introducing the laryngeal mirror into my mouth, I see the reflection of my larynx in the glass before me, and any

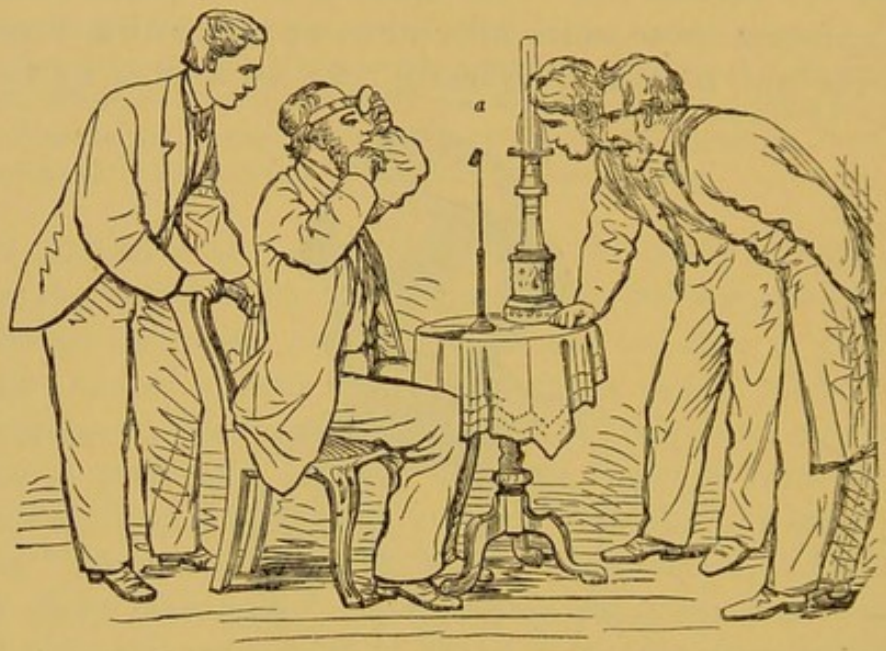


FIG. 43.—AUTO-LARYNGOSCOPY AND DEMONSTRATION.
a. The small mirror on a brass stem.

one looking over my shoulder can also see the reflected image. If I wish to demonstrate my larynx to several persons at once, I do this by having the mirror in front of me of small size—about three inches by two inches (fig. 43). Thus, while three or four persons standing behind me can see the reflection of my larynx in the mirror in front, three or four others, standing before me and looking over or under or by the sides of the demonstrating glass, can see the reflection of my larynx from the laryngeal mirror which I hold in my mouth. Those who stand in front of me have a somewhat better view than those

[1] See *Medical Times and Gazette*, February 14, 1863, p. 157.

who look over my shoulder from behind, for the obvious reason that some loss of light attends the second reflection from the demonstrating-mirror before the image reaches the eye.

For beginners in the art of laryngoscopy, this method affords a very useful means of training and practice. One of the chief difficulties at first is to keep a steady light in the patient's mouth while the laryngeal mirror is being introduced. Now, the student, after arranging his looking-glass and his lamp, may direct the light into his own open mouth in the looking-glass. This process differs scarcely at all from that which he will have to practise on his patients. Then, having learnt to keep the light steadily and automatically directed into the mouth, he may warm and introduce the laryngeal mirror, and he will soon see his own larynx.

The frontal reflector is a very useful means of lighting the throat for the purpose of examining the tonsils, palate, and pharynx. Placing a lamp or a candle by the side of the patient, the operator, with the reflector on his forehead, throws the light into the throat, and has both his hands free to depress the tongue and to apply caustic or other local remedies. In cases of diphtheria and scarlet fever, by this method of illumination a thorough examination of the throat can be made in a much shorter time than by the ordinary method, and without raising the patient's head from the pillow. The operator, too, in this way, runs less risk of infection from inhaling the patient's breath, or from the morbid secretions being coughed into his face.

Rhinoscopy.—We may often obtain useful information by an examination of the posterior nares with a mirror. In the practice of rhinoscopy, as it is called, the patient sits erect, while the light is thrown into the mouth by the frontal reflector. The tongue is held down within the mouth by a right-angled metal depressor. The patient is directed to breathe softly. A forcible inspiration draws the soft palate and uvula upwards and backwards, and so interferes with the examination. A mirror, about the size of a threepenny-piece, is warmed and introduced by the side of the uvula, beneath the palate, with its surface directed upwards and forwards; care being taken not to touch the soft palate or uvula, so as

to excite reflex contraction of these parts. In this way, when the throat is moderately capacious, we may bring into view the posterior openings of the nasal fossæ, the turbinated bones, the opening of the Eustachian tube, the septum narium, and the roof of the pharynx.

In illustration of the practical value of rhinoscopy, I may refer briefly to one case. A gentleman, twenty-four years of age, consulted me on account of complete obstruction of the right nostril, which had existed for two years. On examination with the mirror, the posterior opening of the right nasal fossa was seen to be obstructed by a globular tumour (fig. 44), as

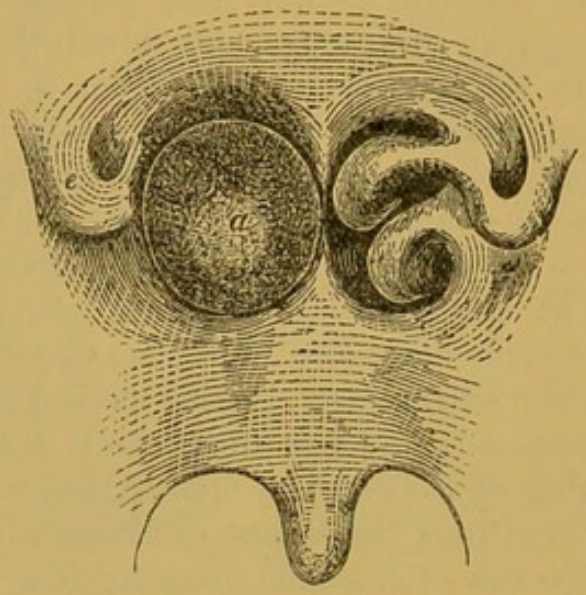


FIG. 44.—POSTERIOR VIEW OF THE UVULA, SOFT PALATE, AND NARES, AS SEEN BY RHINOSCOPY.

a. A globular tumour obstructing the posterior opening of the right nasal fossa.
e. the orifice of the right Eustachian tube.

large as a full-sized marble, and of a yellowish-green colour. I asked my colleague, Mr. John Wood, to devise a plan for removing the tumour. He introduced a slender curved polypus-forceps through the anterior opening of the nostril, and grasped the tumour, which burst, and discharged a glairy fluid like white of egg. The patient felt immediately that the obstruction was removed; and, on rhinoscopic examination, the shreddy remains only of the tumour were seen attached to the middle turbinated bone, which had before been concealed by the tumour (fig. 45). The tumour had evidently been a mucous cyst. This case occurred more than twenty years ago;

and I heard quite recently that there has been no return of the disease. The practical value of rhinoscopy in this case can scarcely be questioned. It is doubtful whether, by any other mode of examination, the position and nature of the tumour could have been determined with sufficient certainty to warrant an operation for its removal. Before the patient came to me

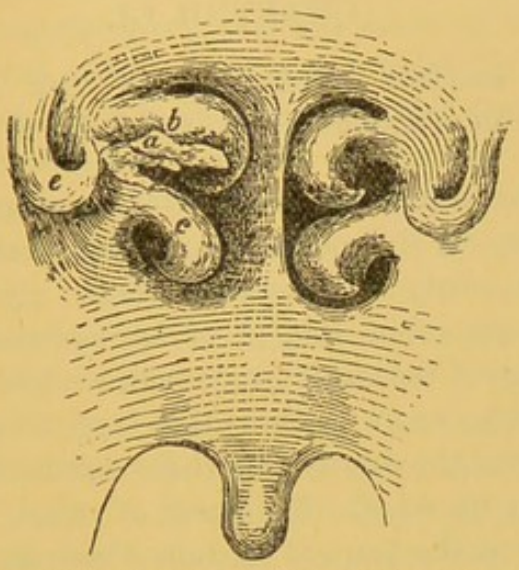


FIG. 45.—THE POSTERIOR NARES, AFTER THE REMOVAL OF THE TUMOUR REPRESENTED IN FIG. 44.

a. The abraded surface from which the tumour was torn. *b*, the middle; and *c*, the inferior turbinated bone. *e*, the Eustachian tube.

he had consulted a very eminent surgeon, who by probing the nostril, had failed to discover the seat and nature of the obstruction.

At the London International Medical Congress (1881) Dr. B. Fränkel, of Berlin, exhibited his modification of Whitehead's mouth-dilator, consisting in the addition of a tongue-depressor, which forms part of the instrument itself, and of a self-retaining Voltolini's palate-hook, which is fixed to the instrument after introduction, by means of a simple screw. By this instrument the tongue is depressed, while the uvula and soft palate are so hooked upwards and forwards as to greatly facilitate the practice of rhinoscopy.[1]

[1] *Transactions of the International Medical Congress*, 1881, vol. iii. p. 333.

CHAPTER LI.

THE HARVEIAN ORATION DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS, JUNE 24, 1882.

MR. PRESIDENT AND GENTLEMEN,—After accepting the invitation with which you, Sir, honoured me, to undertake the arduous and responsible duty of Harveian orator, my first intention was to take as the main subject of my address the additions o our knowledge of the vascular system, with its nerves supply, which have been made within the last thirty years; and thus to continue the history of these interesting discoveries from the point at which, for want of time, it was left by Dr. Sanderson in the learned oration which he delivered here four years ago. On further consideration, however, it appeared to me that my attention should, by preference, be directed to the systematic attempts which have recently been made in Italy to rob our illustrious Harvey of the honour which for two centuries and a half has, by almost universal consent, been conceded to him, and to claim for the Italian Cesalpino (Latin, Cæsalpinus) the credit of having anticipated Harvey in the discovery and demonstration of the circulation of the blood. These attempts to exalt Cesalpino at the expense of Harvey have been referred to and refuted by the late Dr. R. Willis, in his admirable volume entitled *William Harvey*.[1] But the subject is so important and so full of interest for this college that it may well occupy all the time at my disposal to-day.

It will be remembered that Dr. Sieveking, in his interesting oration delivered in 1887, referred to the fact that a monument in honour of Cesalpino had recently been unveiled in Rome. I am indebted to the kindness of Dr. Pantaleoni, the eminent

[1] London, 1878.

Roman physician and senator, for a copy of the two orations [1] which, at the inauguration of that monument, were delivered in the presence of a large assembly of learned professors and other eminent and representative men of Italy. The orators were Professors Scalzi and Maggiorani. The former spoke of Cesalpino as an eminent anatomist, botanist, and mineralogist; while the latter referred to him as a distinguished philosopher. Both these learned professors, in the course of their addresses, mentioned in the terms of praise a work by Dr. Ceradini,[2] professor of physiology in the University of Genoa, the second edition of which had been recently published.

