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CLINICAL LECTURES.

" Multum egerunt, qui ante nos fuerunt, sed non peregerunt ; multum adhuc restat operis, multumque restabit, nec ulli nato post mille sæcula præcluditur occasio aliquid adhuc adjiciendi."—SENECA.

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CLINICAL LECTURES.

Nat Hosp

BY

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SECOND EDITION,

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DEDICATIONS.

[*To the First Volume, published in* 1854.]

TO

WILLIAM STREET, Esq.,

FORMERLY OF NORWOOD, SURREY,

THIS VOLUME IS DEDICATED,

IN ACKNOWLEDGMENT OF A LONG AND VALUED FRIENDSHIP.

[*To the Second Volume, published in* 1857.]

TO

WILLIAM BOWMAN, F.R.S.,

ETC., ETC.:

THESE PAGES ARE INSCRIBED,

BY HIS FRIEND,

THE AUTHOR.

[*To the last Volume, published in* 1859.]

TO THE FORMER AND PRESENT

HOUSE-PHYSICIANS AND CLINICAL CLERKS

OF KING'S COLLEGE HOSPITAL,

WHO HAVE

FOR NEARLY TWENTY YEARS

KINDLY AND ABLY ASSISTED THE AUTHOR IN HIS CLINICAL OBSERVATIONS:

THESE PAGES ARE DEDICATED,

WITH EVERY FEELING OF AFFECTION AND THANKFULNESS,

BY THEIR SINCERE FRIEND,

THE AUTHOR.

EDITOR'S PREFACE.

At the request of the executors of my late friend and former teacher, I have undertaken to revise a new edition of his Clinical Lectures. It seemed to me, on many grounds, desirable that the Lectures, which appeared successively in the years 1854, 1857, and 1859, should be republished, with as little alteration in the arrangement as was consistent with their being collected in a single volume.

I have placed together in a separate form, as an Introduction to the present publication, the remarks on Clinical Instruction, and on the treatment of Acute Disease, which were originally prefixed as Prefaces to the volumes "On Urinary Diseases" and "On Certain Acute Diseases;" while Lectures I and II, "On the Mode of Taking Cases" and "On the Diagnosis of Disease," are now published for the first time. As Dr. Todd has explained his general views on the principles of medicine more fully in the volume "On Certain Acute Diseases," which was finished just before his lamented death, I have ventured to place these lectures first in the present edition: then follow the Lectures "On Urinary Diseases, Dropsies, and Gout" (Lectures XVII to XXXII); and those "On

Paralysis and Diseases of the Nervous System" (Lectures XXXIII to LII). The order of appearance of the Lectures has, therefore, been reversed.

The numbers attached to the cases and lectures have been altered to suit the order in which they occur in the present volume, and a copious index has been added. The Dedications and the Prefaces have been retained, and the dates at which they were written have been affixed.

L. S. B.

61, Grosvenor Street;
December, 1860.

AUTHOR'S PREFACES

TO THE SEPARATE VOLUMES.

ADVERTISEMENT

TO THE LECTURES "ON PARALYSIS, CERTAIN DISEASES OF THE BRAIN, AND OTHER AFFECTIONS OF THE NERVOUS SYSTEM." —FEBRUARY, 1854.

(Lectures XXXIII to LII in the present Volume.)

THESE Lectures were delivered in the theatre of King's College Hospital, on various occasions, during the last ten years. Influenced mainly by the expressed desire of a large number of those to whom they were addressed, I am now induced to collect them.

Having been given from time to time, as cases presented themselves which demanded explanation or afforded illustration, they must not be regarded as forming, or intended to form, a systematic course, or even part of one.

The present series treats of certain affections of the nervous system. They have been printed from the MS. notes of one of my pupils, which were subsequently revised by myself.* Part has already appeared in one of the weekly medical periodicals; the remainder is now published for the first time. All have been subjected to fresh revision, and I have added more cases, and some details not suited to oral discourses.

Should it appear that I have not over-estimated the fitness of these Lectures for publication (and I am not without misgivings on this point), I shall venture to furnish some further contributions of a similar nature, not only on nervous but on other diseases likewise.

* To Dr. Hyde Salter, Dr. Lionel Beale, and Dr. Conway Evans, my best thanks are due, for the assistance which they have rendered me in the lectures of this volume.

ADVERTISEMENT TO THE SECOND EDITION OF THE LECTURES "ON PARALYSIS, ETC."—AUGUST, 1856.

A SECOND edition of this book having been called for, the Author has taken the opportunity of carefully revising the Lectures, and making some additions and alterations.

The object of the Lectures has been to teach, by examples, various important points in the natural history, pathology, and therapeutics of paralytic, cerebral, and other nervous affections. The Author has seen no reason to alter or modify any of the views, whether of pathology or therapeutics, expressed in the first edition; and he now ventures again to submit his opinions, after due reconsideration, to the friendly criticism and impartial judgment of his professional brethren.

CONCLUSION OF PREFACE TO THE "CLINICAL LECTURES ON CERTAIN ACUTE DISEASES."

(Lectures III to XVI in present Volume.)

IN concluding these remarks, which have extended further than the Author intended, it only remains for him to return his most cordial thanks to his highly intelligent friend and former pupil, Dr. Liveing, for his valuable aid in carrying these pages through the press, and to his friends Dr. Hyde Salter, Professor Beale, and Dr. Conway Evans, by whom several of the Lectures were first reported.

26, BROOK STREET, GROSVENOR SQUARE;
December 28th, 1859.

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CLINICAL LECTURES.

INTRODUCTION.

ON CLINICAL INSTRUCTION, AND ON THE TREATMENT OF ACUTE DISEASE.

In these Lectures, my aim has been to teach by examples; to inculcate cardinal points of diagnosis, treatment, and pathology by observations made at the bedside, and by illustrations drawn from suitable cases.

For much of the materials out of which this volume has been formed, I am indebted to several of my former pupils, who, acting as my clinical clerks, and aiding in various ways in chemical and physiological inquiries, have kept the records of the cases which form the basis of the Lectures.

Let me take this opportunity of remarking how important, and I may add, responsible an office in our great hospitals, whether metropolitan or provincial, is that of a clinical clerk. It is not less so as regards the interests of the individual who holds the office, than with reference to the good of the patients, and to the advancement of medical science.

No "Clinic" can be properly conducted without the active co-operation of active and industrious clinical clerks. Nor can any better advice be given to a young man, preparing for the medical profession, than that he should act in this capacity in the wards of a general hospital, even although it may demand a very large portion of his time.

An experience of nearly twenty years as a clinical teacher

enables me to express my opinion that, in our London hospitals and schools, sufficient encouragement is not given to students to devote themselves to this best mode of gaining practical knowledge. I would add, indeed, that it is not merely the *best*, but, in truth, the *only* mode of attaining a practical familiarity with disease; for if the student be not officially a clinical clerk, he must be so for himself, in keeping records of the cases which come before him.

It is not likely that the records of cases kept by a young student, as yet wholly inexperienced in the art of observing, would be of any value even to himself in after years. And there is danger lest the unaided observations of such a beginner might lead him into serious errors, which would give his mind an unfavorable bias. It is, therefore, highly desirable that, in defect of a sufficient number of official posts, there should be ample means and appliances in our hospitals (those especially which form parts of medical schools) for directing and assisting students in making observations, and keeping notes for themselves. Men should work in a hospital, as they do in a laboratory, *under direction:* and the Director should be one who is himself skilled and experienced, and able to teach others, while he is observing and collecting facts for himself.

The London hospitals present a vast field for clinical observation and instruction. They offer, I believe, greater opportunities to the student of medicine and surgery than even the gigantic hospitals of Paris, Berlin, and Vienna, inasmuch as with us the beds are all occupied with important cases, while in the latter institutions many find an asylum who, in this country, would be consigned to the workhouse. But in the foreign hospitals clinical work (under which term I would include observation as well as teaching) is more thoroughly systematized; and they are, on that account, made more available for the advancement of medical science.

A physician, whose mind is thoroughly imbued with the importance of the study of medicine in what I would call a clinical spirit, cannot visit his patients in a hospital and make the necessary inquiries respecting their several maladies without giving clinical instruction to the pupils who accompany him, even although he may offer but few remarks to them. But his

class is not likely to derive the full profit from this kind of teaching, unless they follow him regularly and frequently, and thus become gradually trained to habits of observation. A class constant, as regards the individuals composing it, and spreading its attendance over a lengthened period of time, is quite necessary to give full scope to the teacher; and regularity of attendance on the part of the learner is especially needed, that he may obtain the greatest amount of instruction.

Notwithstanding that there are many physicians and surgeons connected with our London hospitals admirably qualified to give such instruction as I have described, it does not appear to me that the work done in this direction is by any means adequate to our great opportunities; and this, I believe, is due mainly to the existence of certain hindrances arising out of the arrangements connected with the ordinary working of these institutions, which I can scarcely think would be suffered to remain as they are, if the evils to which they give rise were fairly looked at.

The hindrances to which I refer, are—

First—The period of the day at which our hospitals are usually visited. Noon, or soon after, is generally the time fixed upon for this purpose. It is at this hour that men engaged in private business are the most liable to interruption, and punctual attendance is always difficult, and often impossible. Owing also to the pressure of other engagements (and this affects the student as well as the physician or surgeon), the same quantity of time cannot be given as at the early period of the day, and the visit must be often contracted within the shortest limits.

Secondly—Much impediment is thrown in the way of clinical pursuits by the great number and length of the courses of lectures which students are called upon to attend in the schools. Although, in some degree, curtailed of late years, these may be still further diminished with advantage to both teacher and pupil. For example, how unnecessary in the present state of medical literature are long courses of lectures on the practice of medicine or surgery, or, indeed, on any subject not requiring demonstration and experiment! How much better would it be to confine the lectures on these subjects to the discussion of difficult, doubtful, and important points of pathology and practice, preceded by a sufficient statement of

first principles suitable for the uninitiated! And in such lectures care should be taken to indicate the best sources of information, and the most valuable works of reference, and to encourage habits of research and study.*

Men would, under these circumstances, be led to read and digest standard authors, to think for themselves, and to discard the system now prevailing (but only of recent growth) of learning manuals by rote. And much of the time now spent in listening to lectures (the greater part of which can be no more than what may be better read at home) might be devoted to hospital work —and the pupil would acquire by his own sight, and touch, and hearing the knowledge which, in the lecture room, he can receive only upon the description and authority of another.

And if, along with such changes as these, the examinations instituted for medical diplomas and degrees were conducted in a manner adapted to test positive knowledge, rather than to encourage a flippant expertness in answering questions, the result would be a greatly diminished resort to the grinding process, and a more healthy and zealous pursuit of scientific and practical knowledge for its own sake. Idle and indolent men would exist under all systems; but with more guidance, and less dependence on the dicta of a teacher, those disposed to work would devote themselves to their pursuits with much more ease, and with a sense of freedom from the thraldom of scholastic routine.

Thirdly—Further impediment to clinical teaching arises from the present meaningless custom of crowding the hospital visits of physicians and surgeons, and sometimes of all the physicians and surgeons, into one and the same hour. The inevitable consequence of such a practice is a desultory, irregular attendance of the class. To-day, the students throng to see one or two remarkable cases, or grave surgical operations; to-morrow, they rush with equal eagerness after some new curiosity; but the steady, uniform, day-after-day watching of disease in the hospital wards, is greatly discouraged by this want of method, and the

* The lectures of the late Professor Smyth, of Cambridge, on Modern History, seem to me to afford the best model of the kind of prælections which, as applied to Surgery or Medicine or Materia Medica, would at once interest the student, and guide him to study and think for himself.

clinical teacher labours under the disadvantage of not being regularly followed by a class constantly consisting of the same members.

It seems to me that in every hospital there should be clinical teachers specially appointed for that purpose. The office of Clinical Professor may be taken in turns by the hospital physicians and surgeons; and there should be one, or not more than two, constantly engaged in each branch of practice. If there be but one, the visit should take place daily; if two, each clinical teacher should visit on the alternate days. The omission of each alternate day is unfavorable to the clinical watching of patients labouring under acute disease. But the constant attendance of two clinical teachers, two physicians and two surgeons, appears to me to have advantages which more than counterbalance this occasional difficulty, which, indeed, need never amount to anything more than an inconvenience, inasmuch as abundant opportunity would exist for seeing such acute cases daily.*

In making these remarks—founded, as they are, upon a long experience, and suggested by much consideration of the subject, and, I may add, by great interest in the improvement of medical education—I would not be understood to undervalue what has been done of late years in the clinical school of this metropolis, and of Ireland and Scotland.

The many works which have issued from the press of these

* At Guy's Hospital, the excellent plan of clinical wards is adopted: all the most important cases are here collected, as well surgical as medical; one physician and one surgeon takes the duty of Clinical Professor, each for three months, and a daily visit is paid. But the medical and surgical wards are visited at the same hour. At University College, there are Clinical Professors, and a practical class is conducted for training the pupils in the art of physical diagnosis, by the various means now within our reach. At King's College, an early "clinic" at nine a.m. has been established, medical on three days in the week, surgical on the alternate days. The celebrated "clinic" of Graves and Stokes, at the Meath Hospital in Dublin, was established at an early morning hour, and at a time quite separate from the surgical "clinic." It speedily acquired an European fame, and notwithstanding that its beds were few, afforded abundant material not only for the instruction of a large class of pupils, but for those numerous valuable lectures and monographs, by which Graves and Stokes have taught their generation.

countries upon medical subjects within the last twenty-five years, show that, notwithstanding great disadvantages, British physicians and surgeons are in no degree behind those of other countries, either in original observation or in research: and this would be abundantly established by a bare enumeration of the names of men now living, who have (each in his own department) contributed to the rapid advancement of medical and surgical science within that period.

And I would especially suggest to those responsible for, and interested in, the advancement of medical education, that in every school, hospital attendance and clinical study should be made the nucleus of the teaching all other subjects, in preference to the existing system, which, in effect, makes it subordinate to the rest. I would, on the contrary, limit and adapt the teaching of other matters to the increased time which ought to be devoted to the clinical study of medicine and surgery.

It will be objected to any proposition for opening more widely our hospital wards for the study of disease, that the more constant attendance of medical students in them will be distasteful and injurious to the patients. I do not believe that such a consequence would ensue. The presence of medical students in our wards, whether assisting in, or witnessing, what is going on there, is one of the best guarantees that the poor inmates are well looked after. I have never known any patient rendered worse by the presence of a large class, whilst I have seen very many greatly benefited by the kind interest and the careful watching of the young men in attendance. It would be for the physicians and surgeons to give such a tone to the proceedings there, as would afford the most perfect security against any disregard for the comfort and feelings of patients. And I have sufficient faith in the correct and manly feeling of that much improved and improving class, the British students of medicine, to believe that out of such increased facilities of study, they would prove themselves important and valuable auxiliaries in the promotion of clinical investigations.

No one now practising with a knowledge of what medicine and surgery were thirty years ago, will hesitate to admit that in that period both these branches of the healing art have made great advances. How much has been done for diagnosis! How

many forms of disease then but little known, are now familiar to the physician! Our power of controlling the progress of disease, how has it not increased! Nay, can we not boast that the course of that scourge of our race, tubercular phthisis, may be retarded, and life prolonged—partly by its earlier detection, partly by the use of new agents! And in surgery, how great are the advances made, especially in reparative and conservative surgery! How admirable the means now used—suggested, indeed, long ago, by Park, adopted early by Crampton, and others—to save limbs formerly devoted to destruction! and how successful have these means been in the hands of Jones of Jersey, and our best hospital surgeons!

Truly, if much has been done, there is yet much more to do. Let us interest and employ as many as possible in this work of Clinical research; and especially let us enlist young minds, while they are yet free from the baneful influence of routine.

ON THE TREATMENT OF ACUTE DISEASE.

There will (the author believes) be found in the Lectures on Acute Diseases (Lectures III to XVI) evidence enough to show that the ordinary so-called antiphlogistic treatment is unnecessary (to say the least) for the cure of acute internal inflammations; and that the supposed necessity for such treatment rested upon an untenable hypothesis respecting the nature of inflammation and of fevers, and cannot be regarded as a legitimate induction from accurately observed clinical facts.

The conclusions, which the clinical observations detailed in the lectures tend more or less to establish, may be summed up in the following propositions:

1. That the notion so long prevalent in the schools, that acute disease can be prevented or cured by means which depress and reduce vital and nervous power, is altogether fallacious.

2. That acute disease is not curable by the direct influence of any form of drug or any known remedial agent, excepting when it is capable of acting as an antidote, or of neutralizing a poison,

on the presence of which in the system the disease may depend (*materies morbi*).

3. That disease is cured by natural processes, to promote which, in their full vigour, vital power must be upheld. Remedies, whether in the shape of drugs, which exercise a special physiological influence on the system, or in whatever form, are useful only so far as they may excite, assist, or promote these natural curative processes.

4. That it should be the aim of the physician (after he has sedulously studied the clinical history of disease, and made himself master of its diagnosis), to inquire minutely into the intimate nature of these curative processes—their physiology, so to speak—to discover the best means of assisting them, to search for antidotes to morbid poisons, and to ascertain the best and most convenient methods of upholding vital power.

If one may venture a suggestion respecting the future of pathology, and of practice founded on it, it would be that a time is not far distant when all men who practise medicine in a scientific spirit, and divested of the trammels of routine, will discard the distinction of acute inflammations and acute disease in general, into *asthenic* and *sthenic*—that all these maladies will be regarded as more or less asthenic, and as promoting more or less an undue waste of tissue, and that, in treatment, an object of primary importance will be the early adoption of means to uphold vital power, and the watchful and continued use of them throughout the duration of the case.

It will not be affirmed by any one that the doctrines of a science so abstruse and so difficult as pathology, should not be reviewed and reconsidered from time to time. There never was a period when a candid and ample reconsideration of general pathology promised more fruitful results than the present. Our vastly extended acquaintance with anatomy and physiology, the greatly enlarged security of the basis on which our knowledge of function rests, the much increased accumulation of facts of clinical history, all afford most important data for new inductions. And I would remark that such inductions ought to be made from the deranged functions of the living rather than from facts of morbid anatomy, which properly should rank with the facts of clinical history, and which, in reality, are inferior in

value to most of the phenomena of disease during life, being no more than marks of the ravages of disease, and affording comparatively little insight into its intimate nature. The real basis of all pathological inquiry must be clinical research, made with the fullest appreciation of the facts of anatomy and physiology; *mere* morbid anatomy leads necessarily to erroneous views of pathology and practice.

Such a review of pathological doctrine, as I have alluded to, will assign its true value to the influence of the *quantity* of blood in the production of disease; will determine what is the real point of departure of morbid change, whether it is due to a superabundant or a deficient blood supply; or whether the condition of the blood supply is the consequence of a primary morbid change, such as a disturbed innervation, or a contamination, or waste of the elementary tissue.

The following problem lies at the root of the pathology of acute disease, and it has never yet received an adequate explanation, and is uniformly ignored by the zealous advocates of the so-called antiphlogistic method:

A man has a patch of pneumonia in the base of his left lung, brought on (he conjectures) by some exposure to cold. Why is it in his *left* lung? why at the base rather than the apex? how is it limited to a certain patch? In other words, what is the proximate cause of this localized derangement of nutrition?

If so much has yet to be determined as regards the very alphabet of pathology, it cannot surely be thought presumptuous to question the soundness of the practice founded on such crude doctrines.

And more especially are we justified in such a course, when it is considered that much of the practice of former days rests upon the insecure foundation of a partial and imperfect diagnosis of the primary disease, and a very inadequate interpretation of the subsequent phenomena of the case. Thus, in many instances the practitioner found himself treating a disease of the clinical history of which he had but a very imperfect knowledge, and on these occasions, he would be led to attribute changes in the symptoms (whether for better or for worse), which were essentially part of the ordinary course of the malady, to the influence of certain remedies. The temptation to draw

hasty conclusions as to cause and effect, and to adopt the *post hoc ergo propter hoc*, so common among all classes, unlearned or learned, often likewise stood in the way of sound reasoning upon these subjects.

Did space permit, it would be easy to adduce many instances to show that a more exact diagnosis must necessarily lead to altered views of practice. A few may be briefly referred to.

The precise discrimination of the different forms of continued fever has arisen out of the clinical investigations of the last fifteen or twenty years. And it is now ascertained that continued fever may be caused by any one of three separate poisons, each of which developes its characteristic phenomena, namely, those of typhus, typhoid or pythogenic, and relapsing fevers. Nevertheless, there is good reason for believing that any two of these poisons may coexist in the same individual and produce their special phenomena. Dr. Murchison's researches render it highly probable that the great epidemics which formerly ravaged Ireland, and some of the large towns of Scotland, were chiefly the fever which is called *relapsing*, and that it was by this form of fever that the practice of bleeding and low diet was best borne.

However this may be, it is clear that even just to the present day physicians were not in a position to discriminate whether certain changes in the phenomena of the disease were due to the influence of their remedies, or were simply part and parcel of the ordinary train of the clinical phenomena of the disease.

The whole class of the so-called *apoplectic* diseases must now be viewed, as regards their pathology and treatment, in a totally different light from that in which they were regarded formerly, even so lately as the celebrated work of Abercrombie. Both the pathology and practice of that able physician must now be, with but little exception, entirely discarded.

And to what is this owing? Undoubtedly to a more extended knowledge of clinical history, and to the consequent more precise discrimination of the different forms of brain disturbance, which lead to comatose and paralytic phenomena. For example, the author has shown long since that many cases of hemiplegia are the result, not of a clot in, or of a rupture of fibres of the brain, but simply of the influence of an epileptic fit. The

history of these cases is, that the patients suddenly fall into epileptic coma, with or without convulsion, and that they emerge from it with more or less perfect hemiplegia. This paralysis often gets well with remarkable rapidity after a few hours, very often after a few days, and sometimes after some weeks.

A case of this kind falling into the hands of a practitioner accustomed to use the lancet freely in diseases tending to coma (the so-called congestion of the brain), would not suffer any disadvantage from a moderate bleeding and from purging; the patient would speedily recover, and the case would be quoted as a glaring instance of the excellent effect of the treatment, whereas an exact diagnosis might have saved unnecessary practice, and a familiar knowledge of clinical history would have enabled the practitioner to have foreseen and foretold the course which the disease would be likely to take.

The much vaunted powers of mercury as a remedy, not only to promote the resolution of acute inflammation, but also to cause the absorption of its product, lymph, rests first upon a false analogy; and, secondly, upon imperfect knowledge of clinical history.

It was found that iritis, the result of the influence of syphilis, was cured under the use of mercury with a rapidity and certainty which did not belong to any other kind of treatment. Lymph effused in more or less quantity upon the surface of the iris, and even recent adhesions gluing the margin of the pupil to the capsule of the lens, quickly melted away under the peculiar change which mercury was capable of inducing.

Primâ facie, there was no more reasonable suggestion than that mercury would exercise a similar influence on inflammations of like tissues to the membrane of the anterior chamber, and promote the removal of any lymph that might be effused upon them, preventing adhesions or dissolving them if formed.

But although it was a perfectly reasonable suggestion to give full trial to the use of mercury in inflammations of serous membranes, the analogy did not justify the expectation of such decisive results as were obtained in syphilitic iritis, although sufficient to call for experiments. In fact, the analogy was inexact: there was no further resemblance between syphilitic

iritis and rheumatic pericarditis or pleurisy, than in the tendency of both inflammations to develope lymph, and to cause adhesion of opposed surfaces. Nor in any of their other effects was there any such marked similarity between the syphilitic and the rheumatic poisons as would fully justify the expectation that the experimental trial of mercury for the cure of such inflammations would prove successful.

And what has been the result of the long-tried use of mercury in both affections, syphilitic as well as rheumatic? Why, that whilst, in the former, mercurial treatment has never ceased to find favour with practical men, in the latter, such has not been the case. No one would now venture to assert that mercurial influence, however quickly induced, ever checked pericarditis or pleurisy; nor would it be easy to adduce an instance in which, with any reasonable degree of certainty, it could be stated that mercury broke down adhesions, or prevented their occurrence.

Examples were no doubt of frequent occurrence in which such effects *appeared* to follow the use of mercury. But a more intimate acquaintance with clinical history has taught physicians that changes are apt to occur which simulate the absorption of a lymph-deposit. It is very common to find a marked pericardial friction sound disappear for a time, and the hopeful practitioner is led to regard this as the result of his remedies, sometimes of a few leeches or a cupping, sometimes of a blister, but more especially of the use of mercury. In a day or two, the friction sound returns, and the practitioner is forced to conclude that his remedies have not produced the desired result. And there are the best reasons for inferring that the early temporary suspension of the friction sound is due in a large number of cases to a slight liquid effusion, which separates the opposed rough surfaces, and so destroys the friction sound, which, however, returns on the re-absorption of the liquid. Moreover, it is now proved by a multitude of examples (and some will be found recorded in the following pages), that pericarditis will do perfectly well without mercury, nay, better than with it; and that in general the real benefit which the patient derives is to be referred to the opium with which the questionable mercury is combined.

How often and often has the author most anxiously watched

a mitral bellows-murmur, caused by recent endocarditis in patients under mercurial treatment, hoping to discover that it had disappeared under the mercurial influence! Yet in his whole experience he is unable to discover a single case in which such a murmur had been even modified by any influence save that of good nourishment, as tending to maintain a normal state of blood; and of time, as furnishing opportunity for the mechanical wearing down (by attrition) of the deposited lymph.

A curious instance may be mentioned in illustration of the way in which an erroneous opinion may be readily formed with respect to the effects of remedies. The author attended, along with a medical friend, a tradesman of middle age, who became quickly, although not suddenly, comatose, with hemiplegia of the right side, and marked rigidity of the muscles of the arm and leg. It was difficult to determine whether the symptoms indicated a superficial apoplectic clot, or were due to a patch of inflamed brain in the left hemisphere. It was resolved to act on the latter view, and mercury was given freely with the intention of producing salivation. About the second or third day of this treatment the patient recovered his consciousness, although retaining a certain degree of somnolency; the paralysed limbs regained a greater degree of power and the muscles became less rigid. During this and the succeeding day everything favoured the conclusion that the mercury was telling upon the inflammatory process, and that it was undergoing resolution. The gums became affected just at this time. But, in another day, the hopes of the patient's friends and attendants were seriously checked by the rapid recurrence of the comatose and paralytic symptoms, in a more severe form, leading to a profound coma, under which the patient succumbed.

The inspection after death showed a considerable apoplectic clot on the surface of the left hemisphere, causing a deep indentation on the convolutions, which did not disappear when the clot was removed, such was the degree of pressure on the cerebral surface. On cutting through the clot, it was found to consist very distinctly of two portions, one brownish in colour, and looking old—the other consisting of dark, currant-jelly-like coagulum, which had been only quite recently effused.

It was plain then, in this case, that the original cause of the

symptoms was a meningeal effusion of blood compressing the brain to a very great extent. After a few days the watery part of the coagulum was absorbed, and the shock which the brain had received at the first effusion of blood, had subsided. It was then that the restoration of consciousness and the improvement in the other symptoms, which were attributed to the influence of mercury, took place. This improvement, however, soon gave way before a fresh hæmorrhage, which led quickly to a fatal result.

Had an exact diagnosis been possible with certainty in this case, the patient would have been spared the mercurial course to which he was subjected, and the physicians would have had nothing else to do but to support the powers of life, with the hope, that by the brain adapting itself to the pressure, and the gradual absorption or contraction of the clot, the life of the patient might be considerably prolonged. And, indeed, this would most probably have been the case in this patient, had it not been for the second hæmorrhage.

Enough has been said, the author hopes, by way of apology, for his venturing to dissent from current views of pathology and practice, sanctioned as they are by great names, both dead and living—for which the author will yield to no one in admiration and respect; but

Amicus Plato, amicus Socrates, magis amica veritas.

The author has not referred to the hypothesis suggested by some who admit the necessity of a considerable modification of practice in the treatment of acute diseases; namely, that the type of disease has undergone material change of late years, and has assumed a much lower grade as regards vital power, owing either to some change in the human constitution, or to some atmospheric modification which has taken place in recent times. It is supposed that this modification dates from the period of the first introduction of cholera into these countries.

Upon this point the author can say that he has been a not careless observer of disease for several years antecedent to the first cholera epidemic. At no time was the antiphlogistic treatment (so called) more rife than some years prior to the cholera

epidemic, and many excellent observers were then beginning to see that it was carried too far, and was inadequate for its object in either cutting short or curing disease. Certainly opportunities of studying the morbid anatomy of acute diseases, pneumonia, pericarditis, endocarditis, pleurisy, were then, and for many years afterwards, much more common than now, when such inspections are among the least frequent in our hospital theatres. The author has notes of many cases treated in this way, which he is confident would have recovered, had vital power been not only spared, but upheld.

The author would venture to doubt the proposition that disease is of a lower type now than it was twenty or thirty years ago. Certainly we have long been spared those ravaging epidemics of fevers, dysenteries, exanthemata, all of which exhibited innumerable examples of the lowest type of disease. Their comparative disappearance now is due in part, no doubt, to the improved condition of the people, better food, better clothing, cleaner and better-ventilated dwellings, and to many wise preventive sanitary measures. But, on the other hand, population is vastly increased, overcrowding exists to a large extent, and were disease of a very low type, it would spread freely, and epidemics would be common. It is well known that such is not the case, and that the fevers which were formerly the scourge of the poor, occur now on a very limited scale.

LECTURE I.

ON THE MODE OF TAKING CASES,* AND ON THE IMPORTANCE OF CLINICAL OBSERVATION.

Gentlemen,—I much wish to call your attention to a subject about which you very often hear me speak, and which is a very important one, and one, moreover, whose discussion is, perhaps, more important at the commencement of the session than at any other time. The subject to which I allude is that of *taking cases;* and not only should clinical clerks take some notice of the observations which I am going to offer, but also each one of you individually, for it is highly necessary that some arrangement be adopted by which a great number of the students attending the hospital should employ themselves in taking cases. There is no portion of your studies that will be of more value or of greater use to you in your future career than accustoming yourselves to register cases; and you must not imagine that this is a simple, mechanical work, for it is, in reality, a valuable intellectual employment, and one which, if carried out in a proper spirit, exercises you in all the most important parts of your profession.

Case-taking consists in recording accurately the condition of the various functions with reference to anything going astray in those functions; and its importance has a threefold bearing, viz.—

First—Upon medical science, which is purely a science of observation, and one which requires for its basis the possession of a great number of well-observed and well-ascertained facts. Indeed, without these we cannot have an accurate science of medicine at all; and the rapid progress which medical science

* Reported by Dr. Conway Evans.

has made in the present day is almost entirely due to the careful collection and collation of numerous well-observed facts.

Secondly—It is highly important to each individual: it exercises his powers of observation. A man may go into a ward and see a patient, feel his pulse, and look at his tongue, and notice a few general points, and soon get in this manner sufficient knowledge to enable him to form a pretty correct opinion of the nature of the case; but he must do much more than this. He must carefully inquire into the history of the case. Careless habits of this kind, once acquired, are very apt to grow upon a person; and if you closely watch our hospital practice, you will find that the mistakes which are made generally arise from the history of the patient not having been carefully taken, or from some symptom having been omitted to be noted down.

Thirdly—Case-taking is of great importance to others, because by that means only can the experience of others be made generally useful. It is in this way that cases of a past age are available at the present day; but they are less useful than those now recorded, inasmuch as the means of observation in those days were far from being so accurate as they now are. Take, for example, a case of disease within the chest. We can now note with accuracy the condition of the diseased lungs or heart; indeed, to such a degree of perfection has the science of auscultation attained, that we can, to all intents and purposes, see into the chest. By means of the microscope and chemical appliances, also, we can ascertain with a very great degree of exactness, from the actual condition of the urine, the state of the kidneys, and of various other organs, of which no note was taken in former times.

Now, a well-noted case consists of three parts, viz.—

First—It ought to contain a correct and exact description of symptoms, care being taken to note the condition of all the functions. Take, for example, a case of disease of the heart. When the patient presents himself for examination we should deal with the case in reference to our notes just as if we had no idea of the nature of the malady, our office being to note down all the phenomena present, whether normal or otherwise, and then to draw conclusions from our notes as to what are the functions which are gone astray. It is important, also, that

we should ascertain the state of particular functions according to some method; thus, we may take first the nervous system, and then proceed to the circulatory and respiratory systems, the genito-urinary system, and so on; and then, if we find that two or more functions are affected, we should endeavour to determine whether there be any connection between them—whether the error in the one is, or is not, dependent on disease in the other.

The second point in a well-noted case is, that it should give a clear history of the patient's antecedents, and a good account of the mode of access of the present symptoms. It is very important in certain cases; indeed, in many we are unable to form an accurate diagnosis without a knowledge of the patient's history. Take, for instance, a case of chest affection: a patient comes to us with some disturbance of the respiratory organs. He complains of cough, and expectorates, and he tells us that he has been losing flesh, and some suspicion arises in the mind of the practitioner as to whether these symptoms depend on tubercular disease of the lungs. Now, we know from the history of tubercular disease of the lungs that this malady is very apt to be hereditary; and here the history of the patient's family will contribute to the formation of a correct diagnosis. Indeed, oftentimes in these cases the question of hereditary predisposition is of very great importance. The history of the patient himself, too, frequently furnishes important facts; such as, for example, in the case to which I have just referred, the occurrence of hæmoptysis at any time, which is a very unfavorable symptom, and always strongly presumptive of disease of the lung. So, again, in many cases of disease of the heart, the patient's history may afford satisfactory evidence of that organ having been affected during an attack of rheumatic fever, which, perhaps, occurred many years previously. The mode of access of the symptoms, also, is of very great importance in some cases, particularly in those of brain disease. You may have a patient before you, and observe the symptoms present very accurately, but without a knowledge of the manner in which those symptoms came on you can hardly give a correct diagnosis. Take, for instance, a case of hemiplegia: here, whether the paralysis came on suddenly or not, whether it was, or was not, accompanied by coma, and various other points, must be known and

noted to enable us to determine, with any degree of accuracy, the nature of the lesion which gives rise to the symptoms.

The third point in a well-noted case is, that a careful daily register should be kept. The greatest part of the work will have been done when the first two points have been attended to; and I may here observe that the best test of a case having been well taken, so far as I know, is that the physician ought to be able to form a correct diagnosis from the notes without having ever seen the patient.

Accustom yourselves at this early period of your career to take cases; for it often devolves on the medical practitioner to have to transmit the detail of a case under his charge for the opinion of a physician or surgeon. From some circumstance or other the patient is unable to present himself before the physician whom he wishes to consult, but he trusts to you for such a careful analysis of his case that the party consulted may decide, not only as to the nature of the malady, but also as to the mode of treatment which should be adopted; and, unless you can take a case thoroughly and well, you cannot do justice to the patient who commits himself to your care.

Now, to take a case in the way I have described, you should adopt a certain method. A good plan is to examine the patient with respect to the state of all the functions; and the best mode of putting this in practice is to have before you a little tabular view of the various points which are to be noted in the different functions.

These may be arranged under four principal heads:

The first comprehends the head and nervous system, and everything relating to the latter, as the condition of the senses, &c. You should examine the external form of the head, the state of the intellect, and note down the existence of any symptom indicative of disturbance of the functions of the nervous system. For example, you should ascertain whether there be any paralysis; whether the patient has pain in the head, and if he has, you should proceed to inquire into its nature, locality, frequency, continuancy; whether it is superficial or deep, whether it is affected by pressure or by position, or whether it appears to be confined to a particular set of nerves. You should then examine into the condition of the organs

within the cranium; and this done, you should pass on to the organs of sense; and although these particulars take a long time in enumerating, yet in practice you will find they will occupy but a few moments, and I mention them to you thus in detail to show you the necessity of adopting some particular method.

The second head includes the respiratory and circulatory systems, which are so intimately tied together, that the investigation of the one must go along with that of the other. This brings you to the pulse, and to the physical examination of the respiratory organs and the centre of the circulation—the heart. You should note the condition of the walls of the chest, comparing the two sides; then you should compare the movements of the two sides;—and here I may observe that, whenever there is a double organ, you should always compare the one on the right with the one on the left, and ascertain whether the disease be symmetrical or not. In the physical examination the various points which differ from the normal condition should be noted; indeed, the physical examination of the lungs and heart involves a very accurate knowledge of those organs in the healthy state, and, I believe, it is too much the habit of men to set to work with the stethoscope, and to percuss the diseased subject without having made themselves sufficiently familiar with the various sounds of the healthy lung and heart. My advice to those who are beginning their medical studies is, "Practise listening as much as possible to the healthy lung and heart, and then you will be able to detect a very slight departure from the normal condition of those organs." Nothing is better for this purpose than the chest of a child, the listening to which affords an excellent exercise for the ear. The respiratory murmur, which is there heard, is often very closely imitated in the adult lung; and it then leads the practised auscultator to infer that the opposite lung is not in its healthy state. This peculiar respiration, when heard in the adult lung, is termed "puerile;" it is loud, clear, and soft; and it is the very fact of its character which leads to the inference that there is something impeding the breathing in the opposite lung to that in which "the puerile respiration" is audible. From the existence of this alone, I have many times been led to say that there was disease of the

other lung. If, for instance, there be pneumonia, or pleuripneumonia, or pleuritic effusion, or, indeed, if there be anything which interferes materially with the breathing in one lung, and if the other be healthy; then, in the healthy organ "puerile respiration" will invariably be present.

It is also very important that you should make yourselves familiar with the sounds of the heart, and the size and position of that organ are estimated by percussion; for by this means you will soon be able to distinguish differences in the sounds, size, and position of the heart, which you would otherwise be unable to appreciate.