In this volume of three hundred royal octavo pages the author professes to give a true history of the discovery of the circulation of the blood. Professor Scalzi declares the work to be a 'publication of the highest interest for physiological science and the history of medicine;' [3] and Professor Maggiorani,[4] at the conclusion of his address, expresses gratitude to 'Professor Ceradini, who on this festive occasion has presented to the Academy his learned volume, wherein that which heretofore had been only more or less a belief as to Cesalpino's discovery, has become, by means of new arguments, a scientific demonstration.' Professor Ceradini's volume, therefore, is a work of authority and influence—at any rate among his fellow-countrymen—and as such I have deemed it worthy of a careful study and analysis, with results which I will presently endeavour to set before you. A critical examination of this imposing volume is rendered more necessary by the fact that Cesalpino's published writings being very scarce, and to be found only in large libraries, Dr. Ceradini's version of their physiological teaching will be accepted as

[1] *Inaugurazione della lapide ad Andrea Cesalpino;* due discorsi letti in questa occasione dai Prof. F. Scalzi e C. Maggiorani. Roma, 1876.

[2] *La Scoperta della Circolazione del Sangue*, del Dott. G. Ceradini, Prof. di Fisiologia all' Università di Genova. Milano, 1876.

[3] 'Pubblicazione di sommo interesse per la scienza fisiologica, e per la storia della Medicina' (p. 23, note).

[4] 'Giustizia vuole che in tal momento si esprime un senso di riconoscenza al Prof. Giulio Ceradini che si fece occasione di questa festa col dono fatto alla nostra Accademia del suo dotto volume, ove quel che dianzi non era stata che voce più o meno accreditata intorno alla scoperta di Cesalpino, divenne per i nuovi argomenti una dimostrazione scientifica' (p. 62).

trustworthy by all who have not both the opportunity and the inclination to compare his statements with the original treatises.

In the preface to his book Dr. Ceradini suggests, with an evident feeling of satisfaction, that the determination of the Royal Medical Academy of Rome to place within the walls of the University an engraved tablet declaring Cesalpino to be the discoverer of the circulation of the blood, may have been influenced by the more complete demonstration of Cesalpino's claims which he had been able to give in the first edition of his historical treatise.

I now proceed to quote, as fully as time permits, the chief statements and arguments upon which Dr. Ceradini relies to establish his position that the Italian Cesalpino was, and that our English Harvey was not, the actual discoverer of the circulation of the blood, and I will endeavour to estimate at its true value the judgment of Harvey's critic and accuser. Dr. Ceradini's statement with regard to Harvey is to this effect—that during the four years from 1598 to 1602, which Harvey spent as a student at Padua, he must have become acquainted with Cesalpino's writings,[1] some of which had been published about thirty years before; that in these writings Harvey must have seen that the true doctrine of the circulation of the blood was clearly set forth and completely demonstrated; that Harvey designedly delayed the publication of his work, *De Motu Cordis et Sanguinis*, until 1628, twenty-five years after the death of Cesalpino, and nine years after the death of Fabricius, when his adversaries could adduce no proof that his affected ignorance of the discovery of Cesalpino was a mere pretence.[2] Ceradini quotes the well-known passage in which Harvey expresses his fear lest, through the novelty of his discovery, he should have all mankind for his enemies;[3] and on this he makes the following comment: 'Without doubt by these subterfuges the Englishman designed to usurp for himself the glory of a discoverer.'[4]

[1] Ceradini, p. 171. &c. [2] P. 172.

[3] College edition, p. 47; Dr. Willis's translation, p. 45.

[4] 'Nessun dubio che con questi sotterfugi l'Inglese mirasse ad usurpare il vanto di scopritore' (p. 175).

It is interesting to note that Harvey's fears were not without reason. 'John

Harvey's hostile critic asserts that his doctrine of the general circulation was based almost exclusively on the presence of valves in the veins,[1] which had been first discovered or at any rate more fully demonstrated and described, by Harvey's anatomical master, Fabricius; and Ceradini affirms that this evidence in support of the doctrine of the circulation is all that Harvey could add to Cesalpino's prior and complete demonstration. Referring to the Life of Harvey prefixed to the Latin edition of his works which was published by the Royal College of Physicians in 1766, Dr. Ceradini says: 'It lacked the foundation of truth, and therefore, notwithstanding the efforts of the biographer and of his natural allies, the whole College of Physicians, the defence of Harvey was so void of reason, so audaciously partial, so utterly valueless.'[2]

The author admits with regret that some eminent physiologists had pronounced in favour of the claims of Harvey to be the real discoverer of the circulation of the blood; but this erroneous verdict he attributes either to ignorance or to bad faith. In particular he declares his belief that the illustrious Haller[3] and two of Cesalpino's own countrymen, Malpighi and Baglivi,[4] had their judgments perverted by the embarrassing fact that they had been elected Fellows of the Royal Society of London.

Aubrey tells us that he had heard him (Harvey) say that after his book on the "circulation of the blood" came out, he fell mightily in his practice; 'twas believed by the vulgar that he was crack-brained, and all the physicians were against him.'—Dr. R. Willis's *William Harvey*, p. 165.

[1] P. 275.

[2] 'Mancava però al biografo il fondamento della verità, ed ecco perchè malgrado gli sforzi suoi e quelli dei naturali suoi alleati, i colleghi tutti del Collegio Medico di Londra, la difesa di Harvey riesciva cosi vuota di ragioni, cosi sfacciatamente parziale, cosi nulla' (p. 271).

[3] Haller's judgment was as follows: 'Adparet non Cæsalpino, ob paucas aliquas et obscuri sensus voces, sed Harveio, numerosissimorum experimentorum laborioso auctori, gravique scriptori argumentorum omnium quæ ex ævo proferri poterant immortalem gloriam inventi circuitus sanguinis deberi.'—*Elementa Physiologiæ*, vol. i. L. III. sect. 3, § 32.

[4] Upon this Dr. Ceradini remarks: 'Singolare giudizio davvero! ma che sarebbe anche più singolare, se Haller non si fosse trovato nella stessa difficile condizione di Malpighi e Baglivi, nella condizione cioè di membro della Reale Accademia di Londra' (p. 266). [4] P. 205.

Ceradini allows, as an excuse for Harvey's pretensions, that, inasmuch as he had contributed somewhat to the complete demonstration of his predecessor's doctrine, and had undergone great labour in his endeavour to make it known, and to overcome the infinite prejudices by which it was opposed, he may at length have persuaded himself that Cesalpino's discovery was actually his own. But he proceeds to say that these considerations, while they in part justify Harvey's conduct, avail not to excuse that of his fellow-countrymen, who to this day, in spite of truth and justice, believe, or feign to believe, him to be the discoverer of the circulation of the blood—perhaps he sarcastically adds, in order not to deprive themselves of the pretext for an annual celebration of his memory *inter pocula*.[1]

I will venture to say that if Dr. Ceradini had been a contemporary of Harvey he would have received from the object of his attack no other notice than that contained in the following sentence: 'Detractors, censurers (momos), and writers defiled with abuse, as I have resolved with myself never to read them, satisfied that nothing solid or excellent, nothing but reviling was to be expected from them, so have I held them still less worthy of an answer.'[2]

In my endeavour to refute these monstrous charges against the greatest ornament of our College, and one of the greatest benefactors of the human race, I shall make no attempt to emulate the quite inimitable good taste and style of the Genoese Professor; but I shall endeavour, and I trust successfully, to show that, however diligent may have been Harvey's study of Cesalpino's writings, he could never have obtained from them that which is not to be found therein, viz., a knowledge of the circulation of the blood; and that those who pretend to find in these writings the true doctrine of the circulation, endeavour to establish their position by

[1] 'Ma queste considerazioni, se giustificano in parte la condotta di Harvey, non valgono affatto di scusa a quella de' suoi connazionali, i quali oggi ancora a dispetto della verità e della giustizia lo ritengono, o fingono ritenerlo scopritore della circulazione del sangue, forse, per non privarsi, come argutamente osservava Barzelotti, del pretesto di festeggiarne la memoria *inter pocula*' (pp. 298–9).

[2] College edition, p. 109; Dr. Willis's translation, p. 109

giving to some chance expressions a meaning which the context shows could never have been in the mind of their author; while interpreting Cesalpino's vague and contradictory statements by the light of Harvey's researches, they ungratefully turn upon the real discoverer and accuse him of conscious plagiarism. This surely is very like an attempt to pierce the breast of an eagle with an arrow feathered by a plume plucked from his own wing.

If Cesalpino's discovery and demonstration of the course of the blood were so complete and unmistakable as his recent advocates maintain, it is remarkable that his contemporaries and immediate successors, to whom his writings must have been well known, should have remained in ignorance of the true doctrine of the circulation. Professor Scalzi, indeed, in his inaugural address, suggests that Harvey may have learnt the new doctrine of Cesalpino from his famous anatomical teacher Fabricius;[1] but, unfortunately for this suggestion, the work of Fabricius (*De Venarum Ostiolis*), which was published in 1630, a year after Harvey's departure from Padua, and rather more than thirty years after the publication of Cesalpino's chief treatise,[2] affords conclusive evidence of its author's entire ignorance of the circulation of the blood through the systemic vessels.

Fabricius believed that the purpose of the valves in the veins was not to favour the passage of blood to the heart, but to prevent over-distension of the veins by the blood in its passage through the venous trunks to their branches, and also to retard the current of blood, so that time might be given for each part to take up its proper nutriment; and he states that valves are not required in the arteries, because, on account of the thickness and strength of their coats, they are not liable to be over-distended. Neither are valves required to retard the stream of blood, because in the arteries there is a perpetual flux and reflux of blood.[3] This, then, was the doc-

[1] 'Fu gran ventura per Guglielmo Harvey, che trovandosi dal 1598 al 1602 in Padova allo studio della medicina, potesse apprendere la dottrina novella da Fabrizio d'Acquapendente, al quale il grido di anatomico sommo chiamava scolari anche da oltralpe' (pp. 18, 19).

[2] *Quæstionum Peripateticarum libri quinque*, Florent. 1571.

[3] 'Erat profecto necessaria ostiolorum constructio in artuum venis, ut scilicet

trine of the systemic circulation held and taught by the most eminent anatomist of Italy thirty years after the publication of Cesalpino's supposed discovery.

Professor Ceradini even admits that Fabricius had not the most remote idea of a circulation of the blood.[1] Upon this Dr. Willis pertinently asks: 'If this be true, who among his contemporaries could be better informed?' If Cesalpino had given an intelligible account of the circulation through the systemic vessels, his fellow-countryman Fabricius, of all men, would have been the least likely to be ignorant of it, and his ignorance may be be taken as a fair index of the knowledge of his contemporaries.