Accustom yourselves, also, to feel the pulse; for it is only by constant exercise that you will acquire that learned touch which is so highly essential to the medical man. Many men seem to regard the phenomena of the pulse as of little importance; but, if time allowed, I could show you that there is no one symptom which is so important as this. It is not so much the frequency as the volume—the quality—of the pulse, which bears a direct ratio to the vital powers of the patient. Take, for instance, the cases of fever, of which there have lately been so many in the hospital; and, I repeat, there is no single point which it is of so much importance to ascertain with accuracy as the condition of the pulse; as I said before, not so much its rate (though that is not to be despised) as its quality: and I think that if you were to lead me blindfolded through the hospital wards, and take me to the beds of fever-patients (mind they must be genuine fever-cases), I should not hesitate from the character of the pulse alone to give a prognosis in almost every case—to say whether the patient had a fair chance of recovery or not. Of course, there are many incidental points in fever, to which due consideration must be given—such as diarrhœa, and a tendency to ulceration of the bowels, which will sometimes destroy life in twelve hours, and which one cannot foresee; but from the pulse alone you may often make a very accurate prognosis of the case.

The next head comprises the abdomen and digestive organs. It includes the physical examination of the belly, noting its size and so on, and the condition of the tongue; and this brings us to the great organs within the abdomen, and the characters of the fæces.

The fourth head includes the urinary and genital organs, the latter only so far as the nature of the case may suggest; but you should always make it a point to ascertain the condition of the urinary organs and of the urine; for if this had been done in former times, and if our predecessors had possessed the facilities which we now enjoy, medical science would, I have no doubt, have made much greater progress than it hitherto has done. In diseases of the nervous system, it is only of late years that this point has been attended to; and we now know that there are many cases of disease of the brain dependent on a morbid condition of the kidneys. There is now a case in the hospital of this nature; and, indeed, numerous cases are met with in practice in which epileptic fits are dependent on a diseased state of the kidneys, and which are, therefore, perfectly curable if we can only succeed in bringing the latter into a healthy condition. A remarkable case of this kind occurred in the hospital last year, and in which a cure was effected by neglecting the head and directing our treatment solely to the kidneys.

In noting the symptoms under the various heads which I have now enumerated, you should also take cognizance of the condition of the system generally; whether there be any febrile disturbance or not; the state of the skin; the temperature of the body; and, where there are well-marked local symptoms, you should dwell on them particularly and describe them accurately.

If the patient die, and an opportunity is afforded for examining the body, it is equally important that the same method, which was employed during life, should be adopted after death; and it should, also, be followed in the same order, viz., nervous system, respiratory and circulatory system, digestive system, and genito-urinary system, for in this way you will be sure not to omit anything of importance. It is highly necessary, also, in the post-mortem examination, that you should not direct your attention to that part only in which disease was expected; as, for instance, examining the abdomen and neglecting the chest, for in this manner it is that many very important morbid conditions have been overlooked.

Those of you who really take an interest in your profession, and who occupy some of your time in taking cases, should observe certain rules. You should make your notes daily,

especially in acute cases. You should never omit the pulse; and, generally, you should also note the breathing and the condition of the principal secretions. The occurrence of anything remarkable should be noted down *at the time;* and no one should more carefully avoid procrastination than a case-taker; for there are many things, such as how and when certain symptoms showed themselves, how they progressed, and so on, which, if not written out at the time, speedily escape the memory of most men.

ON THE IMPORTANCE OF CLINICAL OBSERVATION.

Let me now say a few words in reference to your attendance on the medical and surgical clinical lectures which are so frequently (three times in the week) delivered in the hospital. I cannot urge upon you too strongly the importance of attending to this part of your studies. There is no part of your education which is of such paramount importance as clinical medicine, and the careful watching of cases that may fall under your notice; there is no branch of study you will feel the lack of so severely when you have embarked in the serious duties of practitioners. Let any engagements you may have bend to enable you to be regular in your attendance at the hospital and on the clinical lectures, so that you may have the opportunity of seeing and watching the cases that may from time to time be brought under your notice, and of observing the various changes that may take place in the character of the symptoms from day to day. You must not be content with occasional visits to the hospital, but must attend regularly, so that you may not lose the opportunity of observing for yourselves the various alterations which frequently occur in the course of any particular malady; for if you do lose the opportunities which are offered to you for the investigation of disease, you will have reason to repent of your neglect whenever you commence practising for yourselves.

If you only pay your visits to the sick-wards now and then, it may happen that you will find little to interest you; but if you watch the cases from day to day, you will often notice that

a case which in the outset presented very few points of interest, becomes afterwards of a most interesting character, in consequence of the development of new and often unlooked-for symptoms. The little changes which usually occur from day to day, in ordinary cases, are important likewise as the results of treatment, and well worthy of your careful observation. For all these reasons let me urge upon you the great importance of daily attendance on hospital practice, and the necessity of watching daily the various cases in the wards. By regular attendance only will you be enabled to see the early treatment of the cases, and the results of that treatment manifested in the further progress of the case.

Let me now tell you what we mean by clinical lectures, in order that you may be better able to understand the advantages you will derive from attendance on them. By clinical lectures we mean lectures arising out of bedside observation, and we refer particularly to those cases in the house which illustrate any especial points in the treatment, remarkable either from the manifestation of new symptoms, or from the change of character that may have occurred in the original symptoms of the malady. Lectures which are given with especial reference to these points are denominated clinical lectures, and no lectures can be so instructive as these, for in them you learn the practice of medicine from examples of disease which you have yourselves seen, and from cases which you have yourselves examined. Clinical medicine may be considered the teaching of medicine by cases and examples, and if you are to derive benefit from the clinical lectures, you should see the cases on which the lecture is founded; for if you are not familiar with the cases to which I refer, how little can you enter into the symptoms to which I particularly call attention, neither will you be able to enter into the views I have taken of the case. If you have not listened to the sounds which were manifested during the course of the case, and have not observed the changes that have taken place in the condition of the patient, how little knowledge you will gain when I come to consider the causes and consequences of the disease. I say nothing is so important to you in reference to your future prospects as practitioners, as seeing cases, and carefully observing the various appearances which disease

puts on under different circumstances. And here let me impress upon you the importance of taking notes, not only of the clinical lectures, but of the symptoms which you observe in the various cases when you visit the wards. Notes impress facts and occurrences on the memory which would probably have not been otherwise remembered, or would perhaps have escaped the observation altogether. The greater the care you take in learning the principles of your profession, the more will your opinion be valued by the public; and, what is much more important than the public opinion, by your professional brethren, for they will soon be able to discover the real knowledge you possess. This knowledge can only be founded upon experience and the careful observation of cases. Take notes, then, while you are going round, and when any alteration has taken place in the character of a particular case, you will find a reference to your notes invaluable, if you have registered the various important symptoms since the accession of the malady. At first, as a matter of course, your notes will be badly—and perhaps somewhat clumsily—taken, but by habit and by paying attention to the remarks which you will hear made at the bedside, you will soon acquire an insight into the cases, and a facility for noting down the points of especial interest or importance in the case. These notes will be of great use to you hereafter, when you are in practice, and you will often refer back to them. Some of the most valuable notes which I have were taken when I was a student, and the cases which I saw then have made an impression on my mind which will remain as long as my mind itself remains. You are at a time of life when impressions are very easily made on your mind, and when your perceptive powers are strong, and it therefore behoves you to make the best use of your time, and to become acquainted with everything bearing on the practice of your profession, during the period set apart for your attendance at the hospital.

LECTURE II.

ON THE DIAGNOSIS OF DISEASE.

GENTLEMEN,—As this is the first opportunity I have had of addressing you on clinical matters this session, I shall make my remarks of as general a kind as possible, and shall endeavour to refer to several of the cases now under my care: my theme shall be diagnosis.

The art of diagnosis, or that of distinguishing diseases, must obtain a large share of your attention, while you avail yourselves of the opportunities which the hospital affords. Indeed, the degree of benefit that you will derive from witnessing hospital practice, will be proportionate to the extent to which you exercise yourselves, in distinguishing the maladies with which the patients are afflicted, or the attention you give to the means by which the physician or the surgeon ascertains the nature of those complaints.

Common sense teaches us that an accurate diagnosis is the first step to the adoption of a rational plan of treatment; and I may add, that an exact diagnosis ought to include the determination of the exciting cause of the malady. If, for, instance, we ascertain that a disease is one of irritation, it will help us greatly in the adoption of a plan of treatment, if we can likewise find out the nature of the irritant. In some cases, indeed, the sole object of the physician is to find out the exciting cause of a certain train of morbid phenomena.

For example—a child was brought into the hospital, not many days ago, labouring under convulsions, which manifested themselves in violent spasmodic twitchings of the extremities and face. Now this affection may arise from various causes;

but experience tells us that one of the most frequent is the presence of undigested matter in the bowels, exciting irritation there, which, through the visceral nerves, is communicated to the cerebro-spinal centre. The history of this case evidently pointed to this cause, and the treatment was directed accordingly. Free purging brought away from the bowels *a large quantity of plumstones*, and other undigested matter, and the little patient very quickly got well.

Several months ago, a man was brought in suffering from constant vomiting and hiccup; he had had no passage from his bowels; his voice was weak and stridulous. Before I saw him he was leeched on the abdomen, and purgatives were administered, which, however, he speedily vomited. On my arrival, some hours after his admission, I found that this treatment had been unavailing. Knowing that these symptoms are often the result of obstruction to the bowels by an incarcerated hernia, I instantly examined the inguinal rings, and found a hernia in one of them, which I had no difficulty in reducing. The man speedily recovered; but the delay of twenty-four hours more, without the removal of the cause of his symptoms, might have been fatal to him.

A patient came into the hospital during the summer, on account of a train of dyspeptic symptoms, under which he had been labouring for a considerable time, and for which he had submitted to various modes of treatment. At first we could make nothing of him: at length the examination of the urine threw light on the cause of his sufferings, and suggested the use of mineral acids; in a week our patient was cured of all his complaints.

These are common cases, and of frequent occurrence; and the inability to recognise their true nature, must have been fatal or most injurious to the poor patients, and highly discreditable to the practitioner.

It sometimes happens that, in two cases, nearly the same train of symptoms is produced by very dissimilar causes, and that a diametrically opposite mode of treatment must be pursued in each. In the expressive language of Dr. Gooch, "Two patients complain occasionally of dimness of sight, swimming of the head, singing in the ears, and observe, that if they turn the

head on one side to look at an object, they feel as if they should fall; but the one is plump, florid, and has a full pulse; the other is pale and thin, has cold hands and feet, and a pulse small and feeble. One practitioner bleeds them both; the other bleeds the one, but does all he can to give blood to the other. The latter cures both his patients; the former cures the one, but ruins the health of the other: but such is the nature of the human mind, that the cases *for* a preconceived opinion are retained easier than those *against* it. He remembers his good deed, forgets the other, or calls the case 'anomalous,' and marches on, without the slightest doubt that bleeding is the universal and sovereign remedy for dimness of sight, swimming of the head, and singing in the ears, save and except only in 'anomalous' cases."

Dr. Gooch relates two cases in point: in one, an erroneous diagnosis led to a fatal result; in the other, the true nature of the malady was detected just in time to adopt the line of treatment, which alone could prove successful.

The diagnosis of any disease necessarily involves a careful analysis of its phenomena, or symptoms; and it is obvious that to enable us to judge what functions or processes are astray, we must have at least some knowledge of them in the state of health. How then do we proceed to make such an analysis as I refer to?

It would be worse than useless to lay down any uniform rule for your guidance in questioning patients, or in analysing their symptoms; you will soon learn that a different plan must be followed in almost every case, and that the leading features of it will afford the best landmarks to direct you to the less prominent ones. It must, then, be your first object to seize upon the more obvious and conspicuous phenomena which each case presents.

There are certain questions which must be answered in every case, as bearing upon both the diagnosis and the treatment. The age of the patient is one of the most important. Age materially modifies disease; and certain maladies, very common at one period of life, are rare, or do not occur at all, at another. Malignant disease seldom occurs before the age of twenty-five, and tubercular phthisis is rarely met with after that of sixty.

You must always inquire into the habits of your patients. Intemperance, the fruitful source of disease, is often a great hindrance to the successful application of remedies, and, even although discontinued for the time, it exerts a weakening influence on the system. You cannot deplete the intemperate man, nor employ the same antiphlogistic means with him, as you would with the temperate.

The usual employment or trade of the patient must be ascertained. It is well known how often disease results from the nature of a man's occupation. The sedentary man is a prey to dyspepsia; the man of easy circumstances, good living, and little activity, is prone to gout; he who is daily exposed to the vicissitudes of weather, suffers from rheumatism; the ill-nourished and badly clad, who are, at the same time, the victims of intemperance, suffer from brain disorders, or from dropsy, consequent on chronic renal, hepatic, or cardiac disease. The painter, and all who work with lead, labour under the effects of a slow poison, which, however, does its work with sufficient speed, if the patient be of intemperate habits.

It is frequently of importance to ascertain the nature of the diseases to which the patient's blood-relations, who preceded him, were subject. The doctrine of hereditary disease is well established; and the more we know of the real nature of many of the processes of the animal economy, the more reasonable does this doctrine appear to be. In gout and scrofula it has been long admitted; in phthisis, so certain is it, that, in a doubtful case, the fact of the occurrence of the disease in a father or mother is a formidable one for the diagnosis; in asthma, too, it can scarcely be doubted that a morbid tendency may be handed down to posterity; and the unquestionable transmissibility of insanity from father to son, shows that the *mens sana in corpore sano* is to be desired by the former, not only for his own sake, but also for that of his offspring.

These points having been inquired into, your next object must be to ascertain the main cause of complaint which has induced your patient to apply for medical assistance. This leading question will soon open a door for others, from the replies to which, in the great majority of cases, you will be able to obtain such information as must direct you to the function

or functions, which are principally involved. Do you discover that the function of respiration is affected, you investigate the nature of the disorder by every means which the present state of knowledge places within your reach; and here let me remark, that all-important as is the employment of the stethoscope to your diagnosis, the careful observation of other signs and symptoms is of the utmost value to the treatment. Not the least valuable of these is the expectoration: how often will it lead us to detect latent disease in fever! the viscid, adherent mucus, scanty in quantity, sometimes pale, not unfrequently rust-coloured, ought always to make us explore for bronchial or pneumonic inflammation. In phthisis, although we pretend not to cure, we may relieve suffering, and retard the progress of disease. The investigation of the sputa in this disease frequently suggests measures of great benefit to the patient. Two patients now in the hospital afford an illustration of this remark: the one, a man in Fisk ward, had on his admission a very violent cough, with rust-coloured sputa, which I considered to result from pneumonia excited around tubercles. Local bleeding and small doses of tartarized antimony speedily subdued the irritation in the lung, and he has since been comparatively easy, although the ulcerative process has advanced. The other, a girl, in Augusta ward, with evident marks of tubercular deposit in the apex of the left lung, complained greatly of violent cough. The sputa were viscid and adherent, and appeared to come from an inflamed or irritated bronchial surface. Leeches applied beneath the clavicle gave marked relief.

I may refer you also to the boy with bronchitis in Rose, and the man with asthma in Sutherland ward; in both the sputa gave useful hints for treatment. In the boy the breathing was accompanied with both rhonchus and sibilus, and the expectorated matter was thick, viscid, and adherent; he was much benefited by antimony, and is now nearly cured. In the man, although his breathing was much more distressing, his sputa were thin, watery, frothy, and he was benefited by a stimulant and antispasmodic treatment. The æthereal tincture of lobelia inflata appeared to afford him much relief.

In heart disease, although we dare not speak with certainty without the aid of auscultation, a tolerably accurate diagnosis

may nevertheless be made, from the careful investigation of symptoms. Thus, a peculiar character of pulse will often at once indicate imperfection of the aortic valves; a violent impulse of the heart against the chest, and its apex striking below its natural position, will denote that the organ labours to overcome an obstacle, and has acquired an increase of size; engorgement of the veins and regurgitation in those of the neck, with enlarged liver, and more or less of dropsy, are evidence of dilated cavities of the right side. I strongly advise you to accustom yourselves to attend closely to the symptoms in these cases, as affording many useful hints respecting the treatment and progress of the disease.

Long before the stethoscope came into vogue, a friend of mine who has favoured me with his presence here to-day (Mr. Adams, of Dublin), distinguished with great accuracy many cases of diseased heart, by the symptoms only, and he has written a paper on this subject in the 'Dublin Hospital Reports,' which would do him credit even in the present improved state of our knowledge of cardiac pathology.

When the motor or sensitive powers are impaired, when the speech is injured, and the mind more or less affected, then we look to the central organ of animal life and consciousness, the brain, as the seat of disease; and here the most success in diagnosis will be obtained by him who keeps best before him the functions of that organ: and when it is considered how complicated are the functions of this wonderful organ, how intricate its structure, it need not excite surprise that the diagnosis of disease in it should be attended with very great difficulties.

The brain is the organ of the mind; that is, it is that part upon which mental changes immediately act, and through which mind and body are connected, and derangement of either will affect the other; a mind diseased will produce disorder of the brain, and organic derangement of the brain will produce mental disorder. You must go to lunatic asylums to see the former class of cases; but here we meet with many instances in which the mental aberration followed the organic disturbance.

And it is not difficult to account for all this; the convolutions of the brain are those parts which are immediately concerned in

the development of mental phenomena, and the gray matter of these convolutions is that portion of them which is intimately connected with the action of the mind. This is a highly vascular substance, and one of extreme delicacy, with very little physical cohesion. Every time I dissect the brain, I am astonished how a mechanism of such exquisite delicacy so often escapes disease; and I am prepared to find that changes apparently trivial in it, produce effects on function of a serious kind. The pia mater is a membrane composed of a very delicate areolar texture, supporting numberless ramifications of blood-vessels, from which still finer and still more numerous ones penetrate the gray substance. This membrane covers every convolution, and therefore spreads over an immense extent of surface, far greater than that of the arachnoid, for it sinks into the furrows between the convolutions.

The mind acts upon the brain; continued mental effort causes a determination to the gray matter: this abundant flow of blood cannot fail, if long continued or frequently repeated, to derange the nutrition of this matter, to weaken its cohesion, and to impair that delicate mechanical process, whatever it be, with which consciousness and mental emotions are associated. On the other hand, any physical cause affecting the blood itself, or the blood-vessels, or any of the adjacent textures, may give rise to a similar disorder of nutrition in the gray matter as the primary affection, and the mental disturbance will follow.

Whether the explanation I have given be correct or not, certain it is that when the mind is disturbed in consequence of brain disease, the gray matter of the convolutions is organically affected; and this explains what at first appears an anomaly, namely, that inflammatory affections of the membranes of the brain produce more serious disturbance of the mental functions than a deep-seated disease of the brain itself. One man may have an apoplectic clot in the centre of the hemisphere, impeding the voluntary power of nearly one half of his body, and yet his mind will be clear and his intellect vigorous. Another may have merely an inflammatory affection of a small portion of the pia mater, and be affected with violent delirium.