Now, in order to correctly estimate Cesalpino's claims as an original observer, and to understand some of the terms which he employs, we have to consider to what extent a knowledge of the circulation had been obtained by his predecessors.

From the time of Galen, in the second century of the Christian era, to that of Servetus, in the sixteenth century, it was supposed that the blood, passing from the vena cava into the right side of the heart, was there divided into two streams, which took different courses. While a part of the blood passed by invisible pores through the septum from the right to the left side of the heart, the other portion was sent by the pulmonary artery (vena arterialis) to nourish the lungs. But as all the blood thus sent to the lungs was not required for their nutrition, a certain portion passed into the pulmonary vein (arteria venalis) by means of a supposed anastomosis between the two vessels, and so reached the left side of the heart. The Spaniard Michaelis Servetus made an advance upon Galen's physiology of the pulmonary circulation in this respect, that he maintained that the blood passes from the right to the left side of the heart, not at all by invisible pores in the septum, but entirely by free communication between the pul-

sanguis ubique eatenus retardaretur, quatenus cuique particulæ alimento fruendi congruum tempus detur. Arteriis autem hæc ostiola non fuere necessaria; neque ad distensionem propter tunicæ crassitiem ac robur, neque ad sanguinem remorandum, quod sanguinis fluxus refluxusque in arteriis perpetuo fiat.'—Fabricius, *De Venarum Ostiolis*, p. 2.

[1] 'È notissimo del resto che Fabricio non ebbe la più lontana idea di una circulazione del sangue.'—Ceradini, p. 148.

monary artery (vena arterialis) and the pulmonary vein (arteria venalis), the dark blood from the right side of the heart assuming a crimson colour in its passage through the lungs.[1]

The Italian Realdus Columbus, who succeeded Vesalius as Professor of Anatomy at Padua, and who died at Rome in 1577, published his work *De Re Anatomicâ* six years after the publication of the *Christianismi Restitutio* of Servetus.

In this book Columbus claims to have been the first to describe the passage of the blood from the right to the left side of the heart by the pulmonary vessels. It is certain that in the date of publication Servetus had anticipated him by six years; but it is possible that the writings of Servetus, which, together with their author, were burnt at the stake at the instigation of Calvin, may not have been known to Columbus. Professor Ceradini suggests[2]—but so far as I can see without adducing any evidence in support of his conjecture—that Servetus may have been a pupil of Columbus at Padua, and may there have learnt from his anatomical teacher the doctrine which he afterwards published as his own.

What was known then and taught with regard to the circulation before the publication of Cesalpino's writings was briefly this—that the blood from the vena cava enters the right side of the heart, and thence passes through the lungs to the left side, whence it is distributed by the aorta over the whole body. What we now call the pulmonary circulation, therefore, was fairly well understood. With regard to the systemic circulation, however, nothing definite was known. It was supposed that while the veins conveyed one kind of blood called *auctive* blood to the tissues, the arteries supplied them with *nutritive* blood of a more spirituous nature; that there were communications between the arteries and veins by invisible inosculations or anastomoses, as the result of which auctive blood passed into the arteries, which in their turn gave back spirituous blood to the veins. By the anastomoses

[1] 'Fit autem communicatio hæc non per parietem cordis medium, ut vulgo creditur. Sed magno artificio a dextro cordis ventriculo, longo per pulmones ductu, agitatur sanguis subtilis: a pulmonibus præparatur, flavus efficitur, et a venâ arteriosâ in arteriam venosam transfunditur.'—*Christianismi Restitutio*, 1553, p. 169.

[2] P. 99.

Galen explained the fact that the wound of a large artery empties not only the arteries but the veins. The movement of blood in the vessels, both arteries and veins, was supposed to be of a to-and-fro character. From the time of Aristotle to that of Cesalpino, and, as we shall presently see, by Cesalpino himself, this movement was compared to the tides of Euripus—that is, to the ebb and flow of the tide in a narrow channel. This, then, was the state of knowledge, or rather of ignorance, with regard to the movement of blood in the systemic vessels before the publication of Cesalpino's writings. We have now to inquire to what extent he succeeded in throwing light upon the subject.

With regard to the structure of the heart and its valves, Cesalpino says that it is so arranged as to allow of continuous motion from the veins to the heart, and from the heart to the arteries.[1] In these statements there was nothing new. In the chapter 'De Pulmonis Constitutione' he says[2] 'the hot blood is carried from the right ventricle by the artery which Galen calls the vena arterialis into the lung, and is again conveyed to the heart by the vein proceeding from the left ventricle, which Galen calls the arteria venalis. Meanwhile, in its passage, the blood is tempered by the cold air inspired into the branches of the windpipe which lie near the veins and arteries, so that by a kind of circulation the blood is converted into the nature of spirit, first in the right ventricle, then in the left. Therefore, the vessel leading from the right side of the heart is a true artery, having a double tunic, in order that the spirits should not escape from it; the vessel entering the left side of the heart is a vein consisting of a single tunic, because it contains blood which has been already refrigerated in the lung.'

[1] 'Ut continuus quidam motus fieret ex venis in cor et ex corde in arterias.'—*Speculum Artis Medicæ*, lib. vi. cap. xix., edition 1670, p. 473.

[2] 'Fertur igitur ex corde sanguis fervidus per arteriam ex dextro ventriculo, quam Galen venam arterialem vocat, in pulmonen iterumque cordi redditur per venam ex sinistro ventriculo prodeuntem, quam Galen arteriam venalem vocat. Interim in itinere contemperatur ab aere frigido inspirato in asperas arterias juxta venas et arterias, ut circulatione quadam sanguis perficiatur in naturam spiritus, prius in dextro ventriculo deinde in sinistro. Ideo vas educens e corde vera arteria est ex duplici tunicâ, ut spiritus non evanescant; vas introducens vena est ex unica tunica constans, quia sanguinem jam refrigeratum continet.'—*Spec. Art. Med.*, lib. vi. cap. ix. p. 443.

Cesalpino's second reference to the circulation through the lungs occurs in the following terms:[1]—'Thus the lung, drawing warm blood from the right ventricle of the heart through the vein which resembles an artery (pulmonary artery), and sending it by anastomosis to the arteria venalis (pulmonary vein), which enters the left ventricle of the heart, tempers it meanwhile by the cold air which is conducted into the branches of the windpipe which lie near the arteria venalis, and this, not by inosculation between the blood-vessels and the air-tubes, as Galen supposes, but by contact alone. With this circulation of the blood from the right ventricle of the heart through the lungs to the left ventricle the appearances on dissection exactly correspond, for there are two vessels connected with the right ventricle and two with the left; but of the two vessels one only intromits, while the other emits, the valves being constructed with that design.' He then refers to the errors of his predecessors in calling all the blood-vessels connected with the right side of the heart veins, while those on the left side are designated arteries; and he says that the vessel on the right side, which had hitherto been called vena arterialis, has the structure of an artery, and pulsates in consequence of receiving blood from the heart, while the vessel on the left side, called arteria venalis, has the structure of the other veins, and does not pulsate, because it only conveys blood to the heart.'[2] 'All things, therefore, are

[1] '*Quæstionum Peripateticarum libri quinque*,' lib. v. p. 125 D, edition 1593. 'Idcirco pulmo per venam arteriis similem ex dextro cordis ventriculo fervidum hauriens sanguinem, eumque per anastomosim arteriæ venali reddens, quæ in sinistrum cordis ventriculum tendit, transmisso interim aere frigido per asperæ arteriæ canales, qui juxta arteriam venalem protenduntur, non tamen osculis communicantes, ut putavit Galen, solo tactu temperat. Huic sanguinis circulationi ex dextro cordis ventriculo per pulmones in sinistrum ejusdem ventriculum optime respondent ea quæ in dissectione apparent. Nam duo sunt vasa in dextrum ventriculum desinentia, duo etiam in sinistrum. Duorum autem unum intromittit tantum, alterum educit, membranis eo ingenio constitutis.'

[2] The pulsation of the pulmonary artery was known to Galen: 'Neque te fugiet vas illud quod cordi connectitur, a cava vena progerminatum, etiam ipsum quemadmodum arteriæ pulsare.'—Galen, *De Utilitate Respirationis* (c. 4). The probable reason why the ancient anatomists called the pulmonary artery a vein, and the vein an artery, is that the former is usually found more or less distended with blood after death, while the latter, being often found empty, was believed to contain only air.

admirably fashioned, for since it was necessary that the blood should be brought to the heart in order that it might become perfect nutriment—first in the right ventricle, in which a thicker blood is contained, and then in the left, which has a purer blood—it is transmitted from the right to the left ventricle, partly through the septum, partly, for the sake of being refrigerated, through the lungs' (note *a*, p. 863).[1]

From the preceding extracts it appears that Cesalpino's account of the pulmonary circulation is identical with that given by his predecessors, Servetus and Columbus, except that in one sentence (being apparently still under the influence of Aristotle and Galen) he speaks of the passage of blood from the right to the left side of the heart as taking place partly through the septum. Dr. Ceradini argues that, since Cesalpino refers to the permeability of the septum cordis only once in one of his works, he did this rather in deference to his admired master Aristotle, and not from a firm belief in the doctrine. But surely a disciple who quotes his master's doctrine without expressing dissent must be held to accept and agree with it. Cesalpino, being ignorant of the real function of the heart and arteries, thought that the purpose of the thick pulmonary artery was to prevent the escape of the spirits, while the thin pulmonary vein sufficed to hold the blood which, as he erroneously supposed, had been cooled by passing through the lungs.

It is a fact that the word *circulation* is here for the first time used to describe the movement of the blood from the right to the left side of the heart. Cesalpino's fellow-countrymen make much of this word, and argue as if it implied a knowledge, not only of the pulmonary, but also of the systemic circulation; but it is a noteworthy and most significant fact that this term is employed by Cesalpino only with reference to the passage of the blood through the lungs, and never in his attempt to explain the movement of the blood in the systemic vessels. This restriction of the word circulation to the pulmonary part of the circuit is *pro tanto* evidence that he was ignorant of there

[1] 'Partim per medium septum partim per medios pulmones, refrigerationis gratia, ex dextro in sinistrum transmittitur.'—*Quæst. Per.*, p. 126 *A*.

being a continuous passage of blood from the systemic arteries to the veins, identical with that which had been found to occur through the pulmonary vessels, and to which the term *circulation* is equally applicable (note *b*, p. 863).

Yet to anyone who reads Dr. Ceradini's account of Cesalpino's doctrine of the general circulation without reference to the original publications, it would seem obvious that the Italian physiologist had completely demonstrated the perpetual passage of blood from the aorta through the capillaries to the veins, from the veins to the right side of the heart, and thence again through the lungs to the left side of the heart; but a careful comparison of the Professor's free translation of Cesalpino's language with the original text, and a comparison of Cesalpino's confused and contradictory statements with each other, will lead any unbiassed critic to a very different conclusion.

The word *capillamenta* is occasionally used by Cesalpino, and this his modern interpreters invariably translate by the term 'capillaries.'[1] Dr. Ceradini, in fact, maintains that so complete were Cesalpino's proofs of the systemic circulation, that Malpighi's microscopical demonstration of the capillary vessels could add nothing to the certainty of his doctrine.[2]

Professor Scalzi also in his address[3] credits Cesalpino with a complete knowledge of the capillary circulation, and laments that Harvey, having misunderstood his Italian predecessor's teaching, wandered from the track which had been so splendidly marked out by Cesalpino, and substituted for the Italian's demonstrated capillaries his own theory of 'porosities in the tissues,' through which the blood was supposed to pass from the arteries to the veins (note *c*, p. 864).

Now, surely to credit Cesalpino with having acquired a knowledge of the capillary vessels without the aid of the microscope, is to suppose him to have been endowed with

[1] 'Cesalpino aveva detto che il sangue porta d'apprima l'alimento nutritivo alle parti, poi l'alimento aumentativo al cuore dopo essere passato dalle arterie nelle vene pei capillari sparsi in tutti gli organi.'—Ceradini, p. 177.

[2] P. 295.

[3] 'Egli dunque conobbe la circolazione capillare che doveva poi farsi più solennemente manifesta da Marcello Malpighi, altro genio d'Italia' (p. 22).

superhuman sagacity. Obviously it was as impossible to form a conception of the capillary circulation until the microscope had rendered the minute vessels visible as it was to discover Jupiter's moons or Saturn's rings before the invention of the telescope. And if we refer to the passages in which Cesalpino introduces the word capillamenta, we shall see that if, as is probable enough, Harvey was acquainted with his predecessor's writings, he could have derived from them no assistance in his attempt to trace the course of the blood from the arteries to the veins.

I now proceed to give some extracts having reference to Cesalpino's use of the word 'capillamenta.' He says: 'The vena cava and the aorta, after entering all the viscera except the heart, pass beyond them, or if any come to an end, they are resolved into capillamenta (hairlike filaments),[1] and do not pour their blood into a cavity, for nowhere, except in the heart, is the blood contained in a cavity out of a vein.' He goes on to say that 'the heart is the origin, not only of all the blood-vessels, but also of the nerves, the heart being the centre of the emotions which pass thence to the external parts, whilst sensations pass from the external parts to the heart.'[2]

Cesalpino confirms Aristotle's doctrine that the main function of the brain is to cool the blood contained within it. 'For this purpose,' he says, 'not a few and large, but many small veins from the aorta and the cava are distributed to the brain, which is supplied by blood, not gross and thick, but thin and pure.'[3] The brain then is compared to the condensing apparatus of a spirit still;[4] 'so when the thinnest part of the

[1] 'Vena cava et arteria aorta reliqua viscera, excepto corde, postquam adierint, transmeant ulterius, aut si quæ desinunt in capillamenta resolvuntur.'—*Quæst. Per.*, lib. v. p. 116 *A*.

[2] 'Cor principium omnium venarum (arteriæ enim sub nomine venarum intelliguntur Aristoteli) ex dictis patet. Sed et nervorum quoque ortum ab eodem duci, hinc manifestum fiet. . . . Sensus enim incipiunt ab exterioribus organis et desinunt in corde' (p. 116 *B*).

[3] 'Propter hoc autem circa cerebrum non paucæ ac magnæ, sed tenues ac frequentes venæ ex cava et aorta sparguntur: neque sanguis copiosus et crassus, sed tenuis sincerusque tendit eodem.'—*Quæst. Perip.*, p. 120 *A*.

[4] 'Sic quoque cum ex sanguine in corde fervente tenuissima pars sublimetur, nisi fuisset locus refrigerationis, optima pars sanguinis in auram evanuisset, nec admiranda naturæ opera perfecisset. Optima igitur ratione cerebri sub-

hot blood of the heart is sublimated, if there were no refrigerating place, the best part of the blood would vanish into the air, and not perform its admirable natural functions. Therefore, as it is the nature of heat to ascend, nature has, for the best of reasons, placed the cold and moist brain above, and those who have most heat and blood, as man for example, have most brain. But the venules scattered through the brain, if they ceased there and were not carried on into the organs of sense, would be useless, for how could these exhaust the pure and tempered blood?'

'It is[1] therefore necessary that the venules should be continued and pass out from the brain to the organs of sense; but nothing is seen to pass out of the brain but the nerves; the nerves, therefore, must be these numerous venules, collected, not into one common canal, but into a body composed of many and most minute canals. Therefore a nerve is divisible lengthways, for the venules terminate in straight fibres constituting the nerves.' This Cesalpino says is Aristotle's doctrine, and he asks: 'What can be more clear than this dictum, for a nerve is nothing more than the extremities of the aorta—some taking the nature of nerves in the head, that is, in the brain; others about the lower parts, that is, in the limbs and joints of the whole body.'[2] . . . 'It is therefore

stantia frigida et humida in superiori loco a natura condita est, quia caloris natura est sursum ascendere et quæ plurimum habent caloris ac sanguinis iis plurimum quoque cerebri datum est, ut homini. Venulæ autem in cerebrum dispersæ, si inibi desinerent nec ad organa sensuum ferrentur, inutiles fuissent, quomodo enim hæc sanguinem sincerum et temperatum haurirent?'—p. 120 *C*.

[1] 'Necesse igitur est continuas esse venulas è cerebro iterum egredientes ad sensuum instrumenta. At nullum aliud corpus è cerebro egredi videmus præter nervos: nervi igitur venulæ illæ fuerint multæ in unum coeuntes non communem canalem, sed corpus ex pluribus canalibus ac tenuissimis compositum: idcirco fissilis est nervus secundum longitudinem: nam venulæ in fibras rectas desinunt nervos constituentes'—p. 120 *D*.

[2] 'Quid potest hoc dicto clarius esse? nihil enim aliud est nervus quam extrema aortæ, alia quidem in capite, id est in cerebro naturam nervi accipientia, alia autem circa imas partes, id est circa crura et articulos totius corporis . . . Sententia igitur est Aristotelis ex aorta ad caput tendente oriri nervos cerebri . . . Meatus igitur quos scribit Aristotelis ad oculos pervenire ex venulis quæ sunt circa cerebrum, quid aliud sunt quam nervi appellati visorii? Stultum autem est credere non esse meatus, si quis amplum quendam canalem in ipsis non percipiat. Ut enim capillum perforatum esse scimus, non tamen visui ob

the opinion of Aristotle that from the branches of the aorta going to the head the nerves of the brain arise. These passages which Aristotle describes going from the veins about the brain to the eyes, what are they but the nerves of vision? But it is unwise to doubt that there are passages within them, because a large canal is not visible. For as we know that a hair is perforated, although its canal is not visible on account of its minuteness, so by other signs we may perceive the nerves to be tubular, although their canals are not visible.'

Again,[1] the blood-vessels emerge from the skull by certain foramina passing on to the organs of sense, conveying the spirit, and as it were the flower of the blood, rather than actual blood itself.' . . . 'Since the nerves the more they are lengthened the colder they are, because they are more distant from the heart, it was necessary that they should be connected with the arteries and the veins, in order that they might be warmed by their heat; for a communication being made, there is effected a tempering of the tissues suitable for sensation.'[2] Lastly,[3] 'if the spirits are conveyed through the nerves for the purpose of sensation, it does not follow that the sentient part is of a sanguineous nature, for the nerves do not convey blood.'

It is evident from these passages, to which others, consistently expressing the same doctrine, might have been added, that Cesalpino's 'capillamenta,' which his modern Italian commentators, Professors Ceradini and Scalzi, convert into capillary blood-vessels, were the supposed filamentous terminations of arteries and veins in nerves; and that through the tubular nerves the spirituous part of the blood was supposed to pass, and thus to confer a sentient power upon the nerves. But in

parvitatem meatus apparet, sic nervos ex aliis signis fistulosos esse cognoscimus, visui tamen non sunt manifesti meatus'—120 *E. F.*

[1] 'Hæc (vascula) iterum ex calvaria multa simul per foramina quædam egressa, ad instrumenta sensuum feruntur, spiritum ac veluti florem sanguinis potius quam sanguinem ferentia'—120 *F.*

[2] 'Præter eum nervi quanto magis protrahuntur, eo frigidiores sunt, quia distantiores a corde fiunt, propter hoc quoque necesse fuit jungi cum arteriis ac venis ut earum calore foverentur; sic enim communicatione facta temperies in carne fit sensui commoda'—p. 131 *A.*

[3] 'Si spiritus per nervos deferuntur ad sensus perficiendos, non erit necesse sanguinem esse quod sensit; nervi enim sanguinem non ferunt.'—*Quæst. Perip.* 130 *F.*

no single passage of Cesalpino's works, is there to be found any mention of the capillamenta as channels by which the blood passes from the arteries to the veins; and it is particularly stated in a passage which I have just now quoted, that the nerves which are supposed to intervene between the capillamentous termination of the arteries and the veins do not convey blood.

I have endeavoured to represent Cesalpino's description by a diagram. The arrows indicate the to-and-fro movement of the blood, alike in the artery, in the vein, and in the anastomoses between the artery and the vein.[1]

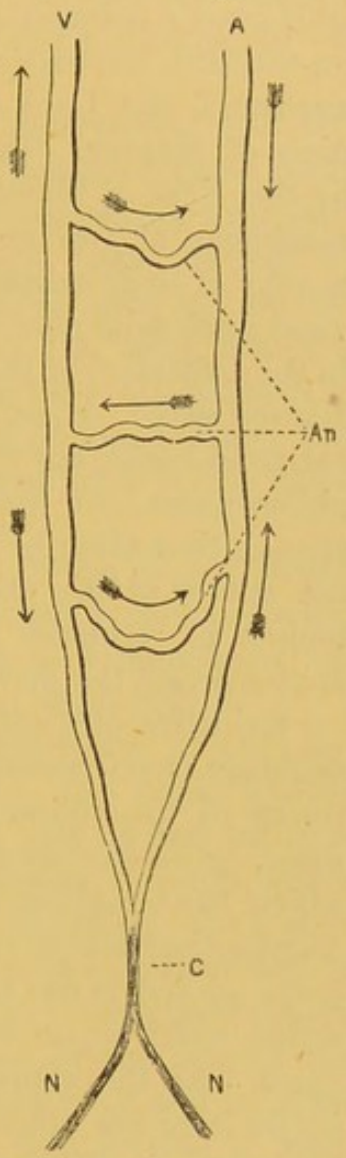


FIG. 46.—DIAGRAMMATIC REPRESENTATION OF CESALPINO'S DOCTRINE OF THE CIRCULATION.
A, Artery; V, Vein; C, Capillamenta; N N, Nerves; An, Anastomoses between the artery and the vein.