Again, the brain is the organ of volition, and also of sen-

sation; and both these functions suffer to a greater or less extent when that organ is the seat of disease. Hence, when you are investigating the nature of any cerebral disorder, you must inquire minutely into the condition of these functions. But remember that the destruction of motion or of sensation in any part, does not necessarily imply central disorganization; the brain is extended into the various parts of the body by its nerves; and it is most probable that there is scarcely a part in the body which does not receive nerves which are continuous, or connected with the cerebral fibres. The local derangement of one of these nerves may produce the same effect, but probably to a less degree, as disease of that precise part of the brain with which the nerve is connected.

Now, of all the points to which I referred in connection with brain disease, we have at present, or have lately had, examples in the hospital. In one case, that of Shoesmith, in Sutherland ward, you have an instance of injury to the motor power, resulting, as the history of the case indicates, from a sudden effusion of blood into some part of the hemisphere; the clot is deep-seated, and affects only the white matter, by compression, or actual solution of continuity of the fibres, to such an extent as to intercept the influence of the will upon one half of the body. At an early period of the case we observed the interesting fact, that although volition exercised no control over the lower extremity, the influence of the spinal cord was unimpaired; for, while the patient was utterly unable, however great his effort, to move the limb, the slightest touch to the sole of his foot caused the whole leg and thigh to move freely and extensively. But no such phenomenon was manifested in the upper extremity.

In a second case, motion is slightly impaired; and such is the nature of the disease that the motor power is also aggravated at particular periods. This man (Callender, in Sutherland ward) came in with imperfect paralysis of the *left* upper extremity; he has also had five epileptic fits, four before, and one after his admission. In the last fit it was observed that the muscles of the left arm and forearm were convulsively twitched prior to the invasion of the fit, and were so affected during the continuance of it. There has been also in this case some

impairment of memory. The disease here is probably superficial, and irritant in its kind, capable of depressing the volition power; and, at times, under the influence of a cause which we cannot detect, of exciting the nervous power, so as to throw the muscles into convulsive action. The disease, as in all such cases, is situate on the opposite side of the brain, and the patient has greatly benefited under mercurial treatment, and local counter-irritation over the *right* parietal bone.

In a third case, motion is impaired, but not as the result of disease in the centre; here the cause is peripheral. The patient, Grogan, in Sutherland ward, is a silk dyer, and while at his work was in the habit of sitting in a draught of cold wind, which blew upon his left side; the whole of that side has been benumbed, and the patient has the appearance of a hemiplegic. There is no dragging of the leg as in paralysis from diseased brain; the muscular power is considerable, but the patient is unable to adjust the actions of his muscles to the finer movements of the hand. The left side of the face looks stiff, and the cheek hangs, and expression is almost entirely absent from it. There is no pain in the head; no affection of the organs of special sense; no vertigo; and the affection has come on gradually, and seems due to a cause very likely to produce such an effect, namely, the influence of cold upon the muscular nerves of the exposed side. It is probable, too, that the nerves are not alone affected, but that the muscles have also suffered in their nutrition from a similar cause. The patient is evidently deriving benefit from a local treatment, and tonics.

A not uncommon example of this peripheral form of palsy, is that of the portio dura, resulting from cold, a case of which has only lately left the hospital cured. In such cases, no doubt, the low temperature impairs the nutrition of the nerve, and its physical structure remains altered, until time admits of the restoration of the nutritive process.

A fourth case in Augusta ward, Felicie Buhler, affords an interesting example of an affection involving a single nerve, and the textures associated with it. In this woman, as you may now see her in the ward, the power of elevating the right upper lid is destroyed; she has that form of paralysis named ptosis; and there is likewise paralysis of all the recti muscles of the

eyeball, except the external one, so that she squints outwards, and of the iris, so that the pupil was permanently dilated. She has at present no other ailment, and she seems to recover gradually the power over the levator palpebræ muscle.

But at the time she was admitted, the local palsy was complicated with considerable general cerebral disturbance, for she was almost speechless, partially comatose, and after admission speedily fell into apparently profound coma attended with some stertor, from which she did not recover for many days.

The disease came on gradually as she was travelling on the Continent with a family in which she served as nurse. There was no distinct evidence of paralysis of any other muscles. A good deal of restlessness, evinced by tossing in the bed, and frequently putting her hand to her head, seemed to show that the other symptoms were accompanied with pain, to which she was not altogether insensible.

This case I considered to be an inflammatory affection of the third nerve just at its emergence from the crus cerebri, destroying the motor power of that nerve, and causing some increased effusion at the base of the brain, which, by its pressure, gave rise to the comatose symptoms, and the loss of the power of speech.

That this was, in all probability, the correct interpretation of the symptoms under which this woman laboured, may be concluded not only from the reasonableness of it, but likewise from the very favorable result of the treatment adopted. She was leeched on the right temple, and brought as quickly as possible under the influence of mercury. Counter-irritation was employed, but nothing seemed to take effect so quickly as an issue in the scalp along the line of the sagittal suture. The day after the application of this remedy she seemed to awake from her comatose condition, and has progressively recovered.

I have briefly narrated these cases to show you that, although the diagnosis of brain disease is surrounded by many difficulties, a careful analysis of the symptoms, founded upon a correct appreciation of the anatomy and physiology of the nervous system, may contribute largely to remove the obscurity which hangs about the phenomena of these affections.

Time would fail me were I to say much upon the diagnosis

of abdominal disease. Here we lose, in a great degree, the valuable assistance which auscultation affords us in chest affections; but percussion and palpation, or the manual examination of the viscera through the abdominal parietes, afford us opportunities of judging of the seat of disease, which we do not possess for head affections. The same attention to the natural functions is necessary, as I insisted on for the latter class of diseases, in the analysis of the morbid phenomena; and in these cases we often derive great assistance from attending to the chemistry of the body, by examining the excretions, the urine, the bile; indeed, without the aid of chemistry we cannot, in many instances, pretend to form a correct diagnosis.

In some instances it becomes a matter of nicety, and often of great importance, to determine truly distinguishing features, when a similar train of symptoms belongs to two different classes of disease; this is, to make the *differential diagnosis*, as it has been called; as, to determine between pleurodynia and pleurisy; between empyema and hydrothorax; between rheumatism and gout, and so on; and here it will often greatly simplify our task, if we can lay hold on some prominent symptom, specially characteristic of one or other malady, or, in technical language, some *pathognomonic* sign; as the existence of the chalky deposit is pathognomonic of gout, or of sugar in the urine of diabetes.

To show you what may be done in the diagnosis of extensive disease, and as an encouragement to you to exercise yourselves in it in the wards, I will briefly recount to you the particulars of the case of dropsy, the post-mortem examination of which you witnessed a few days ago.

The patient, a young man, about thirty years of age, had been dropsical for several weeks before his admission, which took place in August. He was not a temperate man. A year ago he suddenly lost his speech, and had other symptoms of a head affection, which he could not clearly detail. He recovered from this attack, and remained well until the dropsy made its appearance.

We found him in very general dropsy; the face, abdomen, scrotum, and lower extremities all swollen; the last two to a very great extent. In investigating the cause, the liver was

first examined, for he had a sallowness of colour which excited suspicion of the unsoundness of that organ. But nothing abnormal could be found there. We next examined the urine; it was scanty in quantity, of a dark smoky-brown colour, of low specific gravity, and contained albumen in great abundance; and, in auscultating the heart, we perceived a systolic bellows-sound in the course of the aorta, with a feeble diastolic sound.

The symptoms resisted all treatment; the dropsy never gave way. A week before his death diffuse inflammation of the cellular tissue of his right leg and thigh set in, preceded by rigor and other constitutional symptoms; the urine for a day stopped completely, but flowed again. Two days before his death he had an epileptic fit, in which his right side was chiefly convulsed; the abdomen became very painful on pressure, swollen and tympanitic, and he was affected with severe spasmodic singultus. Under these symptoms, which seemed referable to the circulation of a poisonous agent, urea, in the blood, he sank.

Before we opened the body, I stated to you that you would find in the right leg cellular, and, perhaps, venous inflammation; in the abdomen, recent peritonitis, chiefly affecting that portion of the peritoneum which covered the diaphragm; the kidneys in the second stage of granular degeneration, and in a state of great vascular congestion; in the chest, obstructive and slightly regurgitant disease of the aortic valves. I was unable to express an opinion as to the disease in the head, but I said that it was not improbable we should meet with marks of old disease.

And you remember how true the prognostication was, and what a variety and extent of diseased action was exhibited in the body of this poor patient. All the organs were found diseased, as the symptoms during life had led me to expect, and the brain showed marks of an old apoplectic cyst existing in the fissure of Sylvius.

In conclusion, let me exhort you not to content yourselves solely with making the diagnosis of the diseases you witness here, but to watch the progress of each case; to look out for every new symptom; to compare the cases one with another. It has been truly said that "all observation is suggested by com-

parison," and experience is founded in a well-directed and extended observation.

A large proportion of our therapeutics rests upon the result of experience; and the practical tact which each individual acquires in the application of remedies must be derived from the same source. You cannot, therefore, begin too soon to acquire those habits of observation, and that experience, upon which your success as practitioners must, in a great degree, depend.

LECTURE III.

ON CERTAIN ACUTE DISEASES.

RHEUMATIC FEVER.

Gentlemen,—The appropriate treatment of Rheumatic Fever is still, in some degree, a *vexata quæstio*. I propose, therefore, in this and one or two other lectures, to call your attention more especially to this subject; and, by way of introduction, I shall make some remarks on the clinical history and pathology of this interesting form of acute disease.

Case I (vol. xxiii, p. 184).—The case by reference to which I shall particularly illustrate my observations is that of Elizabeth Stocking, aged 23 years, admitted on the 19th of April, —a case in which the prominent characteristics of the disease are very well marked, and which, therefore, may be properly selected as a good example of the malady.

Let me take this opportunity of recommending you to study with care, by taking full and daily notes of them, a few cases of this disease. It is a disease which, by and by, you will be frequently called upon to treat. We are seldom without several examples of rheumatic fever in the hospital; and, by taking careful notes of some eight or ten of these cases now, you will so impress upon your minds the history and symptoms of the disease, that you will be well prepared to treat them for yourselves, and each new case will be the more profitable to your practical knowledge. This is the more to be recommended, because rheumatic fever exhibits remarkably little variety of symptoms, or difference of phase. In one case the symptoms may be more severe than in another; but the same essentials which characterise the disease are present in all. Therefore, I

say, study a few cases carefully, and you will get a good knowledge of this disease before you are called upon to treat it on your own responsibility.

The case of Elizabeth Stocking affords, as I have said, a good opportunity of studying the characteristic symptoms of rheumatic fever. The two most prominent features are—First, a special fever, of the continued kind, varying in intensity in the different cases, but always maintaining the same essential characters. This fever is the essence of the malady—the nucleus, as it were, around which all the other symptoms are grouped. Secondly, a peculiar affection of the joints, involving more or less swelling of them, and also pain, which is aggravated by motion.

The fever may exist without the affection of the joints, and it may be accompanied even by an internal inflammation, such as pleurisy, or pericarditis, or endocarditis, as I have witnessed in several examples. But the articular affection never exists without the fever. You will, therefore, not regard the fever as merely symptomatic of a peculiar morbid state of joints; it is, truth, a fever *sui generis*, of which the articular affection and the other phenomena are but clinical features—attendant symptoms, which may or may not occupy a prominent position. But as the articular affection is very commonly present, and must necessarily demand much of your attention, since it gives rise to much of the patient's suffering, I will assign the first place to the few remarks I have to offer respecting it.

The articular affection almost always commences in the lower joints, and then travels up to the higher; thus it is first found in the ankles and knees, and then it goes to the elbows and wrists. The hip escapes more frequently than any large joint; the shoulder is much more commonly implicated than the hip. When the hip is severely attacked the patient suffers much; the other joints have the affection comparatively mildly; and in several instances it has seemed to me as if the whole force of the rheumatic inflammation had spent itself upon one hip-joint.

The implication of the joints is almost always shown by what may be considered its peculiar characteristic—swelling. Almost invariably there is an increase of the synovial secretion, sometimes to a very great extent, so as to prove a source of great

annoyance to the patient. The synovial membranes in this condition are highly vascular; so much so, that I have sometimes seen them, in cases where I have had an opportunity of examining them, as red as the conjunctiva when in a state of violent inflammation.

Another characteristic of the disease is its tendency to shift its position. To-day it will be in the *right* knee, which will be swollen, hot, and tender; to-morrow all this will have disappeared, and you will have the same symptoms in the *left*. This erratic tendency—this disposition to wander from joint to joint—is a symptom which you should carefully keep in mind; where it exists in a very marked degree it must be considered a bad feature, indicative of a low form of the disease, and a low state of the vital powers; and it is to cases in which this symptom is prominent that depressing treatment is found to be particularly prejudicial, often aggravating the disease generally, and this feature of it in particular.

This erratic tendency is present, not only in rheumatic fever, but likewise in the analogous disease of gout. It was this disposition to shift from one place to another that led the old writers to regard the internal inflammations, which are apt to come on in the course of these diseases, as "metastatic"—an idea which, however it may have some degree of support in gout, is inadmissible in rheumatic fever. It by no means follows that an inflammation of an internal part should be a metastasis of an external inflammation, even should the latter diminish or cease on the appearance of the former. A strong objection to the doctrine of metastasis is founded on the fact that internal and external inflammations often manifest themselves simultaneously, and very frequently the internal inflammation comes first. Moreover, it rarely happens that the external inflammation becomes diminished or exacerbated by the increase or diminution of the internal, and the converse.

Another feature of this disease is the profuse sweating by which it is accompanied. This is a special phenomenon of the fever. It is not distinctly of a critical or sanitary nature, as we sometimes see it in other fevers; for the sweats do not produce any marked immediate good effect, either on the joints which are implicated in the disease, or on the general

state of the patient. In Stocking's case the sweating was profuse: you doubtless recollect how it poured forth from the patient's head and chest, and, indeed, from the surface of her body universally; and from that you may judge how much fluid must have escaped through the channel of the sudatory apparatus. I must say, however, that I do not regard these sweats as otherwise than salutary within certain limits; I think that, in the early days of the fever, they should be encouraged as an important medium for the elimination of noxious matter from the system, and that you ought to be cautious how you stop such sweats, except where they are distinctly debilitating to the patient. Large quantities of free acid are carried off by these sweats: you remember that on several occasions we applied litmus to the skin of this patient, and that it was always deeply reddened. In contrast with this extraordinary action of the skin, we remark generally, as in the case of our patient Stocking, a deficiency in the quantity of the urine, and an alteration in its quality; that fluid being loaded with urea, urates, and often oxalates. Sometimes, as in a patient now in the hospital, it contains blood. The kidneys are in some degree irritated; less water passes off by them, but apparently a large amount of solid ingredients.

Another symptom, which always accompanies this disease to a greater or less extent, is a peculiar furred condition of the tongue. This is very striking, and will be readily recognised by an experienced eye as characteristic of rheumatic fever. A thick, white, velvety fur covers the tongue, and it is not until the fever gives way that this fur begins to pass off. The state of the tongue is the best index to the true condition of the patient; so long as it continues furred you cannot say that you have succeeded in thoroughly eradicating the rheumatic state; and I would warn you not to be confident in the result of your treatment unless you see that the tongue has become quite clean. Even although the pain in the joints and other external signs may have been subdued, yet, if the tongue remain furred, I should greatly fear that the patient may suffer a relapse, or that he may linger on in the rheumatic state for a considerable time.

Further, our case afforded an illustration of the way in which

rheumatic inflammation affects the heart. About the thirteenth day of the disease, a rubbing sound was heard over the base of that organ, leading to the conclusion that there had been an effusion of lymph on the opposed surfaces of the pericardium. This was evidently not metastatic—in other words, there was no direct transference of the inflammation from the external parts to the heart—because it coexisted with an undiminished, or but slightly diminished, inflammation of the joints. All these symptoms—namely, the articular swellings, the profuse sweats, the high-coloured and loaded urine, the furred tongue, the tendency to heart affection—are present in all cases of rheumatic fever; nor can we regard a case as of this nature in which these symptoms do not exist. In too many the heart affection actually comes on; sometimes it ushers in the attack, and takes precedence of the articular affection; in all cases it is to be apprehended, and, if possible, guarded against.

There are, however, two points in the case before us which are peculiar, or, at least, which do not occur constantly in cases of rheumatic fever. First, you will remember that at several of our visits I pointed out to you on the skin of this patient, a copious eruption of what have been called *sudamina* or *miliary vesicles:* they were scattered all over the surface of the thorax, and if you passed your finger over the skin, you found it rough. These sudamina are seen, on close examination, to be minute vesicles filled with pellucid fluid. They do not especially belong to rheumatic fever, but they are characteristic of a sweating state. If a patient, suffering under typhus fever, pneumonia, phthisis, &c., sweats profusely, these sudamina are apt to appear upon the skin in great numbers. The presence of the vesicles must not be regarded as indicative of any special form of disease, but merely as an accompaniment of a state of very free perspiration.

The second peculiarity in the case of this woman is, that the rheumatic fever followed quickly upon the puerperal state. The connection between rheumatic fever and deranged uterine secretions is very remarkable. Some of the most severe cases I have ever seen have followed dysmenorrhœa. It would seem as if, in these cases, the uterus were but imperfectly evacuated, and its contents becoming decomposed, and getting into the circu-

lation, produced a morbid state of the blood, which gives rise to the symptoms under which the patient labours, and requires for its cure the elimination of the unhealthy material by the various emunctories—a state similar and analogous to pyæmia.

Not unfrequently, after the puerperal state, the patient exhibits all the symptoms of ordinary rheumatic fever: the same profuse sweats, the swollen joints, the fever, the highly concentrated urine. But in some cases the disease runs a more formidable course; the joints, instead of getting better after a time, continue to get worse, till at last the cartilages ulcerate, pus is secreted in large quantities, and fills the synovial membranes to distension; the articular extremities of the bones are laid bare, and the rough osseous surfaces grate against each other when the limb is moved. I have seen all the large joints in this condition. At the same time deposits of pus form in the muscles, and in other parts, even in the eyes. Some of the French writers describe this disease under the name of "*puerperal acute rheumatism.*" It is, in fact, a form of puerperal fever, due to inflammation of some of the uterine veins; this gives rise to the formation of pus, which, infecting the blood, excites articular and other inflammations in its passage through the circulation. Such cases throw light on the pathology of rheumatic fever, and show how a morbid matter, generated at one part of the circulation and carried throughout it, may occasion serious disturbance in the local nutrition of the various parts through which it may be undergoing elimination, and give rise to a train of symptoms, closely resembling, and not to be distinguished (save by the history) from those of rheumatic fever.

In our patient Stocking, there was some morbid state of the uterus prior to the development of the rheumatic condition. Immediately after her confinement she seems to have had symptoms of peritonitis, which appeared to yield to treatment; but she had not long recovered from these symptoms when the rheumatic condition showed itself.

I think it will be as well, before proceeding to the treatment, to adduce some other cases for the further illustration of the preceding remarks, as well as for after reference.

Case II (vol. xxxvi, p. 227).—The second case is that of Sarah Green, a girl in her sixteenth year, who came under my care, in the hospital, in January, 1853. This case affords an example of the more severe form of cardiac complication, and is also instructive as regards the treatment. Five days before her admission on the 8th of January, she was taken ill with pain and swelling of both knees; she was, therefore, placed under medical treatment, and, among other things, took gray powder *until slight ptyalism was produced.* While in this condition, *with her gums still sore,* during the night immediately preceding her admission to the hospital, she suffered from uneasiness about the chest, with some cough; there was also a considerable discharge of blood from her throat or nose, to which she has been subject.

On admission, she was suffering from pain in both knees, both shoulders, and ankles; her pulse numbered 110, and the respirations 36 in a minute; a slight sound, like a bellows-sound, accompanied the systole, and was heard best over the base of the heart. A mustard poultice was applied to the chest, and this was followed by a blister. She commenced taking one grain of opium every three hours, and large sweating doses (six drachms) of the liquor ammoniæ acetatis, with camphor mixture.

The next day, on examining her chest, I detected a loud, harsh, to-and-fro, pericardial rubbing sound; this was audible all over the region of the heart. On the 10th we have the following note:—"She complains of much pain in her elbows, knees, and ankles; pulse 106, full and strong; tongue very red where not coated with a whitish-yellow fur; she sweats profusely, sleeps little, and looks pale and anæmic. Her pupils are but slightly affected by the opium." The blister was then dressed with Unguentum Sabinæ. On the 11th the pains in the joints, especially in the left knee and right elbow, were aggravated; the sweating had abated, and her skin was hot and dry; the tongue dry and red. The to-and-fro rubbing sound was very loud and harsh, and heard very distinctly in the course of the left subclavian artery, and underneath the left clavicle, as if the pleura over the pericardium were involved; the rubbing, however, was not synchronous with respiration. A blister was

again applied to the chest, over the situation of the rubbing sound.

I would just remark, with reference to this case, that you find here all the phenomena of rheumatic fever; and, in addition, the remarkable fact that a severe internal inflammation, involving the pericardium, and likewise, probably, the endocardium and the pleura, supervened *whilst the patient was in a state of salivation from the early administration of mercury.*

On the 12th we found our patient suffering rather less pain; she had also been refreshed with some sound sleep. From that day until the 24th she continued steadily improving: the pain in her joints diminished, and then ceased altogether; the pulse and respirations became less frequent, and the sweating diminished. The pericardial friction sound continued to retain its to-and-fro character, but shifted towards the apex of the heart, becoming less extensively heard and softening down. A pleuritic friction sound was heard, for one day only, the 15th, over about the sixth rib on the left side.

On the 24th she experienced a return of pain in the left side of the chest, and in the corresponding shoulder. On the 26th we found her not nearly so well—her tongue was dry and furred; the pulse was feeble and had risen to 140; she was restless and could not sleep. On examining her chest, we found extensive dulness on percussion in the pericardial region; the heart sounds were distant and indistinct; the to-and-fro sound had ceased, and in its place we heard a distant systolic murmur; a pleuritic rubbing sound was detected also over the lower part of the right lung in front. These changes in the physical signs seemed to point, clearly enough, to a considerable effusion of fluid into the pericardium. The quantity of opium taken, which had been reduced on the 18th to half a grain three times a day, was again increased to a grain; a blister was also applied over her heart.

With the exception of the extension of the pleuritic rubbing over nearly the whole of the right lung in front, some oppression of breathing, and a change in the joints affected, there was no marked alteration in the condition of our patient or in the physical signs until the 29th: all oppression of breathing then ceased; there was no longer extensive dulness on percussion

over the heart; the former to-and-fro rubbing sound returned, loud and harsh, and was heard pretty extensively over all the front of the chest, but most marked towards the base of the heart and under the clavicles; the pleuritic rubbing was still heard on the right side. From these changes the re-absorption of the effused fluid was at once inferred.

By the 3d of February the general condition of our patient had much improved: both the pleuritic and pericardial friction sounds had disappeared, and the systolic bellows-sound, which had been masked by the to-and-fro rubbing, was again distinctly heard. On the 5th there was distinct evidence of a circumscribed patch of pleuritic inflammation on the left side. From this time, however, a rapid and steady improvement took place, and by the 10th the pulse had fallen to 70, and improved in tone and quality; all pain and abnormal sounds had ceased, excepting a mitral systolic murmur, which continued audible at the apex of the heart. There was, too, some return of colour in her lips and cheeks. This was after about five weeks' residence in the hospital.

The opium was now discontinued, and a tonic plan of treatment commenced, the patient taking a grain of quinine three times in the day, which was subsequently changed for three grains of the ammonio-citrate of iron.

A rapid convalescence, after so severe an illness, was hardly to be expected; our patient accordingly remained in the hospital for some weeks, and, although on the whole improving, had occasional accessions of pain and swelling in some of her joints.

I will now give the history of three other cases which I have selected as average examples of the course and duration of this disease under the plan of treatment which I now pursue; and with these I must bring this lecture to a conclusion.

Case III* (vol. xlv, p. 103).—Matthew Baldwin, aged 29 years, a labourer, accustomed to liberal potations of beer. His father, he says, suffered much from rheumatic gout, but his own health has been generally good.

* The record of this case was kept by my clinical clerk, Mr. Goodall.

For about four weeks previous to his admission on September 21st, 1854, he had been generally ailing, with feverishness, headache, disordered bowels, and loss of appetite. In the course of the last of those weeks there had been an accession of rheumatic pains in the ankle, knee, and hip of the left side, then of the right, with increasing severity. Finally, the day before admission, he was seized with pain in the right shoulder and præcordial region, with a sense of tightness and difficulty of breathing.

The first night after his admission, he slept little and perspired profusely. When examined, the following day, he appeared pale and ill, and was distressed by shortness of breath; his tongue was furred, his skin warm and moist; the right wrist was the only painful joint. The urine, which was highly acid, and of specific gravity 1035, contained much urate of ammonia. The pulse numbered 100, and the respirations 40. A distinct rubbing sound was heard all over the region of the heart.

As his bowels were confined, he was ordered some of the hospital white mixture, consisting of the sulphate and carbonate of magnesia, and this was followed by frequent doses of the usual alkaline mixture, containing fifteen grains of the bicarbonate and five of the nitrate of potash in each dose. A blister was also applied over the heart.

His breathing appeared almost immediately relieved after the application of the blister. On the third night he slept well; and on the following day, September 23d, he had almost lost the pain in his chest; the respirations were much easier, and had fallen to 27 in a minute, his pulse to 98.

On the 25th there was an increase in the number of joints affected, the right knee, ankle, and wrist being very painful; small blisters were therefore applied to them, and the blister to the chest was repeated. He was then sweating freely; his tongue was cleaner, bowels open, and appetite improved; the urine was clear, and had fallen in specific gravity to 1028; the pericardial friction sound still continued. The following day (26th) he was ordered two drachms of brandy every two hours, or six ounces daily.

There was no important alteration for some days. On the 2d of October the Decoctum Cinchonæ was substituted for water

in the alkaline mixture, and he was ordered five grains of the pil. saponis comp., with three of calomel, in pill every night. On the 3d, the blister to the chest was repeated. On the 4th, his back was observed to be covered with sudamina. On the 7th, being the seventeenth day from his admission, the following note was taken:—"He is now improving daily. Pulse 84, respirations 26; sleeps well, and does not sweat; the pain is confined to some of the muscles; a slight friction sound alone remains audible at the apex of the heart; tongue clean, appetite good, bowels open; he still continues the brandy."

After this he remained some weeks in the hospital, gaining strength, and improving much in health; there was a return of slight pain in the joints, especially the shoulders, for which iodine paint was applied. A tonic mixture of quinine and acid was substituted for the alkaline one.

Case IV* (vol. xxix, p. 188).—Johannah White, a servant girl, sixteen years of age, came under my care in the hospital on the 24th of January, 1850; for some months before her admission, she had not had her usual good health. On the night of the 22d she awoke with severe pain in her back; this was soon followed by pain in her knees and shoulders, gradually increasing in severity.

On the 25th, the day after her admission, she was suffering from great pain in the right shoulder, with pain and effusion into both knee-joints; her skin was hot and sweating, the perspiration had an acid reaction and smell; she had no appetite, was thirsty and sleepless; the tongue was thickly coated with a blankety white fur; pulse 120, respirations 36; urine very acid, and loaded with urates. She at once commenced taking the ordinary mixture of the bicarbonate and nitrate of potash, with five minims of the tincture of opium every four hours. A blister was also applied above the left knee.

The next day, January 26th, the joints were much the same, and, in addition, the right wrist had become extremely painful, and much swollen; she had slept badly; her pulse still numbered 120, and respirations 36; the urine exhibited the same characters.

* This case was recorded by my clinical clerk, Dr. Monckton.

On the 27th, the third day of the treatment, her pulse had fallen to 84, and the respirations to 30; she had slept well, her appetite had returned, and she was free from pain. The following day she had slight pain in the right shoulder, but she continued improving in other respects.

On the 31st of January, the tenth day of the attack, she was quite free from pain; her tongue was clean; pulse 72; respirations 20. There was no relapse, and she was soon after discharged, well.

Case V (vol. liv, p. 140).—Deborah Monssey, a servant girl, fourteen years of age; accustomed to much out-of-door work and exposure. About six days before her admission she was seized with rheumatic pains in her ankles; the knees were next affected; and, the following day, all the joints of her limbs.

On admission, January 31st, 1857, her face was flushed; skin hot, but not perspiring much; features constrained; tongue covered with a thin white fur; bowels confined; pulse 126, and respirations 36. The joints most affected were the shoulders and knees. The urine deposited a dense brick-coloured sediment. She complained of uneasiness, or slight pain in the præcordial region; on listening here we detected a distinct pericardial rubbing sound. Soon afterwards, a soft systolic bellows-sound, heard most distinctly over the base of the heart, was also observed. She was ordered to take two-drachm doses of the liq. ammon. acet. with three minims of tincture of opium every four hours. Six leeches were applied over the region of the heart, an alkaline wash to the lower limbs, and a blister above the most painful joints.

On the third day of the treatment, February 2d, the rubbing sound had almost ceased, the systolic bruit remaining distinct; she had sweated more freely during the night, and the limbs, on the whole, were easier. Her medicine was now changed for the mixture of the bicarbonate and nitrate of potash, and fifteen minims of the liquor morphiæ hydrochloratis were ordered to be taken at night. On the fifth day of the treatment, February 4th, the pain was almost confined to her wrists and hands; the tongue was cleaning in the centre; her countenance was natural; pulse 124; respirations 36.

She continued in the hospital for about a fortnight after this, improving, but suffering more or less from pains in the different joints, which shifted about and varied in intensity; she was also much troubled with nausea and vomiting. Her pulse steadily declined to 65; her tongue became quite clean; the urine clear and bright; and the bellows-sound softened down.

Before I enter upon the description of the treatment of the disease, let me come to some understanding with you as to its nature; for we cannot adopt a particular plan of treatment without having some theory of the nature of the malady. Now, what is the most reasonable view of the pathology of this disease? I have not time to enter into the discussion of this question with you as fully as I could wish; and I must, therefore, be content with simply recounting to you the articles of my own creed upon the subject.

Rheumatic fever, then, I would say, is a state of high febrile excitement, induced by the accumulation of a peculiar morbid product, or *materies morbi*, in the circulation; and the other symptoms which accompany it are merely caused by certain local derangements and disturbances produced at those points whence its elimination from the system is taking place. This materies morbi is the result of a vitiated state either of primary or secondary assimilation, or of both, and the parts where it accumulates are just those, which, while they are very vascular, and therefore contain a large quantity of the diseased material, present the least obstruction to its escape from the circulation. These are the delicate synovial membranes of the joints, and the almost identical structures, the serous membranes—the pericardium, endocardium, and pleura, the air-cells of the lung itself, and even the peritoneum—parts where the blood-vessels are naked, or covered by but a thin film of membrane. These membranes, being largely supplied with rheumatic blood, pour forth into their cavities an enormous amount of their ordinary secretion, contaminated with the diseased material. Thus the synovial membranes become distended with a morbid synovia, which, instead of being alkaline, as it is in health, has a reaction decidedly acid. Thus, likewise, the skin is covered with profuse sweats, which are due to the irritation established in the

sweat-glands by the morbid product; and the abundant fluid thus got rid of has, like the synovia, a marked acid reaction. The functions of the kidneys are, doubtless, similarly affected, and you get an abundance of urea and uric acid in the urine. But this morbid matter may escape likewise through the serous membranes, as it does from the synovial, from the lungs, or at the heart; and hence, in any of these situations it may excite inflammation, and in all of them is disposed to do so. It is evident that the more its elimination is encouraged and favoured at the skin, at the kidneys, at the joints, and at the mucous membrane of the alimentary canal, the less likely are the other important parts to suffer—the less chance have you of pleurisy, pneumonia, pericarditis, &c.

I have thus given you an outline, and illustrated the principal features, both essential and accidental, of rheumatic fever, and we have come to an understanding as to the pathology of the disease. I must defer to another lecture some account of the treatment which appears to me the most appropriate.

LECTURE IV.

ON CERTAIN ACUTE DISEASES.

RHEUMATIC FEVER.

Having, in my last lecture, described and illustrated the prominent points in the clinical history of rheumatic fever, I must now proceed to discuss that part of the subject upon which I wish to dwell particularly, and which, indeed, is the main object of these lectures, namely, the treatment of the disease. Upon this subject there still exists much difference of opinion among practitioners; and as I have myself, after much inquiry, come to some decided conclusions as to the line of practice which should be pursued in these cases, and as these conclusions are confirmed by my daily experience, I am anxious to bring the whole subject before you, and explain to you fully the principles which regulate my practice in the treatment of this formidable malady.

It is important that we should determine what are the particular objects to be kept in view in the treatment of diseases of an acute kind. They are these:

1st. To relieve pain.

2d. To strike at the root of the malady.

3d. To cure our patient with as little trial to his constitution as possible, so that afterwards he may not be in a worse condition than he was before. We often hear in society such expressions as these:—"I was always very well till Dr. So-and-so treated me for rheumatic fever, and he purged and bled me to such a degree, and treated me so violently, that my constitution could not stand it, and I have never been the man I was before." Now, let us endeavour to conquer this frightful malady, and let it be our boast that, when we have done so, we leave our patient a constitution unimpaired, at least by our remedies. In some

cases it is not possible to accomplish this; the lungs may become affected, or the pleura, or the pericardium, or the endocardium, and so much organic mischief may be done in a short time, as to leave important organs permanently damaged; still, notwithstanding these lesions, the general nutritive powers need not be materially impaired.

4th. A good plan of treatment should aim at securing for the patient a short convalescence. I do not speak of a speedy cure, because that is, to a certain extent, implied in a short convalescence. At the same time, I must caution you against the so-called rapid cures said to be effected by the heroic treatment of rheumatic fever. If these cures are rapid, they leave a tedious and painful convalescence; indeed, it may be more properly said in such cases, that the treatment converts an acute into a chronic disease, rather than that it cures the former. Such a cure, if cure it can be called, is not what you should aim at obtaining for your patients; nor is that a *bonâ fide* cure of rheumatic fever unless the febrile and constitutional symptoms are subdued, the secretions re-established in their normal quality and quantity, the tongue rendered clean, and the joints relieved of their swelling and pain: if such a cure as this can be effected in a short time, not entailing a tedious convalescence, your patients will have good reason to be satisfied. Now I must tell you that I do not believe that a *bonâ fide* cure and a short convalescence are, in the generality of cases, really obtainable by the heroic modes of treatment; and I would add my conviction that it is not desirable to shorten very much the period of cure in this disease, as it is not likely that a sufficient elimination of morbid matters can be effected in a very short time. These, so-called, rapid cures, are also apt to leave the patient very subject to relapse, which you should endeavour to guard against as much as against a tedious convalescence.

The most instructive way, as it seems to me, in which we can discuss the treatment of this disease, will be to enumerate the various methods which have been proposed for this purpose, and to point out the reasons for rejecting some and for adopting others. As many as seven different plans may be specified, of which I shall place last that which I am in the habit of following here, and which I call the *treatment by elimination*.

The first plan is that by venesection. It was formerly the prevailing opinion, and it is still, unfortunately, thought by some, that when called to a case of rheumatic fever one had only to open a vein, and if he could succeed in taking away a sufficient quantity of blood, which, in many instances, it was laid down should be little short of one or two pints, that by this large and rapid abstraction of blood, the disease may be cut short, and a malady, which ordinarily lasts some weeks, may be converted into one of a few days' duration. Frequently, not content with one large bleeding, those, who hold these views, will bleed a second, a third, or a fourth time, at short intervals, and in large quantities.

The chief advocate of this practice at the present day is Bouillaud, of Paris. Now, if you look through the record of cases, as given in his book, you will see that his patients, although some of the more urgent symptoms are apparently very quickly overcome, yet linger on in the hospital for a considerable period, suffering much from chronic rheumatism, and exhibiting an extreme anæmia, from which they but slowly, if ever, recover. This plan of treatment has been advocated by some English physicians, and among others by the celebrated Sydenham, who, however, in the latter part of his career, abandoned, or greatly moderated it; and, I am happy to say, the number of its supporters at present is very small. It is a practice from the adoption of which I would most earnestly dissuade you, as having the support neither of reason nor of experience, and as being fraught with the most dangerous consequences to your patients.

I could tell you of several cases in which a fatal result has been clearly produced by the adoption of this method of treatment, which, most probably, would have recovered completely had they been left alone, or treated by a milder method. One case in particular made a deep impression upon me. The subject of the case was a young and strong man, of great promise in his profession; he was seized with rheumatic fever, and one of the knee-joints was severely affected. On a previous occasion a similar attack seemed to yield readily to a very large bleeding, and the patient recovered. His medical attendant, naturally enough, determined on the second attack to adopt the same

treatment, which had seemed so successful before, and accordingly bled him very largely, and applied leeches to the inflamed joint. The result was violent delirium, and death by exhaustion in the course of about eight-and-forty hours.

The following case, treated some years ago in the hospital, will serve to show what venesection, and the loss of blood by other means, can do, as well as what it cannot do :

Case VI (vol. i, p. 260).—Charles Davis, a porter, twenty-eight years of age, was admitted into King's College Hospital on the 12th of October, 1840. He stated that about a week previously, after exposure to cold, he was seized with lumbago; that the pain afterwards left his back, and attacked his arms and legs.

On the day of admission (the 12th), the left knee and ankle and the right wrist were painful and somewhat swollen; his pulse numbered 90. Fourteen ounces of blood were immediately taken from the arm, and a purgative draught administered; the blood drawn was buffed and much cupped, and the clot large. That night he sweated freely, but the severe pain prevented sleep. The following day (13th) the pulse was rather less frequent, 80, and a white fur on the tongue, so usual in acute rheumatism, was noticed. He was ordered to take a quarter of a grain of the muriate of morphia twice in the day. On the third day he was suffering severe pain in the left hip, and along the back of the thigh; the sweating had continued; his pulse had risen to 100, was thumping, full, and compressible. The morphia was discontinued; he was ordered two grains of the sulphate of quina three times a day, and was cupped near the painful hip to twelve ounces. The pain diminished after the cupping.