Dr. W. Ogle, in his able and interesting translation of Aristotle's '*De Partibus Animalium*,'[2] points out that by the word *νεῦρα*, which had misled some commentators, Aristotle meant not nerves merely but *sinews*[3] and he believed that 'the small arteries ceased to be tubular and were solidified into tendinous fibres, which being continuous on the one hand with the heart by the aorta, and on the other with the tendons and bones, were the instruments of motion.'[4]

It is obvious that Cesalpino's description of the arteries terminating in capillamentous nerves is a mere reproduction of Aristotle's ancient doctrine without modification or addition of any kind.

In contrast with these capillamentous fictions of Cesalpino I would direct attention for a moment

[1] Note *d*, p. 865.

[2] *Aristotle on the Parts of Animals*, translated, with Introduction and Notes by W. Ogle, M.A., M.D. London, 1882.

[3] P. 196, note 20.

[4] P. 203, note 7.

to Harvey's minute dissections of the blood-vessels and the nerves of the human body, which are preserved in the glass cases on the wall of our library. Those interesting memorials of Harvey's industry had long been carefully kept at Burley-on-the-Hill, the seat of the Earls of Winchelsea, one of whose ancestors, the Lord Chancellor Nottingham, had married a niece of Harvey. They were presented to the College in 1823 by the Earl of Winchelsea, who expressed a hope that those specimens of Harvey's scientific research would be deemed worthy of their acceptance.[1] These precious memorials of the great anatomist afford an interesting llustration of a passage in the dedication of the treatise '*De Motu Cordis et Sanguinis*.'[2] Harvey says: 'I profess both to learn and to teach anatomy, not from books, but from dissections; not from the positions of philosophers, but from the fabric of nature.' It is probable enough that when he penned this sentence he had in his mind the author of the *Quæstiones Peripateticæ*, and his vain attempts to reconcile Aristotle's philosophy with the facts of anatomy and the marvellous phenomena of living beings.

Evidence of Cesalpino's entire ignorance of a system of capillary blood-vessels is afforded by the fact that when he refers to the communication between the arteries and the veins, he always speaks of this as occurring by inosculations, which the Greeks call anastomosis; thus he says,[3] 'there is a constant movement from the heart through the arteries to all parts of the body, because there is a constant generation of spirit, which by its expansion is ready to be diffused into all parts. At the same time it carries nutritive aliment, and elicits from the veins auctive aliment, by means of the inosculations which the Greeks call anastomosis; but the spirits at

[1] See *The Roll of the Royal College of Physicians*, by Wm. Munk, M.D. vol. i. p. 144.

[2] Coll. ed., p. 7; Dr. Willis, p. 7.

[3] 'Motus igitur continuus a corde in omnes corporis partes agitur quia continua est spiritus generatio qui sua amplificatione diffundi celerrime in omnes partes aptus est, simul autem alimentum nutritivum fert et auctivum ex venis elicit per osculorum communionem quam Græci anastomosim vocant: tandem vero spiritu in aerem ambientem difflante alimenti corpulentia remanet, partim frigore partim calore coagulata. Ad difflationem autem spiritus et sanguinis distributionem in partes, vasa residere oportet, donec novus spiritus iterum repleat ac distendat.'—*Quæst. Perip.* 123 *B*.

length escaping into the ambient air, the bodily part of the aliment remains, being coagulated partly by cold and partly by heat. After the diffusion of the spirit and the distribution of the blood, the vessels remain at rest until a fresh supply of the spirit again fills and distends them.' [1]

This passage, while it is quite in accordance with the Galenic doctrine of the veins and arteries containing two kinds of blood, auctive and nutritive, and communicating with each other by anastomosis, is quite inconsistent with the idea of a capillary system of vessels. Some blood, with its auctive nutriment, is supposed to be drawn from the veins into the arteries, and that which is carried beyond the anastomosis is said to be partly evaporated and partly rendered solid; but no reference is made to the passage of any part of the blood from the terminal arteries into the veins.

It will be seen, too, that the diffusion of the blood through the arteries was supposed to be due, not to the propulsive power of the heart, which Harvey was the first to demonstrate, but to the constant generation and expansion of the spirits.

Again, a reference to Cesalpino's description of the supposed cooling influence of the air upon the blood affords collateral evidence, if such were needed, that he had no conception of thin-walled capillary blood-vessels, through which the air and the blood exert a mutual influence upon each other. Thus, in describing the relation of the blood-vessels to the air-tubes in the lungs, he remarks that,[2] 'with good reason the branches of the wind-pipe are placed by the side of the pulmonary vein, not by the pulmonary artery;' and he gives the following reason for this: 'For the vein, having a more simple texture and consisting of only one thin tunic, can be more readily refrigerated (*i.e.* by contact with the air), and besides, the dilatation and contraction of the air-tubes would have interfered with the pulsation of the artery if they had been placed near together.'

[1] Note *e*, p. 866.

[2] 'Et merito huic vasi (i.e. venæ), protenduntur asperæ arteriæ canales non alteri educenti (i.e. arteriæ): nam hoc simplicius existens ex unica eaque tenui tunica constitutum, refrigerari facilius poterat, præterea pulsationi alterius vasis impedimento fuisset asperæ arteriæ dilatatio et constrictio si juxta essent posita.'—*Quæst. Per.* 125 *F.*

In this passage we have conclusive evidence that the author had no knowledge of minute capillaries through which the blood is exposed to the influence of the air, but on the contrary he supposed this to occur in thin-walled veins, of such a size that, if they were arteries, their pulsation would be impeded by the supposed dilatation and contraction of the contiguous air-tubes.

In another passage, the theory that the veins are cooled by being placed near the air-tubes again finds expression. After having shown, as he states, that the brain is a blood refrigerator, he goes on to say: [1] 'For this cause inspiration of air occurs through the nostrils, which terminate near the brain, not only on account of the sense of smell, but also in order that the veins ascending from the neck to the brain should be cooled; for the veins passing towards the brain are placed near the wind-pipe.' Here we have expressed not only the theory of blood cooling by the contact of veins with the trachea, but the veins are spoken of as 'ascending through the neck to the brain,' a statement which would never have been made by one who knew that the course of the blood in the veins of the neck is perpetually downwards.

Dr. Ceradini repeatedly asserts that Cesalpino proved and demonstrated the systemic circulation by his observation of the effect of *obstructing the flow of blood through the veins*. And I propose now to examine Cesalpino's statements in connection with Ceradini's comments and conclusions. In the treatise *Quæstiones Medicæ*, p. 234, Cesalpino says: [2] 'It is worthy of inquiry why, when a ligature is applied, the veins swell beyond and not on this side of the obstruction, which those who practise venesection know by experience; but the

[1] 'Ob hanc vero causam fit aeris inspiratio per nares juxta cerebrum desinentes, non solum olfactus gratia, sed etiam ut venæ per collum ascendentes ad cerebrum usque, refrigerentur, protenduntur enim juxta asperam arteriam venæ cerebrum petentes.'—*Quæst. Per.* 126 *A*.

[2] 'Sed illud speculatione dignum videtur propter quid ex vinculo intumescunt venæ ultra locum apprehensum, non citra, quod experimento sciant qui venam secant: vinculum enim adhibent citra locum sectionis, non ultra; quia venæ tument ultra vinculum, non citra. Debuisset autem opposito modo contingere, si motus sanguinis et spiritus a visceribus fit in totum corpus: intercepto enim meatu non ultra datur progressus: tumor igitur venarum citra vinculum debuisset fieri.'—*Quæst. Med.* 234 *A*.

opposite ought to occur if the motion of the blood and spirit is from the viscera over the entire body; for the passage outward being blocked, a swelling of the veins should occur on this side of the ligature.' Then, after quoting Aristotle's explanation, which is quite unintelligible, he gives his own: 'In explanation of this it is to be observed that the orifices of the heart are so arranged by nature that there is an entrance from the vena cava into the right ventricle, whence there is an open passage into the lungs, and from the lungs there is an entrance into the left ventricle, and thence into the aorta, valves being placed at the orifice of the vessels to prevent reflux. There is thus a perpetual movement from the vena cava through the heart and lungs to the aorta, as we have explained in our peripatetic questions.' Up to this point Cesalpino's description is clear and correct; but it goes not beyond the knowledge of his contemporaries. After this, confusion begins to be apparent.[1] 'As in waking hours the motion of the native heat is outwards, viz., to the organs of sense, but during sleep it is inwards towards the heart, it is to be supposed that during wakefulness much spirit and blood are carried to the arteries, whence there is a passage to the nerves; but during sleep the same heat returns to the heart by the veins, not by the arteries; for the only entrance to the heart is by the vena cava, not by the aorta. The proof of this is to be found in the pulse, which in those who are awake is large, strong, quick, and with a certain vibration,[2] but during sleep it is small, feeble, and slow; for during sleep less native heat goes to the arteries, while it passes into them more forcibly when we are awake. But in the veins the reverse occurs, for they are fuller during sleep and smaller during wakefulness, as may be seen in those of the hand; for during sleep the native heat passes from the arteries to the

[1] 'Cum autem in vigilia motus caloris nativi fiat extra, scilicet ad sensoria: in somno autem intra, scilicet ad cor; putandum est in vigilia multum spiritus et sanguinis ferri ad arterias, inde enim in nervos est iter. In somno autem eumdem calorem per venas reverti ad cor, non per arterias; ingressus enim naturalis per venam cavam datur in cor, non per arteriam. Indicio sunt pulsus, &c.'—*Quæst. Med.* 234 *B.*

[2] This description of the pulse, as Ceradini admits (p. 272, *note*), is literally copied from Galen.

veins by inosculations, which are called anastomosis, and then to the heart; but as the outward flow of the blood to the superior parts, and its return to the inferior parts, like Euripus, is manifest during sleep and wakefulness, so is this kind of motion in whatever part of the body a ligature is applied, or when in any other way the veins are obstructed, not obscure. For as when the passage is closed rivulets swell up where they are accustomed to flow, so perhaps does the blood at the time return to its source, lest being cut off it should be extinguished.[1]

Cesalpino's statements and arguments may be summed up thus: During wakefulness the blood passes from the aorta to the nerves, as before explained in connection with the word *capillamenta*, while during sleep it passes from the arteries by the before-mentioned anastomoses (not by capillaries) through the veins to the heart. According to this view the swelling of the veins on the distal side of the ligature should occur only during sleep, and the only suggested explanation of its occurrence at other times is, that perhaps when a vein is obstructed 'the blood returns to its source, lest being cut off it should be extinguished.'[2]

Now, surely if Cesalpino had known that there is a continual flow of blood from the branches of the aorta to the veins, and so back to the heart, he would in a sentence have given the simple and obvious explanation of the fact constantly observed, as he says, by those who practise venesection; but for want of this knowledge he suggests an explanation of the phenomenon which is partly metaphysical and wholly unintelligible.