When he had been eight days in the hospital he was nearly free from pain in the joints, but complained of a pain in the præcordial region, where an indistinct bellows-sound, following on the systole, was detected. He was cupped over the heart to six ounces. On the twelfth day he complained of a sense of weight in his chest, for which a blister was applied, apparently with benefit. But the next day there was a pericardial rubbing sound heard in addition to the bellows-sound; the pulse had risen to

96, and there was a return of pain in several joints, and a red blush over the left ankle. He was again bled from the arm to eight ounces; the quina was discontinued, and a pill, containing one grain of calomel and a fourth of a grain of opium, was ordered to be taken three times a day. For five or six days afterwards he continued suffering chiefly from wandering pains; the rubbing sound was persistent; the pulse about 96, full, and slightly thrilling. He had taken two colchicum draughts with morphia, in addition to the medicines above prescribed; but these were all discontinued on the eighteenth day of admission and the quina was resumed.

The last report of him was made when he had been ill about five weeks, and in the hospital about a month. He was then complaining of slight pain in the chest, and had a throbbing pulse numbering 80, but was otherwise improving.

This patient remained another fortnight in the hospital, and was then discharged; but had hardly been away a fortnight when he had a slight fit of shivering, soon followed by pain and stiffness of several joints, and considerable swelling of the feet and knees. He waited a fortnight and was then admitted, for the second time, to the hospital, December 23d, 1840, with all the symptoms of rheumatic fever. He was immediately bled from the arm to sixteen ounces, ordered to take a purgative draught at once, and an ounce and a half of guaiacum mixture three times a day. Besides suffering pain in almost all his joints, he complained, for many days, of a constant painful sense of weight and tightness in his chest, for which he was cupped to twelve ounces on the third day of admission. At first the heart sounds were normal, but, on the fourth day, a slight systolic bruit was heard, and there was occasional hiccough. He continued taking the guaiacum mixture for a week without any decided benefit; at the end of that time the left shoulder and wrist were still painful, and the pain in the chest continued. A grain dose of the acetous extract of colchicum every six hours was then substituted for the guaiacum. On the fourteenth day from his second admission, a blister was applied to the chest, as the pain there continued; and on the twenty-first day there was some increased articular affection; the left wrist especially was swollen and red. Six leeches were applied to the wrist, ten

grains of Dover's powder were ordered to be taken at night, and the colchicum to be omitted.

On the following or twenty-second day, there was great præcordial pain, preventing him from lying down, with hurried respiration, and a slight pericardial friction sound. He was cupped, over the heart, to seven ounces, and a pill containing three grains of calomel and half a grain of opium was ordered to be taken every three hours. After the cupping he was in less pain, and able to breathe more comfortably for two days, but his pulse continued up to 100, and the rubbing remained.

On the twenty-fifth day, there was a fresh accession of pain in the chest, with dyspnœa; he was therefore bled again to sixteen ounces; the blood was much buffed and cupped; the pain was less after the bleeding. The next day the pulse was 100, and the respirations 48, with considerable dyspnœa on any exertion; a distinct pericardial rubbing round was heard near the sternum, between the third and fourth ribs, and over the anterior surface of the heart; a bellows-sound was also distinctly audible at the apex. He was again bled to twelve ounces; the blood was not buffed.

During the two or three following days our patient continued suffering much pain in his chest as well as in some of the joints; the rubbing and bellows sounds continued; there was considerable sweating, and sudamina made their appearance on the neck and chest. As we might have expected, he was now very pale and feeble, with a quick pulse, sleeping badly, and suffering from palpitation on any exertion. After slight ptyalism the calomel was discontinued, and some Dover's powder given alone; three ounces of wine were also added to his diet, but soon changed for a pint of porter.

He continued very slowly improving for nearly three weeks, but at the end of that time, seven weeks from his admission, he had more pain again in his chest, with catching breathing, and a quick, throbbing pulse; a return of pain soon followed in the joints. A blister was applied to the chest, and ten leeches to the left knee and ankle. The pain then shifted to other joints; the right pleura became affected—there was pain in that side increased by deep inspiration, and a rough rubbing could be distinctly felt and heard; pain continued over the heart, and

there was hiccough. When the pleuritic symptoms appeared, leeches were applied to the right side, and also to one hand; the next day he was cupped to seven ounces, and a sixth of a grain of the muriate of morphia was ordered every six hours.

He remained more than five weeks longer in the hospital, improving very slowly: he continued to have pain with a sense of dragging in the right side, the creaking sound also remaining audible. The morphia was soon discontinued, and the quina resumed; occasional blisters were applied to the right side. He was at length discharged better, but not free from pain, on the 30th of March, having then been ill sixteen weeks, and an inmate of the hospital, the second time, for nearly fourteen.

The case, however, does not end here; he was discharged only to be re-admitted two months afterwards, having then been suffering for a month from a fresh attack of rheumatism. He was admitted on the 26th of May; the left elbow was then painful, and both ankles painful and swollen, with effusion into the sheaths of the neighbouring tendons. When he had been in the hospital a day or two, he had an attack of acute pain in the lower part of the chest, on the right side, with dulness on percussion. Ten leeches were applied, and he lost the pain, but continued to complain of a feeling of oppression, for which a blister was tried. He again got better, and left the hospital, but the date of his discharge is not recorded. During the two former periods of his residence in the hospital he had been bled altogether five times, and cupped five, so that 110 ounces of blood had been taken, besides what was lost by leeches! I shall content myself with remarking upon this case, simply, that it seems to me one well calculated to show that bleeding, mercury, and colchicum are not all-powerful for good in the treatment of rheumatic fever.

This subject is so important, that I shall offer no apology for bringing before you a second case treated on a similar plan.

Case VII (vol. i, p. 228).—Elizabeth Freethy, a servant, twenty-one years of age, was admitted to the hospital, September 9th, 1840, with rheumatic fever. She had then been ill three weeks, and attributed the commencement of the attack to having caught cold in a damp kitchen. She was first seized with

violent pain in her back; her limbs soon became affected, and two days afterwards she had nearly lost the use of them, but she did not take to her bed until about three days before her admission.

When admitted (9th) she was ordered to take a dose of the hospital white mixture, and ten grains of Dover's powder at night. On the following day (10th) the aperient mixture was repeated, and she was cupped in the loins, but the amount of blood taken was very small.

On September 11th she was bled from the arm to the amount of eight ounces, and the Dover's powder was continued. On the 12th the bleeding was repeated, and twelve ounces more of blood were taken; she was then ordered two grains of the sulphate of quina three times a day. On the 19th twelve ounces of blood were again taken from the arm, and on the following day the quina and Dover's powder were omitted, and a small dose of the muriate of morphia substituted.

On the 25th she complained of pain in the region of the heart, and a slight bellows-sound was heard. There was some effusion into both knee-joints; the pulse numbered 122; the tongue continued white and furred. Twelve leeches were applied to the chest over the heart. The next day she was perspiring profusely.

On the 27th, when she had been ill rather more than five weeks, and under treatment in the hospital nearly three, the following note was made:—"She chiefly complains of her knees, which are very painful, and full of fluid, the left one especially, the skin of which is slightly red on the inner side, and very painful, even when lightly touched; pulse 112; no sleep." Six leeches were applied to each knee. On the 28th there was no material change; a systolic bellows-sound continued audible; her pulse numbered 120; she still perspired profusely, and had not slept. Ten-grain doses of bicarbonate of potash, three times a day, in soda-water, were then ordered. On the following day there was some general amendment, and less pain, and by the 1st of October all feverish symptoms had subsided; her appetite was returning, and she could sleep better. The knees and ankles, however, continued painful, and somewhat swollen.

After this she made little progress; and on October 6th, when

the last report was made before her discharge, the knees, though not swollen, were still painful, and her pulse continued as high as 98. She had then been ill very nearly seven weeks, and had been rather less than a month in the hospital.

In this case, again, you have a striking example in which the disease was of long duration, despite of free and repeated bleeding.

I think it but right to state, in justice to those who have adopted this practice, that patients so treated sometimes do very well; and the next case that I shall quote will furnish an example of this. At the same time, I must caution you against allowing such a case to lead you to adopt a similar plan of treatment. That it might justly so influence you, it would be necessary to show that such favorable cases are not only as common, but more common, under an antiphlogistic plan of treatment, than when less violent remedies are used; and not only so, but that, among unfavorable cases also, those are most disastrous in which the abstraction of blood has been abstained from. Judging from no inconsiderable number of cases, treated on both plans, I am convinced that the opposite conditions obtain. I would go so far as to say that, even were we certain that venesection would produce the desired effect on the leading symptoms of the disease, we should yet hesitate ere we make use of a remedy which, in the *general* effect it may have, is often uncertain, and most perilous. In one case you may relieve your patient, in another you may send him to a premature grave; or, in the same individual, in a first attack, you may obtain complete relief by this method, and in a second attack you may place his life in jeopardy, or subject him to a tedious convalescence.

Case VIII (vol. i, p. 210).—Francis Barrett, a carpenter, twenty-six years of age, was admitted an in-patient of King's College Hospital, with rheumatic fever, on September 11th, 1840. The attack began the day before, with pain, redness, and swelling of the left foot and ankle, accompanied by feverish symptoms, great thirst, and heat of skin. The pain soon extended to the muscles of the calf.

When admitted, he was bled to twelve ounces, and purged. He gradually became worse, and by the 12th all his joints were

affected, and the pain severe. On the 14th he was still suffering severe pain in every joint, and also complained of some uneasiness about the heart; his pulse was observed to be intermittent, and occasionally there was a sort of double systole of the ventricle. He was then cupped, and sixteen ounces of blood taken; a mixture was prescribed, consisting of sulphate of quina, with one eighth of a grain of muriate of morphia in each dose. At night he perspired freely, and on the morning of the 15th he was in less pain. During that day, however, and the two following, there was no great improvement: the redness and swelling of the wrists remained; his tongue was coated with a white fur; there was profuse perspiration, and a high pulse of about 100.

On the 18th he was again bled from the arm to sixteen ounces, and the clot formed was much cupped and buffed. On the 19th the joints of the right upper extremity were still acutely painful, but his pulse had fallen to 88. On the 20th there was decided improvement, and on the 21st he was much easier: the swelling had disappeared from the right wrist, he had slept better, was sweating less, and his tongue was cleaning; pulse 68. The quina was repeated without the morphia.

He continued improving; and on the 24th, being the fifteenth day of the disease, he was free from pain, but weak; his pulse was 60, his appetite improving, and tongue clean. A chop and a pint of porter were now added to his diet, and we have no history of a relapse.

2. The second plan of treatment is that by moderate bleeding and diaphoretics. This may be called an "expectant" treatment; but it is more than that as regards the venesection; while in other respects it sufficiently merits the name. The advocate of such a plan will say,—"When I am called in to a case of rheumatic fever, I think it advisable to commence the treatment by abstracting about ten or twelve ounces of blood, and then to give sudorifics and purgatives."

Now, the objection which I entertain to such treatment is this—that the routine abstraction of blood can scarcely be called *necessary* in any case, and that in many it is injurious. The tendency of rheumatic fever is to impoverish the blood, especially

as regards that highly important portion of it, its colouring matter. All that bleeding really effects is to relieve pain (which, however, may quickly return), for a few hours, while it undoubtedly aids the bleaching power of the rheumatic matter, and, as I have observed in several cases, it increases much the tendency to a chronic rheumatic state, and consequently prolongs the convalescence. That bleeding in rheumatic fever is unnecessary, and that its omission diminishes rather than increases the tendency to certain internal inflammations, I am so convinced, that for several years I have not abstracted blood, in any way, in a single case of the disease. The treatment of rheumatic fever by the abstraction of blood, even in moderate quantity, but more especially in large quantity, appears to me to increase the danger of internal effusions into the pericardium and the pleura, and also into the synovial sacs of the joints (*vide* Case VII). Under this treatment we also meet with the most violent and troublesome cases of delirium, which, under other methods, either does not occur, or is developed in a form sufficiently easily controlled. I am very much disposed to believe that this treatment predisposes to pericarditis and endocarditis; and that, if these affections occur in a case in which venesection has been freely practised, they are much less tractable than when you have to deal with them in a patient who has not suffered from loss of blood.

3. A third plan is that by mercury. Some recommend that calomel and opium should be freely administered until salivation is produced. The great objection to this treatment is, that it is an attempt to cure one fever by setting up another, and, in some respects, a worse. Even supposing the original disease succumbs, your patient comes out of his rheumatic fever with loose teeth, ulcerated gums, and all the painful and offensive concomitants of ptyalism. Now, I say that, under such circumstances, the remedy is nearly as bad as the disease; and, moreover, it does not in the least guard the patient against what may be termed the accidents of his malady—those severe internal inflammations —pericarditis, endocarditis, pneumonia, pleuritis, peritonitis. I have more than once seen pericardial inflammation supervene while the patient was in a state of salivation, of which the case

(II) of Sarah Green, detailed in the third lecture, is a good example. When we consider how differently various persons are affected by a mercurial course, and how much some suffer from it, even if given in small quantity, it would seem highly inexpedient to adopt this plan of treatment, for it assuredly offers no prospect of effecting either a speedy cure or a speedy convalescence, much less both together.

It is worthy of remark, that rheumatic patients sometimes exhibit a distinct tolerance of mercury, and are with difficulty salivated. The following case is an instance in point, affording, at the same time, but little encouragement to the supporters of the mercurial treatment, and yielding no evidence of the anti-rheumatic power of mercury.

Case IX* (vol. xv, p. 70).—John Smith, a lad, fifteen years of age, after a day of unusual exertion, November 29th, 1845, went home and to bed very much fatigued, and awoke the following morning to find his knees and ankles painful, hot, and swollen. He rested all that day, and then returned to his work in much pain, with feverish symptoms, and general indisposition. On December 3d, the fourth day of the disease, he could not leave his bed, and the next day was sent to the hospital. When admitted the same joints were still affected; pulse 100; respirations 36. The knees and ankles were wrapped in cotton wool, and he was ordered ten grains of Dover's powder in saline mixture three times a day, and a dose of the hospital white mixture.

On the night of the 5th (sixth day), he perspired freely and rested well; but on the morning of the 6th (seventh day) he felt pain all over his chest, with difficulty of breathing and an unusual throbbing at his heart; his pulse had risen to 110. On the 8th there was no improvement; the pain in his chest continued; his face wore a constrained appearance; he was breathing 36 times a minute, and his pulse was 104; some roughness of the first sound was heard towards the base of the heart; the urine gave a copious precipitate of urates. He commenced taking a grain of calomel with each Dover's powder, the ape-

* Reported by my former clinical clerk, Dr. Sturt.

rient mixture was repeated, and a blister applied over the heart.

On the 9th, the tenth day of the disease, there was a general improvement, though the same symptoms remained. This continued ten days, the articular affection subsiding, while our patient's general condition improved, and his pulse fell to the natural standard, so that on the 18th he was ordered two grains of the sulphate of quina three times a day. On the following day, the 19th, he was not so well, complaining of pain in his back and shoulders, palpitation, and difficulty in deeply respiring. A blister was applied to the chest, the quina discontinued, and the Dover's powder and calomel resumed, the dose of the calomel being increased from one to two grains. On the 20th he was better again; but a mitral systolic bellows-sound was heard, and the blister was repeated, and afterwards dressed with a mixture of equal parts of mercurial and savin ointment. The improvement continued, and on the 26th he left off the calomel, after having taken it, with but one day's omission, for eighteen days without salivation. He then resumed the quina; and by the 7th of January was well enough to leave the hospital, after a residence there of about five weeks.

He had not been away a week when he was again attacked with pain in his chest and palpitations, together with pain and swelling of the left knee; he was therefore re-admitted on the 17th of January, and a blister applied to his chest. On the 18th, he commenced taking the alkaline mixture of the bicarbonate and nitrate of potash, which I now so constantly prescribe, with five grains of Dover's powder at night. On the 19th the blister was repeated, and three grains of calomel added to each Dover's powder; although better, he was still suffering from some articular pains, from palpitation, and from symptoms of general disorder, with a rapid pulse of about 110. A rough systolic bellows-sound was audible both at the base and apex of the heart on the 21st; and on the 24th a slight pericardial rubbing sound was also heard at the base. By the 27th he was free from pain and feverish symptoms, but his breathing remained quick, and the rubbing sound was still heard.

The last note was taken between a fortnight and three weeks

later, on the 14th of February. He had then been taking citrate of iron for some days, but was evidently much exhausted, as his pulse, which was 88 when reclining, rose to 108 on standing up. We have no record of the date of his discharge.

4. Another plan of treatment which has been proposed is by colchicum and by guaiacum. These drugs, but especially colchicum, have long been considered to possess a specific influence over rheumatic and gouty affections; and it has been laid down that the rheumatic condition will be subdued in just such proportion as you get your patient under the influence of the colchicum, somewhat in the same way as quinine exercises a specific influence on ague. Now I think it requires only two or three cases to prove to a candid mind the fallacy of this doctrine; I myself have frequently given this remedy the fairest trials, but I could never discover any effect from it sufficient to entitle it to the character of a specific. That it is capable of exerting a remarkable influence, as well for evil as for good, on gout, I do not deny; but even this must be admitted with considerable limitation; it is certainly far from exercising any similar or analogous influence in rheumatism, whether acute or chronic. The effect of guaiacum has also been supposed to be specific, and similar to that of colchicum; but it has even less claims than the latter. Both these medicines, when given in large doses, purge, and, in such doses, I have no doubt they may do some good, on the principle of eliminating the morbid material by the alimentary canal; but unless you give them in such quantity as to produce colliquative purging, you do but little towards cutting short the disease; and if you do give them in these large doses, you produce a degree of prostration and debility which is sometimes more dangerous than the disease, and you leave your patient to linger through a tedious convalescence. Colchicum given in small doses produces no good effect in rheumatic fever, according to my experience; on the contrary, I fear that in some cases it has a prejudicial influence on the nervous system, making it more irritable and susceptible of impressions, and rendering the patient more obnoxious to the various accidents that are liable to occur in the course of the disease.

5. Treatment by opium.—This plan of treatment has been lately revived by a very able physician, Dr. Corrigan, of Dublin. It has much to recommend it, and, on the whole, you will find it extremely serviceable in practice; but I do not recommend it alone: its great value consists in relieving suffering, and soothing the nervous system, while it promotes diaphoresis. The opium is given in large and frequently repeated doses, care being taken not to produce too much narcotism; but upon this point, in general, there is not much need for fear, as there seems to be in the generality of patients a remarkable tolerance of opium. Our patient, Elizabeth Stocking, whose case I have described in the last lecture, was ordered on the 23d a grain of opium, to be given every three hours, in addition to half a grain of the muriate of morphia, which she had previously been taking at night: in forty-eight hours she thus took sixteen grains of opium, exclusive of the morphia, yet her pupils were not at all contracted, nor was she in any degree narcotized. The effect upon her has been most beneficial — her nervous excitement has been calmed down, and her pain materially relieved. The same plan was pursued in the case of S. Green, also detailed in the last lecture: she had one grain of opium every three hours from January 8th to February 10th, excepting for one week, in which the dose was reduced to half a grain; the same tolerance of the remedy was observed. It will not, however, do to employ this plan alone; it should be conjoined with other treatment. I do not recommend it by itself.