Dr. Ceradini admits that Cesalpino's expression ('exundatio ad superiora, retrocessus ad inferiora') 'outward flow to the

[1] 'Cum enim tollitur permeatio intumescunt rivuli qua parte fluere solent, forte recurrit eo tempore sanguis ad principium, ne intercisus extinguatur'—234 *C*. (See note *f*, p. 866).

[2] If Cesalpino had been asked what became of the blood which during the waking state was continually being sent through the arteries, since during sleep only did he suppose it to return by the veins, he would probably have replied, in the terms of a previous extract (pp. 852–3), that while the spirituous part escaped into the air, the grosser part was coagulated by cold and heat.

superior parts, and return to the inferior parts, like Euripus,' is a comparison borrowed from Aristotle, and therefore it may be assumed not to contain any new doctrine. But he quotes a sentence from Harvey with an attempt to show that it is identical with Cesalpino's statement. The passage occurs in a letter to Dr. Hofmann,[1] and is as follows: 'I do indeed assert that the blood is incessantly moving out from the heart by the arteries to the general system and returning from this by the veins back to the heart, and with such a flux and reflux in such mass and quantity that it must necessarily move in some way in a circuit.'

Now it is strange that Dr. Ceradini does not see the essential difference between these two passages.[2] Obviously the comparison with Euripus implies a flux and reflux through the same and not through different channels; while Harvey, in a few words, gives a clear description of the systemic circulation as a *constant* outflow (not during the waking state only) by the *arteries*, and a reflux by the *veins*, and in such quantity that it must necessarily move in a circuit. If Cesalpino had written this sentence, he might indeed have been credited with a knowledge of the systemic circulation; but neither that nor anything like it is to be found in any part of his writings.

The passages in Cesalpino's works which show most conclusively that he could have had no idea of a continuous flow of blood from the systemic arteries to the veins, on its way back to the heart, are those which describe, or evidently imply, a passage of the blood *from the trunk to the branches* of the veins. Thus in the *Ars Medica*[3] the following statement occurs: 'But the vena cava distributes branches throughout the whole body, in order that, together with the arteries, they may nourish every part. From the same vena cava some large branches, called emulgent veins, go to the kidney, by

[1] Coll. ed., p. 635; Dr. Willis, p. 595.

[2] In the Latin edition of Harvey, which Dr. Ceradini quotes, the words are: 'Eo fluxu et refluxu, *ea quantitate et copia* ut in circuitu quodam modo moveri sit necesse.' In quoting this passage the four significant words here printed in italics are omitted. Was this in order to render Harvey's statement more like Cesalpino's than it would otherwise appear to be?

[3] P. 488, ed. 1670.

which (veins) the superfluous water of the blood is excreted, that it may be carried by the ureters to the bladder.' [1]

This passage requires no comment and admits but one interpretation. Neither does the following: 'As rivulets draw water from a fountain, so do the veins and arteries draw blood from the heart.' [2] There is a parallel passage in the *Ars Medica*: [3] 'The fountain of blood in the heart being distributed into four vessels—viz., the vena cava, the aorta, the pulmonary vein and artery—irrigates the whole body like the four rivers proceeding from Paradise.' [4] Curiously enough, Dr. Ceradini quotes this passage in illustration of Cesalpino's clear view of the circulation.[5]

There is another remarkable passage in which it is clearly assumed that the blood, under certain conditions, passes by the veins of the neck upwards to the brain. In opposition to Galen, who taught that the nerves and nervous influence proceed from the brain and spinal cord, and in support of Aristotle's doctrine that the *heart* is the centre and source of all nervous influence, Cesalpino says no one denies the fact mentioned by Galen, that division or ligature of the spinal cord, or of a nerve, paralyses the parts below. But he goes on to argue that the same result follows the obstruction of all the vessels of the neck, because then the influence of the heart can no longer pass to the nerves. 'But it is not sufficient that the *arteries* of the neck alone be closed, which Galen sometimes found might be done without harm, for then a power is transmitted from the heart by the *veins* to the same parts, since there are inosculations between the veins and

[1] 'Vena autem cava ramos in totum corpus dispergit ut simul cum arteriis universas partes nutriant. Ab eadem cava rami quidem insignes ad renes tendunt, venæ emulgentes vocatæ, per quas superflua sanguinis aquositas excernitur, ut per vasa urinaria in vesicam feratur.' The name 'venæ emulgentes' was doubtless suggested by the theory that those veins drain out the excess of water from the blood.

[2] 'Ut igitur rivuli ex fonte aquam hauriunt, sic venæ et arteriæ ex corde.'—*Quæst. Per.* 116 *A*.

[3] P. 1.

[4] 'Fons sanguinis in corde distributus in quatuor venas, scilicet cavam, aortam, arteriam venalem et venam arterialem, totum corpus irrigat instar quatuor fluminum ex Paradiso prodeuntium.'

[5] P. 294.

the arteries not only in the heart, but along their whole course.' [1]

In the preceding passages we have conclusive evidence of Cesalpino's doctrine that in certain veins, and at certain times, the blood flows from the trunks to the branches, and Dr. Ceradini [2] himself admits Cesalpino's belief that during the waking condition of animals there is a reflux from the *aorta* into the left ventricle of the heart, in consequence of the difficulty with which the blood escapes from the terminal branches of the aorta to the nerves. Yet so determined is Dr. Ceradini to maintain his fellow-countryman in the position of the discoverer of the circulation that this theory of a reverse current from the aorta to the heart, which, if it occurred as a fact, would be instantaneously fatal, and which, to an impartial critic, affords conclusive proof of Cesalpino's ignorance of that which he is declared to have discovered, is held by his advocate not to be of the slightest consequence.[3]

The most curious illustration of the manner in which Cesalpino's compatriots endeavour to obtain for him the credit of originality and completeness with regard to the circulation is afforded by a lecture by Dr. Del-Vita,[4] which is made up of extracts from all Cesalpino's writings, the orginal Latin and the Italian translation being placed in parallel columns; and the quotations from various treatises are so pieced together as to read like a clear and continuous description of the circulation as we now understand it. The source of each word and sentence is given, and although the lecture occupies less than three pages of large type, so numerous are the references that all the letters of the alphabet are insufficient to indicate their

[1] 'Non sufficit autem arterias solum, ut Galenus aliquando expertus est sine noxa in collo constringere, nam ex venis in eadem loca transfertur virtus a corde, cum osculorum sit communio non solum in corde sed etiam per totum venarum atque arteriarum ductum.'—*Quæst. Per.* 121 *D.* In *Quæst. Med.* (229 *F*) it is also stated that when the carotid arteries are obstructed, the jugular veins, by means of anastomosis, carry on the blood and spirit to the brain.

[2] 'È vero del resto che negli animali vigili Cesalpino ammise un rigurgito dall' aorta verso il cuore' (p. 273).

[3] 'Ciò non pregiudica minimamente l'essenzialità della dottrina stessa della circolazione'—p. 269. (See note *g*, p. 866.)

[4] *Della Circolazione del Sangue Scoperta da Andrea Cesalpino:* Lezione Anatomico-Fisiologica da Antonio Del Vita di Arezzo, 1876.

source. The following brief paragraph is an illustration of the method adopted by this author:—'The blood, therefore, is carried from the right ventricle of the heart by the pulmonary artery, and again returned to the heart by the pulmonary vein, which proceeds from the left ventricle; from the heart it passes into the arteries, from the arteries to the veins, from the veins to the heart; so there is a perpetual movement from the vena cava through the heart and lungs to the aorta, and by the arteries into the whole body.' This description of the circulation, which occupies ten short lines, is not taken from any one part of Cesalpino's writings, but is extracted, as the letters of reference show, from six different parts of three separate treatises, namely: *Quæstiones Peripateticæ*, *Quæstiones Medicæ*, and the work *De Plantis*. If Cesalpino had anywhere described the course of the blood, as it was first revealed by Harvey, there would have been no need to resort to such a patchwork proceeding as this, which does no credit to its author, Dr. Del-Vita, or to the orator, Dr. Maggiorani, who refers to the lecture with approval. It is manifest that by stringing together isolated words and sentences the doctrine of the circulation might be extracted from the Pentateuch, or any other ancient writings.

At the risk, and, I almost fear, at the cost of being tedious, I have made these numerous quotations from Cesalpino's various writings, in order to prove by his *ipsissima verba* what was the amount of his knowledge with regard to the systemic circulation.

I think that I have shown conclusively that great and various as were his acquirements in different departments of natural science, more especially in botany, in which science he had the merit of great originality, as regards the physiology of the circulation his information was not in advance of that possessed by his immediate predecessors and contemporaries. It will have been seen that Professor Ceradini relies mainly upon three distinct pieces of evidence to establish his fellow-countryman's claim to be regarded as the discoverer of the circulation: (1) His use of the term 'circulation;' (2) his employment of the word 'capillamenta;' and (3) his explanation of the effect of obstructing the current of blood in

the veins. But a critical examination of each of these points has afforded evidence of Cesalpino's ignorance rather than of his knowledge of the circulation. (1) The term circulation, being applied only to the passage of the blood through the lungs, implies a want of knowledge of the systemic circulation; (2) the word 'capillamenta,' which is erroneously translated 'capillaries,' is used only to designate an imaginary transition from arteries and veins into nerves, the nerves and the capillamenta being declared to be impervious to blood; (3) and lastly, no intelligible explanation is given of the fact, which those who practise venesection had observed for centuries, that an obstructed vein swells on the distal side of the ligature or other obstructing cause.

To turn from Cesalpino's doubtful and contradictory utterances, and his peripatetic fancies with regard to the circulation, to Harvey[1]'s clear statements and exact reasoning, is like coming from a dark and stifling cave into fresh air and bright sunshine.

I propose, in conclusion, to give one extract from Harvey's treatise *On the Motion of the Heart and Blood*, which affords a good example of his style, and which is especially interesting from its containing in a few sentences an illustration of the process of observation and reasoning which led up to his great discovery of the systemic circulation: [1]—

'When I surveyed my mass of evidence, whether derived from vivisections and my various reflections on them, or from the ventricles of the heart and the vessels that enter into and issue from them, the symmetry and size of these conduits—for nature, doing nothing in vain, would never have given them so large a relative size without a purpose—or from the arrangement and intimaté structure of the valves in particular, and of the other parts of the heart in general, with many things besides, I frequently and seriously bethought me, and long revolved in my mind, what might be the quantity of

[1] M. Flourens, in his *Histoire de la Découverte de la Circulation du Sang*, 1854, p. 30, says of Harvey's treatise: 'Le livre d'Harvey est un chef-d'œuvre. Ce petit livre de cent pages est le plus beau livre de physiologie.' For this favourable judgment of Harvey, however, the author is severely taken to task by Dr. Ceradini, pp. 206–7.

blood which was transmitted, in how short a time its passage might be effected, and the like; and not finding it possible that this could be supplied by the juices of the ingested aliment, without the veins on the one hand becoming drained, and the arteries on the other getting ruptured through the excessive charge of blood, unless the blood should somehow find its way from the arteries into the veins and so return to the right side of the heart; I began to think whether there might not be A MOTION AS IT WERE IN A CIRCLE. Now this I afterwards found to be true; and I finally saw that the blood, forced by the action of the left ventricle into the arteries, was distributed to the body at large and its several parts, in the same manner as it is sent through the lungs, impelled by the right ventricle into the pulmonary artery; and that it then passed through the veins and along the vena cava, and so round to the left ventricle in the manner already indicated; which motion we may be allowed to call circular.' [1]

It was the recognition and proof, by numerous observations and experiments, of the incessant propulsion of the blood by the contractions of the left ventricle of the heart through the systemic arteries into the veins, and so back to the right side of the heart, that constituted Harvey's grand discovery of the greater or systemic circulation.

Harvey, as we have seen, obtained his anatomical knowledge at Padua under the famous Fabricius, of whom he speaks with gratitude and reverence,[2] as 'the celebrated Hieronymus Fabricius of Acquapendente, a most skilful anatomist and venerable old man.' While, therefore, we cannot concede to Cesalpino the honour of having discovered the circulation of the blood, a distinction which he himself would probably never have thought of claiming, we willingly express our gratitude to Italy for having given our celebrated countryman the anatomical training, without which he could not have made his great discovery—a discovery which, throughout all ages and by all civilised nations, will be looked upon as the foundation of modern physiology, and therefore of scientific medicine.

[1] Coll. edition, p. 48; Dr. Willis's translation, pp. 45-6.

[2] Coll. edition, p. 65; Dr. Willis's translation, p. 62.

Notes to the Harveian Oration.

NOTE *a*, page 846.

Harvey demonstrated the passage of blood through the lungs and the impermeability of the septum cordis by an experiment which he thus describes in one of his letters to Schlegel:

'Having tied the pulmonary artery, the pulmonary veins, and the aorta in the body of a man who had been hanged, and then opened the left ventricle of the heart, we passed a tube through the vena cava into the right ventricle of the heart, and having at the same time attached an ox's bladder to the tube, we filled it nearly full of warm water, and forcibly injected the fluid into the heart, so that a greater part of a pound of fluid was injected into the right auricle and ventricle. The result was that the right ventricle and auricle were enormously distended, but not a drop of water or of blood made its escape through the orifice in the left ventricle. The ligatures having been undone, the same tube was passed into the pulmonary artery, and a tight ligature having been put round it to prevent any reflux into the right ventricle, the water in the bladder was now pushed towards the lungs, upon which a torrent of the fluid, mixed with a quantity of blood, immediately gushed forth from the perforation in the left ventricle, so that a quantity of water equal to that which was passed from the bladder into the lungs at each effort, instantly escaped by the perforation mentioned.'[1] Dr. Ceradini (p. 194) is so good as to express his admiration of this experiment having been performed by Harvey at the advanced age of seventy-three years.

NOTE *b*, page 847.

Dr. Ceradini endeavours to prove that Cesalpino's use of the word *circulation* implied his recognition of the systemic as well as of the pulmonary circulation, by first quoting Aristotle's definition of the word:—'Conversio est motus qui fit ex sese in idem' (Conversion is motion from itself into itself). 'Motus autem per rectum qui ab sese in aliud' (but straight motion is that from itself into another).[2]

And then he quotes a metaphysical discussion of Cesalpino's, having no reference to the movement of the blood, but in which the

[1] Dr. Willis's translation, p. 597; Coll. edition, 613–4. The references to Dr. Willis's translation are to the Sydenham Society's edition of the works of Harvey, translated by Robert Willis, M.D., 1847.

[2] Ceradini, p. 253. Cesalpino, *Quæst. Per.* p. 33 *A*.

celestial circulation is compared with the divine intelligence:— 'Circulatio autem tanquam fine carens infinito tempore agitur' (the celestial circulation, as if without end, goes on perpetually). And again, 'quatenus autem continua motione ab eodem in idem transit (idem enim est circuli principium, medium et finis) maxime assimilatur intellectioni quæ est sui ipsius.' Since by a perpetual motion there is a passage from the same to the same (for the beginning, the middle, and the end of a circle is the same), there is the greatest resemblance to the (divine) intelligence, which is self-originating.

Dr. Ceradini maintains that Cesalpino having thus accepted Aristotle's definition of a circular movement, could never have intended his use of the word to be limited to the passage of blood from the right to the left side of the heart through the lungs, but he must have meant to express the idea of a general circulation throughout the system.

It will be seen, however, that Cesalpino's views of the systemic circulation are utterly inconsistent with his modern interpreter's argument.

NOTE *c*, page 847.

It is manifest that without the aid of the microscope the mode in which the blood passes from the arteries to the veins could only be a subject for speculation. Harvey, in his original treatise, speaks of 'pores in the flesh' (*porositates carnis*), through which he supposed the blood to pass;[1] but in his letter to Schlegel,[2] after expressing his opinion that the passage of the blood from the extremities of the arteries into those of the veins could not be effected without some 'admirable artifice,' suggests that the minute arteries may gradually pass into the coats of the accompanying veins, and 'that the same thing takes place here as we observe in the conjunction between the ureters and the bladder, and of the biliary duct with the duodenum.' Malpighi was the first who, with the aid of lenses, had the delight of seeing the blood actually circulating through the capillaries in the lung and mesentery of the living frog.[3] Malpighi speaks of the appearances as beyond the power of his pen to describe ('qui calami subterfugiunt descriptionem'), and we can imagine with what delight Harvey would have witnessed the circulation in the magnified web of a frog's foot.

[1] See Coll. edition, p. 69; Dr. Willis, p. 68.

[2] Coll. edition, p. 627; Dr. Willis's translation, p. 600.

[3] *Marcelli Malpighi Opera*, London 1687, pp. 141–2; and *Opera Postuma*. Amsterdam, 1700, p. 122.

NOTE *d*, page 851.

Dr. Ceradini (pp. 67 and 286) refers to an observation of Hippocrates,[1] that when an artery is wounded the blood which first flows is of a brighter red colour than the dark blood which subsequently escapes; also to Galen's statement that a wound of an artery empties not only the arteries but the veins, the result, as Galen supposed, of the anastomosis between the arteries and the veins throughout the body.

Reference is also made to Cesalpino's statement,[2] that 'the veins are so connected with the arteries by little mouths, that when a vein is wounded dark venous blood first escapes, then succeeds lighter coloured arterial blood.' ('Venas cum arteriis adeo copulari osculis, ut vena secta primum exeat sanguis venalis nigrior, deinde succedat arterialis flavior.') Dr. Ceradini remarks that this observation of Cesalpino, together with that of the veins swelling beyond a ligature, affords proof of the circulation as complete as it is possible to furnish.

What the quotation really appears to prove is, that Cesalpino, in common with Galen, believed that the veins were everywhere connected with the arteries by inosculations; and as the observations of Hippocrates and Galen seemed to prove the passage of blood from veins to arteries, his own doubtful observation on the effect of venesection appeared to him to prove the passage from arteries to veins, not, be it observed, by capillaries, but by inosculations.

Harvey was the first to prove the absence of the supposed anastomoses between the veins and arteries by an experiment which he thus describes in his 'Anatomical Disquisition addressed to John Riolan:[3]—

'Having laid open the thorax of an animal, and tied the vena cava near the heart so that nothing shall pass from that vessel into its cavities, and immediately afterwards having divided the carotid arteries on both sides, the jugular veins being left untouched, if the arteries be now perceived to become empty, but not the veins, I think it will be manifest that the blood does nowhere pass from the veins into the arteries, except through the ventricles of the heart. Were it not so, as observed by Galen, we should see the veins as well as the arteries emptied in a very short time by the efflux from the corresponding arteries.'

[1] *De Natura Hominis*, sect. iii. p. 9. [2] *Quæst. Medicarum*, p. 212 c.
[3] Coll. edition, p. 106; Dr. Willis's translation, p. 104.

Note *e*, page 853.

Prof. Ceradini maintains that Cesalpino meant by *auctive* blood in this passage, blood which had passed through the organs and is returning towards the heart to augment the blood which is there being constantly fabricated ; and he accuses Harvey's biographer, in the College edition of his works,[1] of having with bad faith inverted the sense of the passage.[2] But it clearly admits of no other interpretation. For as, in accordance with Aristotle's doctrine, Cesalpino believed that the veins contained the auctive blood, while the nutrient blood is in the arteries,[3] it is evident that the arteries could 'elicit' auctive blood from the veins through the anastomoses, only by a direct current from the veins into the arteries. Cesalpino, in his entire ignorance of the hydraulics of the circulation, could not know that if there were such anastomoses as he supposed between the veins and the arteries, the current of blood must of necessity, in consequence of the greater pressure in the arteries, set from them to the veins—as in a case of aneurism by anastomosis.

Note *f*, page 856.

Dr. Ceradini refers to Cesalpino's statement that *all* the veins swell when obstructed, as conclusive proof that he had actually seen the effect of obstructing all the veins either while witnessing operations on the human body or as a result of the vivisection of animals, which he assumes that he must have practised by the necessary aid of artificial respiration, which Vesalius may have taught him to practise.[4] Thus, while almost ignoring Harvey's numerous experiments, and asserting that the presence of valves in the veins was the only evidence adduced by him in proof of the systemic circulation, he gives Cesalpino the credit of having performed experiments of which no mention whatever is made in that author's writings. In fact, Cesalpino mentions no experiments or observations of his own, but refers only to a fact known, as he says, by those who practise venesection.

Note *g*, page 859.

It is not without interest to contrast Dr. Ceradini's severe and sometimes unjust and erroneous criticism of Harvey with his very lenient judgment of Cesalpino's errors.

Thus we have seen that Cesalpino's admission of a reflux of blood from the aorta to the heart during the state of wakefulness is

[1] P. xx. [2] Pp. 244–5. [3] *Quæst. Per.* 117 *E*. [4] P. 265.

looked upon as a pardonable error, but, on the other hand, the slight inaccuracy (an inaccuracy which can be shown to be such only by the aid of modern instruments) implied in Harvey's statement that the arterial pulse is felt simultaneously over the whole body is severely criticised.[1]

Again, while he censures Harvey for neglecting to mention Cesalpino's name and writings, the latter's omission to quote his fellow-countryman Colombo in reference to the pulmonary circulation and the impermeability of the septum is excused, amongst other reasons for this, that 'Colombo's zeal and the energy with which he maintained the impermeability of the septum cordis, without furnishing any new anatomical or physiological proof, appeared to Cesalpino to be rather comical than otherwise.'[2] It does not appear to have occurred to the learned Professor that Harvey may have thought that Cesalpino's speculations on the outward flow of the blood during the waking state, and its reflux during sleep, had more of the ludicrous than the luminous about them; and that, if Harvey had wished to exalt himself by comparison with others, he could not have done this better than by printing in parallel columns his own brief and clear description of the circulation, and the fanciful theories of Cesalpino.