I may here adduce another case in illustration of the benefit to be derived from opium, where there is much disturbance of the nervous system, with restlessness and delirium.

Case X* (vol. xxxiii, p. 55).—George Rinning, a tailor, forty-five years of age, of intemperate habits, and with some history of rheumatism in his family, obtained admission to the hospital on February 12th, 1851, with severe pain and swelling of the knees, ankles, and wrists, and profuse sweats. He stated that these symptoms came on, with loss of appetite and confined bowels, ten days previously, after exposure to wet and

* This case was reported by Mr. (now Dr.) E. Liddon, of Taunton.

cold. Blisters and cotton wool to the joints, nitrate of potash with liquor ammoniæ citratis every four hours, and a dose of hospital white mixture, were prescribed.

On the 13th, the day after admission, a slight systolic bruit was heard at the base of the heart; he was suffering rather less pain, but his tongue was coated, dry, and brown; the perspiration and urine were very acid; his thirst great, and appetite bad. Throughout the night he was delirious, and could get no sleep, and continued forgetful and talkative the next morning. The bellows-sound was more marked on the 14th, and a blister was applied to the chest; the nitrate of potash was discontinued, and a grain of opium was given every four hours. At night there was much less wandering, and towards the morning of the 15th he slept. During the day he was drowsy. His pupils were contracted, but there was much less pain, and general improvement had taken place. The opium was discontinued during the day, but repeated at night.

The case afterwards followed an ordinary and favorable course. By the 5th of March he was able to sit up, and was discharged on the 8th.

6. A sixth plan of treatment, proposed long ago by Dr. Haygarth, consists in giving bark in large doses, for which, more recently, the less bulky disulphate of quina has been substituted. Now just imagine the state in which the pathology of a disease must be, when measures so completely at the opposite extremes of our therapeutical resources are advocated for it—as venesection, to the amount of two or three pints, on the one hand, and large doses of quinine on the other; some would even give as much as five or ten grains two or three times a day. I have tried both methods of treatment, and I approve of neither; but if I were tied down to one or other of them, I should not hesitate to choose that by bark. In cases where the sweating is colliquative, and the urine copious and pale, with abundant precipitates of *pale* urates, I have seen great good rapidly result from the use of quinine; but I am not prepared to advise you to adopt this treatment from the beginning, because it tends to check secretion, and so may favour the development of internal inflammations.

7. The seventh and last mode of treatment that I shall mention to you is, that which you have seen me adopt frequently at this hospital, namely, *the treatment by elimination.* I give it this name, in order that you may keep well in view its main object—to promote the elimination of morbid matter by the various emunctories, and also that you may bear in mind the view of the pathology of the disease upon which it is founded.

It is probable that *the materies morbi* in rheumatic fever is lactic acid or some analogous agent. We know that the natural emunctory of this is the skin. Many chemists maintain that it will also escape by the kidneys; and if it ever does so, perhaps this is more likely during rheumatic fever than at any other time. Again, since vitiated digestion is apt to produce it in undue quantity, and it, therefore, is formed abundantly in the stomach, there is every reason to think a certain proportion of it may be carried off through the alimentary canal. The indications are, then, to promote the action of the skin, the kidneys, and the bowels; to use antacid remedies; and to give large quantities of fluid for the free dilution of the materies morbi, and to supply the waste caused by the drainage from diaphoresis and diuresis.

The best way to promote the action of the skin is by opium, especially if you combine with it nitre and ipecacuanha. For this purpose I sometimes use a compound which resembles the original Dover's powder, in containing nitrate of potash instead of sulphate of potash, as prescribed in the compound ipecacuan powder of the Pharmacopœia. Our usual prescription is one grain of opium, one grain of ipecacuanha, and five grains of nitre; this must be given every two, three, or four hours, according to the urgency of the symptoms and the need the patient has for opium. This drug quiets the nervous system, and procures sleep, and with the ipecacuan promotes sweating; while the nitre acts upon the kidneys, and the ipecacuan may exercise some influence on the liver.

The best alkali on the whole is the bicarbonate of potash, which may be given in large and often-repeated doses—a scruple or half a drachm every third hour. Sometimes the acetate of potash answers very well in similar doses, and many physicians much prefer it to any other alkaline salt.

Next you must give purgatives to such an extent as to keep the bowels in a loose state, taking care not to carry this treatment so far as to weaken your patient, or worry him by obliging him to be frequently moved in and out of bed. You will find it advantageous to use an alkaline purgative; and there cannot be a better medicine for this purpose than our hospital nostrum—the white mixture containing magnesia and sulphate of magnesia. Sometimes you may give the potassio-tartrate of antimony with advantage; but as it is a depressing remedy, it is seldom advisable to use it.

But while we are thus alkalizing our patient, and giving internally sudorifics and diaphoretics, ought we not to attend to the state of the joints? The diligent physician will tell you by all means to attack them at once—but there is such a thing as "nimium diligentiæ" in physic as well as in other matters. Many will say, the best thing you can do is to leech a painful and swollen joint: I formerly tried this practice extensively, but for some time past I have not done so, as I generally found it either useless or injurious. You may apply leeches, and in a short time after you will find the pain and swelling removed, and you may be disposed to say, "here is a proof of their efficacy;" but wait twenty-four hours, and then you will generally find the pain and swelling as bad as ever, and the joint in just the same condition as before. Now apply leeches, and you will probably fail to give any relief. You have by the first application relieved the pain for a time, but you have produced no permanent good—you have rendered the disease more erratic, and less amenable to subsequent treatment. Frequently when you leech a joint, the pain and swelling subside, but its fellow becomes swollen; leech it, and the swelling and pain return to the original joint. Nothing is more important to avoid, nor more troublesome if not prevented, than the erratic tendency of the rheumatic state. It will fly from joint to joint, and in pursuing it with leeches you only drive it out of one joint into another. I am satisfied that leeching the joints favours this erratic tendency.

I am not prepared, however, to advise you to neglect the local treatment of the joints. When they are much swollen and painful, you may give great ease to your patient by enveloping them

in a large quantity of the soft, carded cotton—commonly called *cotton-wool*. Over this you must wrap a sheet of oiled silk, so as to cover in the wool completely, taking care to have no part of it exposed. By this air-tight covering you keep the joints in a complete vapour-bath ; and when you come to remove the oiled silk and wool, after twelve or twenty-four hours, you find the wool completely saturated with moisture, which generally is strongly acid. You have seen this in Elizabeth Stocking's case. We find the plan so generally useful, that it is adopted in the hospital in nearly every case : it affords great relief, supports and keeps the limb steady, and at the same time promotes sweating. I may just mention, that this plan of enveloping the joint in wool and oiled silk is also very beneficial in gout.

In a few, and only a very few, cases, I have found the pain aggravated by the heat which this mode of wrapping generates ; and in cases where it is desirable to keep down the sweating, it is not advantageous to carry this plan beyond a day or two.

The best additional local treatment is that by blisters of small size, applied on or near the affected joints ; they are very useful both in acute rheumatic and acute gouty joints. I shall refer to this subject again. (*Vide infra*, p. 74.)

You perceive that all the means employed in this mode of treatment tend to elimination, and to the relief of pain : the opiate sudorific affecting the skin ; the nitre and alkaline salts acting on the kidneys ; the purgatives on the mucous membrane of the bowels ; the wool and blisters on the joints.

During this treatment, while you allow your patients the liberal use of simple diluents, you must give a fair amount of nourishment from the first ; and I think this may be best supplied by a small quantity of good beef-tea, given frequently throughout the day.

Often you will find it useful, and always when there is a tendency to delirium, to give stimulants, such as brandy or wine. A good example of the benefit from the timely use of stimulants is afforded by the following case.

Case XI* (vol. xxix, p. 236).—John Wilks, æt. 24, was admitted February 9th, 1350. He had been attacked, about a

* Reported by Mr. Dickinson.

fortnight before, with pain in his left great toe, soon followed by swelling; at the same time he began to suffer from languor, loss of appetite and thirst; he sweated much at night, and noticed that his urine was high-coloured and deposited a deep-red sediment. Pain and swelling soon followed in most of the larger joints.

When admitted, he was sweating profusely, and the perspiration had the peculiar odour and acid reaction so constant in acute rheumatism; the right ankle, the knees and wrists, were painful and swollen, the least motion of the last causing excruciating pain; his pulse was 100; his tongue coated with a white fur. Fifteen grains of the bicarbonate with ten of the nitrate of potash, and five minims of tincture of opium, were given every four hours; and two blisters were applied to the wrists. A decided diminution of pain in the wrists followed the application of the blisters; at the same time the pulse became more frequent, and continued above 110; in other respects the symptoms remained the same on the 10th and 11th. On the night of the 11th he became delirious, and the delirium recurred the following night; he was therefore ordered half an ounce of brandy every two hours. There was no return of delirium. By the 15th, there was general improvement; on the 13th and 14th he had suffered from diarrhœa, but this had ceased; he was free from pain; his tongue was moist and cleaning; he slept well, and his pulse had fallen to 84, and improved in tone.

He continued to make favorable progress until the 5th of March, on which day he had a slight relapse, but soon recovered, and was discharged well on the 16th.

I have many more remarks to make on other points in the treatment of rheumatic fever, but must content myself now with having given you an outline of the eliminatory mode of treatment, and reserve my further observations for another lecture.

LECTURE V.

ON CERTAIN ACUTE DISEASES.

RHEUMATIC FEVER.

In my last lecture, gentlemen, after having passed in review six different methods of treating rheumatic fever, I particularly recommended to your attention one which we have been in the habit of using here, the object of which is to promote as much as possible the elimination of morbid matters from the system through the natural emunctories—through the skin, through the kidneys, through the bowels. I advised you to use opium freely, with potash and nitre, to give alkaline purgatives, and to relieve the pain and swelling of the joints by enveloping them in cotton-wool, surrounded by oiled silk. And all this I ventured to recommend to you in preference either to the plan of treatment by venesection, or that by colchicum or guaiacum, or that by calomel.

Now, it may sometimes happen that you will have to deal with a patient who is unable to take opium. What are you to do under these circumstances? There is no reason why you should change the general plan of treatment—you may still give sudorifics—and if your patient will bear sedatives, you can give hyoscyamus, or hop, or extract of lettuce. But it will, I believe, very seldom happen that, in this severe and painful malady, patients will be unable to bear opium in some shape or other; and the benefits to be derived from the proper use of this drug are so great that you ought to try it in various ways, and in different preparations, before you abandon it altogether. I think that practitioners often fail in obtaining all the good effects of opium from being too timid in the use of it, giving it in too small a dose, and employing it in a vacillating manner; you must give it in a large dose, not less than a grain, frequently repeated, taking the state of the pupils as your guide in its

administration. You will, of course, proceed with great caution if you find a very contracted pupil in addition to some degree of narcotism. Before you abandon the use of opium, remember that you have a great variety of forms in which to prescribe it; you have, among others, the compound camphor tincture, which is often borne when the other preparations fail; the acetate and muriate of morphia; Mr. Battley's liquor opii sedativus; and a preparation introduced by Mr. Squire, the solution of the bimeconate of morphia, which may be given in the same doses as laudanum; and codeine, as prepared by Pelletier, in Paris.

Again, it may happen, and this is by no means of unfrequent occurrence, that the swollen and painful state of the joints does not yield to the cotton-wool and oiled silk only, or that the heat, which that application generates, cannot be tolerated by the patient. What further treatment of the joints will you pursue? I have no hesitation in advising you to apply blisters; and I would recommend you to use every means in your power to get them to rise well. I do not think it advisable to apply *large* blisters; on the contrary, they are injurious, and their use is to be deprecated. The plan I generally follow is this: I order a small mustard cataplasm to be applied to the affected joint, and to be kept on for half an hour to redden the skin; after its removal the skin is to be carefully washed and dried, and the blister may then be applied; you must not let the size of this exceed that of a crown piece. It is better to apply two or three blisters in rapid succession, and to different parts of the joints, than one large blister. After the blister has risen well, if the swelling of the joint subsides quickly, as it very frequently does, you may let the blister heal as fast as it will; but if the swelling has not subsided, then you had better cut away the cuticle completely, and promote a free discharge from the blistered surface by dressing it with stimulating ointments. Some prefer to apply the blister above rather than over the joint.

You need not be afraid to apply blisters in the early stages of the rheumatic inflammation of the joints. I believe the dread which some physicians had, and have, of applying blisters near inflamed parts—as near an inflamed lung, or pleura, or pericardium—is due to their having used blisters of too great a size.

I have applied them very early to rheumatic joints in numerous cases, and always with more or less advantage, provided the blisters have not been too large. A very large blister is apt to do mischief and augment the inflammation of the joint; but a small one, varying in size from that of a crown to a half-crown, is almost invariably beneficial. When a very copious effusion has taken place into a joint, the plan of applying two or three small blisters in succession, at different parts of the joint, provided the first should fail in getting rid of the effusion, is productive of the best effects.

I have seen excellent results from the application of blisters to gouty joints, even in the most acute stage. A discharge of a large quantity of serum from the vessels of a gouty joint has all the good effects of the abstraction of blood from it, without any of the evil consequences of that mode of treatment.

You must exercise a proper caution not to carry the sweating or the purging process too far with your patients. It is impossible to lay down precise general rules on this subject; the state of the patient's pulse, his countenance, the mode in which he expresses his feelings, will sufficiently indicate the condition of his general powers to enable you to judge whether you are going too far or not. On this point of the treatment I would advise you to take as your motto, *ne quid nimis*—neither too much sweating, nor too much purging, nor too much opium. I shall not caution you against too much bleeding, but I deliberately, and without hesitation, advise you to omit that from your practice altogether in the treatment of this malady; and I do this from a large experience and observation of its little efficacy for good, and its great liability to do serious mischief.

All the world now knows how important it is in acute rheumatic cases—and, I would add, even in chronic also—to pay close attention to the heart. You should watch it from day to day, and from the very commencement of the attack; and if you find the smallest indication of a departure from its normal mode of action, attack it specially and at once. I say you should watch the heart from the very first moment the patient comes under your charge, for the cardiac symptoms are apt to come on very early, and in some instances they precede the articular affection. In our patient Elizabeth Stocking, the cardiac

symptoms must have developed themselves very early, as they were already well marked on her admission into the hospital.

The circumstances that will denote to you that the heart is beginning to suffer, are, irregularity of the pulse *in any way*, either as affecting its force or its rhythm—*i. e.* whether the intermission be partial or complete; or its becoming suddenly quicker or slower. Should any of these signs present themselves, you should at once institute the most minute scrutiny into the physical signs of the heart's action, and if you should find the slightest indication of a rubbing or bellows-sound, you may infer that either the pericardium or endocardium, or both, are beginning to suffer. Disease of the endocardium is especially to be feared if the bellows-sound is mitral systolic, *i. e.* if it be heard most distinctly over the apex of the heart, and beneath the left scapula behind, and accompany the first or systolic sound; under such circumstances, you may be sure that the endocardium is suffering, and that some portion of the mitral valve is implicated in the lesion. If, however, the systolic bellows-sound be heard most distinctly over the *base* of the heart, and along the course of the great vessels, and is therefore aortic, you must not at once infer that this is a sure sign of the existence of endocarditis affecting the aortic valves; you must bear in mind that an aortic bellows-sound may, and very frequently does, arise from an anæmic state of the system. I have already told you that the rheumatic state tends to diminish very much the proportion of the colouring matter of the blood, even in patients who have not been bled or otherwise roughly treated. The rheumatic state itself, then, by bleaching the blood, may give rise to aortic and even venous murmurs. How much more likely to be produced is the condition favorable to these murmurs when bleeding has been practised. You must be very careful not to fall into the mistake of treating an aortic murmur as due to endocarditis, which is really the result of the already bleached state of the blood. Such a mistake is not unlikely to be made, as the diagnosis is difficult between the anæmic murmur and that from aortic obstruction, and you can readily understand how an antiphlogistic process, especially if it included bleeding, would make matters infinitely worse in a case where the murmur was simply of the anæmic kind. The more you proceeded with

such a treatment, the more, of course, would the conditions favorable to such a bellows-murmur be developed, and the louder it would become. The following points will aid you in deciding upon the endocardial character of the murmur: First, if the sound come on very early in the disease; second, if it be rough in character; third, if it be not accompanied with venous murmur; fourth, if the patient has not yet displayed much anæmia; lastly, the probability of an endocardial affection is much increased if the murmur have been ushered in with some disturbance of the heart's action, such as I have already referred to.

It is important, especially with reference to prognosis, to keep in view that the mitral valves may be affected, first, so as to induce valvular imperfection; and, secondly, so as not in any degree to impair the function of the valve.

If the deposit of lymph take place on the auricular surface of either or both curtains of the valve, then you will have valvular imperfection; the curtains will not meet exactly, and a fissure will remain, of larger or smaller size, through which more or less of regurgitation will take place into the auricle at each ventricular systole.

But if the deposit take place on the *ventricular* surface of the valve—and it generally does so on the ventricular surface of its inner curtain—then you have no disturbance of valvular function. In both cases, however, you have a systolic bellows-sound, and in both cases that sound is best heard at the apex of the heart.

How are you to distinguish the one from the other? If the bellows-sound be purely regurgitant, its position is strictly at the apex; it becomes in a marked way faint as you proceed to the base of the heart, and it is distinctly audible beneath the left scapula; and, in addition, the sign pointed out by Skoda exists, namely, a marked intensification of the second sound. If the bellows-sound be not regurgitant, you hear it well up to the base of the heart; you hear it only feebly, or not at all, at the left scapula, and there is no intensification of the second sound. I may add that, in this latter case, the heart's disturbance, and the sufferings of the patient, are in a marked manner less than in the former.

It must also not be lost sight of, that a bellows-sound, now present, may be the result of a previous attack of rheumatic fever. You must rely mainly upon your knowledge of the patient, or upon such a history of his previous state as you can pick up by inquiry among his friends and relations, to distinguish whether the endocarditis is new or old. Of course, certain symptoms of heart disturbance of recent origin would favour the former view, but I know of no physical sign or sound which aids the diagnosis.

In the treatment of the heart affection, I am in the habit of acting upon much the same principles as in that of the joints; and I trust to free vesication and the promotion of a copious discharge, serous or sero-purulent, as the local treatment. I shall describe to you the plan I am in the habit of following, and which we have used with the most satisfactory results in Elizabeth Stocking's case. On the first indication or suspicion of heart affection, a large mustard poultice, made with flour of mustard and hot water, is applied over and beyond the region of the heart; this is to be kept on as long as possible. After its removal, and after the skin has been properly cleansed, put on a blister of good size; and you must be guided as to the dimensions of it by your opinion of the extent to which the heart is affected. You need not be afraid of large blisters here, as in the treatment of the joints, because the inflamed organ is much more distant from the surface than the synovial or other articular tissues.