Both Professors Ceradini[3] and Scalzi[4] contrast what they designate Cesalpino's gentleness and modesty with Harvey's vehemence and pomp of words ('violenza e pompa di parole'). And this Dr. Ceradini considers a sufficient explanation of the little notice taken of Cesalpino's physiological writings, while Harvey found himself at once assailed by a host of enemies. The simple explanation of this contrast is that one was little more in physiology than an expounder of ancient speculations, which being for the most part unintelligible, excited no controversy, while the other set forth novel doctrines with such unmistakable clearness as to excite at once the attention of the world and the opposition of all to whom previously unrecognised truths are unwelcome, and even hateful.

Dr. Ceradini asserts that Harvey's doctrine of the blood being propelled into the ventricle by the contraction of the auricle is erroneous; and this notwithstanding the decisive evidence[5] which

[1] P. 185. [2] Pp. 256–7. [3] P. 298. [4] P. 19.

[5] 'After the heart (i.e. the ventricles) had ceased to beat, the auricles, however, still contracting, a finger placed upon the ventricles perceives the several pulsations of the auricles, precisely in the same way and for the same reason, as we have said, that the pulses of the ventricles are felt in the arteries, to wit, the distension produced by the jet of blood. And if at this time, the auricles alone pulsating, the point of the heart be cut off with a pair of scissors, you will perceive

Harvey adduces in support of his statement.[1] Dr. Ceradini expresses his determination to enter upon an experimental inquiry with regard to this question. In the course of this inquiry he will do well to take into consideration the evidence in support of Harvey's doctrine of the propulsive influence of the auricles, which is afforded by the clinical phenomena of the presystolic (auriculo-systolic) thrill and murmur which result from constriction of the mitral orifice.[2]

Dr. Ceradini not only exaggerates greatly the importance which Harvey attaches to the valves in the veins as evidence of the course of the blood, but, in order, as it would seem, to depreciate as much as possible what he most inaccurately asserts to be the only new evidence which Harvey could adduce in proof of the circulation, he maintains that the valves in the veins are not all necessary for the circulation of the blood.[3] Perhaps it has not happened to the learned professor of physiology to witness the many distressing and even fatal consequences of the impeded circulation that result from such a dilated and varicose condition of the veins as renders their valves incompetent to prevent the reflux and backward gravitation of the blood.

The best piece of criticism in Dr. Ceradini's book is that to be found at the commencement of the first chapter, in which, after referring to a tablet in the Veterinary Institute of the University of Bologna, attributing to Carlo Ruini the discovery of the circulation of the blood, he mercilessly exposes the plagiarisms of Ruini, and demonstrates the utter absurdity of the attempt to make of him a great discoverer. At the end of the chapter[4] he expresses a hope, in which he believes that every friend of truth and justice will join, that the lying inscription (l' epigrafe menzognera) which speaks of Ruini as the discoverer of the circulation, may be removed from the walls of the University of Bologna. It is somewhat embarrassing for zealous patriots that Italy should simultaneously have monuments in honour of two supposed discoverers of the circulation—that of Cesalpino at Rome, and that of Ruini at Bologna.

the blood flowing out upon each contraction of the auricles.'—Dr. Willis's translation, p. 27. Coll. edition, p. 29.

[1] Ceradini, p. 87.

[2] See *ante*, p. 500.

[3] 'Le valvole delle vene non sono punto necessarie alla circolazione del sangue,' p. 202.

[4] P. 74.

EPILOGUE.

I sent a copy of my Harveian Oration, with the appended notes, to each of the three learned Professors Ceradini, Scalzi, and Maggiorani, and, although I received no direct reply from any one of them, I learned from the *Bulletino della Reale Accademia Medica di Roma*, Anno ix. No. 1, that, at a sitting of the Academy in December 1882, the subject of my oration was discussed with so much warmth that, at the ordinary meeting in January 1883, when the secretary's minutes of the previous meeting were read, the president and more than one member suggested that the record of some too lively expressions with regard to my discourse ('alcune espressioni troppo vivaci dette a proposito del discorso del dott. Johnson') should be modified, and, above all, that it should not be published in the Proceedings of the Academy.

The secretary stated that, while he had deemed it right to report literally the speech of Professor Scalzi, the record of expressions which might have been uttered in the heat of discussion should, as is customary, be modified in the direction of greater mildness ('nel senso della maggior mitezza'). It was also announced that Professor Scalzi had undertaken the task of replying to me.

I must here express my surprise and regret that my defence of Harvey against his Italian accusers should have so much disturbed the equanimity of the Royal Academy of Rome.

Professor Scalzi's promised reply was read, and received with applause, at the sitting of the Academy, February 25, 1883—the title of the discourse being *In difesa di Andrea Cesalpino Scopritore della grande Circolazione del Sangue. Risposta al chiarissimo prof. Johnson di Londra.* It was published in the *Bulletino*, Anno ix. No. 2, and I am indebted for the opportunity of reading it, not to its illustrious author—from whom surely I might have expected to receive it—but to my friend Sir James Paget.

Although Professor Scalzi's advocacy of Cesalpino's claims is a mere repetition of statements and arguments which I had

met and refuted in my oration, I replied to him at some length in *A Defence of Harvey*,[1] which was published in 1884, and I will here give one illustration of the Professor's method of defending Cesalpino and attacking Harvey. He repeats the statement of Dr. Ceradini, that from Cesalpino's writings Harvey might have obtained a complete knowledge of the circulation. He also maintains that Cesalpino's works, having arrived at a fourth edition, and having become very notorious in consequence of the controversy (mainly, however, theological) which they provoked, it is not to be supposed that Harvey could have been ignorant of them. He goes on to say that Harvey's silence with regard to these writings excites a suspicion that he, wishing it to appear that the new doctrine was a product of his own brain, was unwilling to indicate where he had obtained the fundamental facts. If once he had uttered the name of Cesalpino, he feared that he might reveal the true source of his discovery.[2]

One obvious remark suggested by this monstrous charge of contemptible dishonesty on the part of Harvey is that, since Cesalpino's writings were so well known as they are declared to have been, Harvey's silence would not have prevented his contemporaries—some of whom were ready enough to use every means of attack—from exposing his plagiarism, if they could have shown that Cesalpino had discovered and taught the true doctrine of the systemic circulation.

The best and most complete refutation of these calumnious charges against Harvey is to be found in the indisputable proofs which I have given that Cesalpino was entirely ignorant of that which his fellow-countrymen, apparently misled by an unreasoning chauvinism, now maintain that he discovered.

Professor Scalzi appeals to me to listen to what he calls 'the impartial verdict of my illustrious and learned fellow-countrymen, the brothers Hunter,' who he believes, on the authority of Brambilla, have expressed their surprise that Harvey should have had the credit of discovering the circulation.

[1] Smith, Elder, & Co.

[2] 'Il nome del Cesalpino, solo una volta uscito dalla sua bocca, temeva potesse rivelare la sorgente vera del ritrovato.'

But here the Professor has been misinformed. Dr. Wm. Hunter, who alone of the brothers has referred to this subject,[1] while he admits that Harvey discovered the circulation, differs from the rest of the scientific world in his opinion that very little credit is due to him for having made a discovery to which the facts previously known so obviously pointed.

'There seems,' he says, 'to have been nothing more required for making the discovery than laying aside gross prejudices, and considering fairly some obvious truths. It is the more amazing that this discovery was left for Harvey, when we consider that he was near a hundred years after Vesalius, in which interval many great men had appeared, and anatomical schools had flourished in many different parts of Europe. And what is still more astonishing, Servetus first and Columbus afterwards—both in the time of Vesalius—had clearly given the circulation through the lungs, which we may reckon at least three quarters of the discovery; and Cesalpinus had, many years before Harvey, published in three different treatises all that was wanting to Servetus to make the circulation quite complete. But Providence meant to reserve this honour for Harvey, and would not let men see what was before them, nor understand what they read.'

Whatever may be our opinion with regard to Dr. William Hunter's singular estimate of the problem to be solved, and the supposed interposition of Providence in favour of Harvey, it is evident that he considered Harvey, and not Cesalpino, to have been the actual discoverer of the circulation, and this Professor Scalzi would have seen if he had not been misled by quoting his authority at second hand.

It would appear that Dr. Hunter very much under-estimated the difficulty which the vast majority of mankind experience in 'laying aside gross prejudices, and considering fairly some obvious truths.'

Professor Scalzi concludes his address by a comparison of the relative position of Copernicus and Galileo in the history of astronomical science with that of Cesalpino and Harvey in physiological research. As Copernicus discovered and Galileo

[1] Two Introductory Lectures to his course of Anatomical Lectures. London, 1784.

demonstrated the solar system, so, he says, Cesalpino was the discoverer and Harvey the demonstrator of the circulation.

Now upon this I venture to suggest that if Copernicus had made some such assertion as that while the sun is the centre of our system during the day the moon occupies that central position during the night, he would not have been hailed as a great astronomical discoverer: yet such a proposition would not have been more inconsistent with truth, nor have afforded more conclusive proof of his ignorance, than do Cesalpino's statements, that while the blood passes from the heart to the extremities during the waking state, it returns to the centre during sleep; that all the blood-vessels, both arteries and veins, terminate in nerves; that the urine is secreted by the emulgent veins which pass from the vena cava to the kidneys; that as rivulets draw water from a fountain, so do the veins and arteries draw blood from the heart; and that when the arteries of the neck are obstructed, the blood finds its way to the brain through the veins which communicate by inosculation with the arteries.

These confused utterances would alone suffice to prove Cesalpino's ignorance of the course and the cause of the blood's movement through the systemic vessels. He supposed that the veins and the arteries alike *draw* blood from the heart; and it was left for Harvey to demonstrate that the heart *propels* the blood into the arteries and receives it back through the veins. The difference between Cesalpino's imaginings and Harvey's demonstration is the difference between darkness and light; between almost entire ignorance and the fulness of knowledge—knowledge obtained, as Harvey most truly declared,[1] 'not from books, but from dissections; not from the positions of philosophers, but from the fabric of nature.'

To maintain that Harvey acquired his knowledge of the systemic circulation from the writings of Cesalpino is about as reasonable as it would be to assert that Copernicus, Galileo, and Newton learnt scientific astronomy from the book of Genesis.

[1] See *ante*, p. 852.

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