If you pursue the plan which I have thus pointed out, and have drawn a large quantity of blood to the surface by the long-continued stimulation of mustard, you will generally succeed in producing very free and large vesication, from which you may obtain a considerable quantity of serum—or rather, I should say, of *liquor sanguinis*, for the fluid of the blister is serum containing more or less fibrine. If you examine the fluid from blisters, especially when the skin has been previously irritated by mustard, you will almost invariably find that it contains more or less of fibrine. In very many instances, if not in all, the coagulated fibrine disposes itself in a membranous layer in immediate juxtaposition with the deep surface of the elevated cuticle. On removing the cuticle slowly and

cautiously, the serum will not flow away; it is still retained by a very complete, but soft, moist, and almost spongy membrane. This is coagulated fibrine, which has entangled in it a large number of the white corpuscles. How these latter escape from the blood-vessels, or whether they are not the result of the organizing tendency of the liquor sanguinis, I cannot pretend to decide. It is clear, however, that blisters will take away the liquor sanguinis with its dissolved elements, and perhaps the rudiments of the white corpuscles. By blistering you take away that part of the blood which is the great agent in the development of new formations, and these are what you have to guard against in the cardiac inflammations. Moreover, by blistering you spare that most important part of the blood, the colouring matter, which seems especially valuable for preserving the nervous functions in a state of integrity, and which is no less important for maintaining the healthy action of the heart.

But some of you will say, "What! do you advise us to lay aside that which has so long been regarded as the sheet-anchor in the treatment of inflammations—namely, bleeding; and not only general bleeding, but topical bleeding likewise? If we are neither to cup nor to leech in pericarditis or endocarditis, what security, then, shall we have against the progress of inflammation—against the formation of excrescences on the valves—against ulcerative or suppurative processes being established in the heart, destroying its valves, and infecting the blood?" I am quite aware that the doctrine which I recommend for your adoption is likely to be regarded as extremely heterodox by many, but I believe the number of those who think so is daily diminishing. In the treatment of the cardiac affections which accompany rheumatic fever, you have two objects to keep in view—the first is, to check the morbid process completely, or to restrain it from producing such changes as may prove destructive to the tissues, and consequently to the mechanism of the heart; and the second, to obviate liquid effusions which may distend the pericardium, compress the heart, and so embarrass its actions, as well as the respiratory movements, as to prove seriously detrimental to life. Now, with regard to the first point, there can be little doubt that bleeding will not stop or prevent the formation of those fibrinous concretions which are

so apt to form upon the valves. The formation of these concretions is in a great measure mechanical, and in certain states of the blood they would form around or upon any opposing material, just as fibrine will coagulate round the bunch of twigs by which blood is beaten as it flows from a vein. In this rheumatic state, the contractile tendency of fibrine is apparently increased, as is shown by the uniform formation of a tough, buffy coat in the blood removed from rheumatic subjects; there is also a considerable increase in the number of white corpuscles; the buffy coat is formed of these two constituents, and the constancy of its formation denotes a tendency in these two elements to separate from the other elements of the blood in the rheumatic state. Doubtless, a disturbed state of the nutrition of the serous membrane or the endocardium, or of certain parts of them, precedes the formation of fibrinous deposits upon them; and this disturbance of nutrition is caused by the accumulation of the rheumatic matter in the vessels of the part. The effect of this is analogous to, if not identical with, that produced by a blister on the vessels of the skin, which I have just now described to you. The liquor sanguinis transudes through the parietes of the blood-vessels, and the plastic matter coagulates upon the surface of the endocardial and the pericardial membrane, forming there a substance identical, or nearly so, with the buffy coat of the blood. In the endocardium, which is in contact with the blood as it flows through the heart, this layer of plastic matter forms a nucleus, around which fibrine from the blood which flows over it may coagulate.

Now, if this be a correct account of the manner in which the plastic concretions develop themselves in pericarditis and endocarditis—and I believe it is that which is most consistent with our present improved knowledge of the blood and of inflammation—it is evident that the object of the practitioner should be to prevent the development of that altered state of nutrition which *precedes* the fibrinous formation, or to arrest it prior to the pouring out of the fibrine. Will bleeding do this? I think our experience of the effects of bleeding upon the joints ought to convince us that it will not, for bleeding certainly will not remove the rheumatic state from them; for, however it may relieve for a short time, by diminishing hyperæmia, or by some

influence on the nervous system, the flow of blood speedily returns with as great or greater activity than before. I apprehend that the state of the joints and that of the heart are as nearly as possible the same, the difference being that the nature of the synovial secretion offers a much greater physical impediment to the formation of fibrinous or plastic concretions in the joints than exists in the endocardium or in serous membranes.

And I would put another question—will bleeding cut short that state of blood which is so favorable to the formation of the plastic deposits? To this I answer likewise in the negative. Among the best of the modern researches upon the relative quantities of the elements of the blood in various conditions of that fluid, are those of Becquerel and Rodier. What do these observers say as to the influence of bleeding upon the blood? Why, that it considerably diminishes the red particles, that it very much augments the proportion of water, and that it affects but little or not at all the fibrine; thus, in short, you get a thinner liquor sanguinis, holding in solution the same, or nearly the same, amount of fibrine. In other words, you get a state of liquor sanguinis very favorable to transudation, and therefore very favorable to plastic formations.*

If, then, bleeding will not stop the inflammatory state which creates the undue determination of the blood to the pericardial and endocardial surfaces, and if it will not prevent the plastic formations, but rather favour them, surely it is not the remedy for pericarditis and endocarditis. And if the effects of venesection be—as beyond all doubt they are—to diminish all the solids of the blood but the fibrine, and to augment the water, surely the employment of this treatment is fraught with the greatest danger of creating liquid effusions into the serous and synovial sacs, which are so exposed to the action of the rheumatic matter.

These are, as concisely as I can put them before you, the theoretical grounds upon which I object to the practice of

* The analyses of Dr. Christison show an increase of fibrine under bleeding—and those of Dr. Beale show the same fact to a remarkable extent, in the blood of a dog bled on four successive days to the extent of six ounces each day.—Vide Todd and Bowman's Physiology, p. 312, vol. ii.

bleeding, whether local or general, for the cardiac affections of rheumatic fever. And my experience confirms me in the belief that the practice of bleeding is altogether unsatisfactory in its remedial results, and prejudicial in its consequences. I have likewise learned by experience that the practice of abstaining from this mode of treatment is perfectly safe, and tends to the best results. By the general plan of elimination—locally, by blisters—generally, through the sweating and other augmented processes of secretion,—you divert the rheumatic fever very freely from those great central and highly vascular organs which we are so anxious to protect from mischief.

Besides the local treatment that I have prescribed, you must, when the heart or any of the great internal organs is affected, still keep up the influence of opium upon your patient, whereby you secure a powerful means of keeping down excessive action of the heart, of calming the nervous system, and of promoting cutaneous elimination.*

You will bear in mind that both pneumonia and pleurisy are very common complications of rheumatic fever; but for the treatment of these affections I have nothing to add here to what I have said respecting the treatment of the cardiac affections. The treatment of both should be exactly the same—*mutatis locis.*

There is a very formidable complication of rheumatic fever,

* In the impression of this lecture, which first appeared in the *London Medical Gazette* for October 20th, 1848, the following paragraph appears:—

"I know that there are many physicians who speak lightly of the remedial powers of mercury in these rheumatic affections. But I confess to you that I am not prepared to give up the dogma of Dr. Farre, that mercury is opposed to, and breaks down, plastic formations. Still I must admit, and this is satisfactory for patients who may be prevented by idiosyncrasy from the use of mercury, that I have seen patients do extremely well without having taken a single grain of that medicine."

I have omitted this paragraph from the text in this edition of the lecture, because my subsequent experience has led me more and more to agree with those who repudiate the necessity for the employment of mercury in these affections; and I feel myself justified in declaring my belief, that under the treatment described in the text, the results are more favorable, and altogether more satisfactory, as regards the future of the patient, than when mercury is used. At the same time, I am quite ready to use calomel or blue-pill as a *purgative*, whenever either of them is suitable to the patient's condition.

respecting which I must say a few words. I allude to the delirium which is apt to manifest itself in the course of the attack; sometimes with thoracic inflammation, sometimes without it. It is very important that you should be prepared for this symptom, and that you should understand its nature, and its proper mode of treatment; it is not in itself a dangerous symptom, unless the practitioner fails in taking the precautions which are rendered imperative by its occurrence.

The delirium of rheumatic fever sometimes comes on gradually, the patient having been a little talkative and wandering for two or three nights; sometimes it comes on quite suddenly. In its general characters it resembles delirium tremens—generally, however, exhibiting less of the nervous tremor which belongs to intemperance. The patient is restless, busy, talkative, picking or pulling the bed-clothes, frequently rising in bed, and wanting to get out of bed, reaching out his hand as if to catch hold of some object before or behind him, and sometimes—a most unfortunate symptom—obstinately refusing to take either food or medicine.

In many instances, as I have already said, this delirium ushers in pericarditis, pleurisy, or pneumonia; frequently, however, it occurs after one or other of these maladies has set in, and sometimes it occurs without them. It has, therefore, I think, no necessary connexion with these internal inflammations, although it frequently accompanies them.

Now, what is the nature of this delirium? It used formerly to be viewed as a metastasis of rheumatism to the brain, and to be treated antiphlogistically. I have treated some cases in this way, and on this hypothesis, and I have had the opportunity, in consequence, I believe, of this treatment, of examining the state of the contents of the cranium in a few such cases. I can therefore assure you that there is no more inflammation, either of the brain or its membranes, in these cases than in delirium tremens. The membranes are perfectly free from abnormal deposit, the pia mater is pale, and the gray matter of the convolutions remarkably so, and the subarachnoid fluid is increased in quantity. These signs indicate, not only that the brain has been imperfectly supplied with blood during life, but that the vascular pressure upon it is less than it ought to be,

and that, consequently, an increase of the subarachnoid fluid has taken place.

When, then, we consider the circumstances in which the brain is placed in these cases, we cannot wonder at its functions being disturbed. In the first place, the organ is supplied by a depraved blood—a blood deficient in its most important staminal principle, its colouring matter—a blood infected with an abnormal material, the rheumatic virus, whatever that may be; and a watery blood, which is the more apt to exist, if the patient, as is very often the case, have been treated by sanguineous depletions. Such a blood is ill suited for the proper stimulation of the heart, and consequently it is not propelled by that organ with its proper force, although the rapidity of the heart's action may be much increased; and if the heart be inflamed, there can be no doubt that the effect of that inflammation will be to weaken still more the propelling power. Hence, in cases of this kind, the brain is feebly furnished with a blood, poisoned, poor in colouring matter, and abounding in water.

I have met with a few cases, in which the patient, having evinced previously little or no delirium, has become rapidly comatose, with dilated pupils, and sunk quickly. And it sometimes happens that patients who have been actively delirious will suddenly fall into coma and die; and sometimes they die suddenly, while making some effort beyond their strength, in the midst of their delirious ravings. The state of the kidneys may have some influence in determining the mode of death in those patients who pass quickly into coma, as we know that defective action of those organs so often exercises a baneful influence on the brain.

A case occurred to me in private practice which shows how rapidly rheumatic fever will sometimes run through all its stages, and exhibit all its phenomena—articular, cardiac, cerebral—notwithstanding the active and early treatment by calomel, colchicum, purging, &c. I will give you the case briefly.

Case XII.—A student of one of the universities, aged twenty-two, had complained for two or three days of pain in the left foot. This became suddenly very much aggravated at the railway station, as he was starting for his college, on the 17th of

April, 1855. He went to an hotel, and put it into mustard and water, but proceeded next day to his destination. The rheumatic affection then extended to all the large joints; there was free sweating, and numerous sudamina made their appearance. He was treated by mercury, colchicum, liquor antimonialis, liquor ammoniæ acetatis, and latterly Dover's powder. I was telegraphed for to see him, and found him, on the evening of April 27th, extremely ill: much purged—passing watery stools, apparently from the liquor antimonialis; he had a full throbbing pulse, and soon became very restless and delirious. I saw him again at midnight, and detected a friction sound over the heart; the delirium had increased, with a comatose tendency; but he could still be roused, and then recognised me and others about him. I ordered him half a drachm of the bicarbonate of potass and a grain of opium every three hours, and a small quantity of brandy. In the night the delirium increased, and he refused to take food or medicine; his breathing became catching; pulse 120; and soon after nine o'clock the next morning he died, eleven days from the supervention of the acute symptoms.

If I were to treat such a case from the beginning, I should employ opium at once; possibly also, liquor ammoniæ acetatis; or, more probably, large and frequent doses of bicarbonate of potass, wool or blisters to the joints, beef tea, and brandy or wine freely. Purgatives should be given carefully, but not so as to cause colliquative purging. Such a treatment would husband the strength, and enable the patient to resist the influence of a large accumulation of the rheumatic poison, such as must have been present in this case.

You will find a valuable collection of cases of delirium and other disturbances of the nervous system, in connexion with rheumatic cardiac affections, in Dr. George Burrows' interesting and most valuable work on Disorders of the Cerebral Circulation. The evidence which Dr. Burrows has adduced in that work should teach us, that whenever we meet with a case of delirium, especially of rheumatic delirium, we should diligently explore the region of the heart, and watch the condition of that organ most carefully from day to day.

But this delirium, as I have before said, has no *necessary*

connexion with the heart affection—at least, with endocarditis and pericarditis—for it occurs in cases of general gout, in which there are no such heart affections as those in rheumatic fever, and the delirium of gout resembles precisely that which I have described to you as belonging to rheumatic fever.

I have seen, indeed, this delirium in persons of strongly-marked rheumatic or gouty diathesis, accompanied by all the signs of rheumatic fever—the sweats, the furred tongue, and the lithic urine, and not only without cardiac, but even without articular affection.

I may make this further remark before I refer to the mode of treating this delirium, that what I have seen of it has strongly impressed me with the belief that it is much more apt to occur after bleeding, and in weakly subjects, than when depletion by bloodletting has not been employed, or in sthenic cases. It is also often an indication that your patient is being reduced too much by sweating, or purging, or some other means.

The development of this delirium should be, as I have already remarked, a warning to the practitioner to look out for cardiac or other internal inflammations, as pneumonia or pleurisy, or even peritonitis—which sometimes, although rarely, occurs in rheumatic fever—if such have not been previously detected. But it should likewise be regarded as a signal of distress, denoting that the powers of the constitution are unequal to the severe trial through which the patient is passing; and he should immediately come to the patient's aid, and make arrangements for having him constantly watched by competent nurses or other attendants, taking care that the patient shall never be left alone. If he have been sweating freely, that must be checked; the amount of bedclothes may be reduced; if his joints have been enveloped with wool, it must be removed. In like manner, any other too free evacuation must be stopped, as purging, or the too copious discharge from a blister. Nourishment must be given very frequently, but in small quantities, so as not to embarrass the stomach; and this should consist of beef-tea, arrow-root, milk; and it will be always necessary to conjoin with this wine or brandy, or porter when that has been an habitual beverage, also to be given in small and carefully-adjusted quantities. If the patient be wakeful, sleep must be procured by the free ad-

ministration of opium. These are the points to which you will have to direct your most watchful care. Provide against your patient being allowed to exert himself beyond his strength; remember that it is in this state that patients often die suddenly by syncope, and be careful to nourish and support them well. Eschew all local treatment to the head; even the application of ice is calculated to do mischief, by depressing the heart's action.

When, however, the patient evinces a marked tendency to coma, then of course you will not use opium; I would advise you to shave the head, and to counter-irritate it and the back of the neck, by sinapisms first, and afterwards, if you find it necessary, by blisters, pursuing at the same time those measures for the support of the patient which I have already pointed out, and which, you may be assured, are not less necessary in the comatose cases than in those in which active delirium prevails.

The two following cases afford interesting examples of the more severe forms of nervous symptoms—delirium, convulsions, and coma—as they occur in the course of rheumatic fever; and the post-mortem examinations are instructive as pointing out the non-inflammatory nature of the brain affection. I bring them before you with the view of illustrating the real nature of this delirium.* The treatment was not such as I should adopt now. It was on the whole antiphlogistic (so called), although moderately so; negative, as I believe, so far as regards any good effects; but injurious, if not directly, yet by excluding other means which would have done good.

Case XIII (vol. xii, p. 162).—The first of these cases is that of Maria Edwards, a servant, seventeen years of age, whose health had been previously good. On the 17th of October, 1844, she was seized with rigors, followed by a reaction, with pain in her limbs; at the same time she lost her appetite, became very thirsty, and suffered from headache and giddiness. On the following day both ankles became hot, swollen, tender, and painful; subsequently other joints became painful, and she was admitted on the 25th, the ninth day of the attack, with all

* For a full discussion of the pathology of this and other forms of delirium, see the Lumleian Lectures delivered at the College of Physicians, 1850; and published in the *London Medical Gazette* of that year.

the symptoms of rheumatic fever. Both knees and ankles were then tender and painful, but not much swollen; the pulse was 120; the skin hot and perspiring; tongue furred. A systolic bellows sound was heard all over the præcordial region, and there was a slight catching pain in that situation. The urine was dark-coloured, and loaded with urates and phosphates. The joints were wrapped in cotton wool and oiled silk, a blister was applied to the chest, and some Dover's powder ordered to be taken every fourth hour. On the day after admission blisters were applied to both knees.

By the 27th there was little alteration; she had no sleep at night. On the 28th she became delirious. For several days the same symptoms continued with but little variation, delirium recurring at night.

On the 31st (the fifteenth day) a distinct to-and-fro rubbing sound was heard over a great extent of the cardiac region. On the 1st of November, despite of two grains of calomel with Dover's powder and a grain of digitalis every fourth hour, with forty minims of Battley's solution of opium at night, the delirium, which had hitherto been confined to the night, was prolonged into the day. On the night of the 1st she was very restless, wandered much, and would get out of bed. Three ounces of wine were administered, and a little sleep procured, but the delirium soon returned, and about noon on the 2d forty minims of the liquor opii sedativus were given; her pulse was then 124, weak and compressible; the rubbing sound remained; her pupils were contracted; she was drowsy, and evidently much affected by the opium; she also suffered severely from headache, for which ice was applied, apparently with benefit.

On November 3d the delirium had ceased, and her pulse had fallen to 96, and improved in tone; the three ounces of wine were continued during the day, and thirty minims of the liquor opii sedativus were given at night. For several days our patient continued better, nearly free from pain, and sleeping more quietly at night; the rubbing sound being still audible. On the 8th her gums were found tender and white; the saliva was slightly increased, but no fœtor was observed; the calomel was reduced.

On the 11th (twenty-sixth day) there was an unfavorable

change, marked by a return of pain in the spine, shoulders, and right side, by restlessness and rambling talk at night, and by a weak and rapid pulse numbering 140. These symptoms seemed to call loudly for supporting treatment, the daily supply of wine was therefore increased from three to five ounces (an insufficient quantity), and ten minims were added to the opiate at night. Some rest and comfort followed, and thus encouraged, we pushed the treatment a little further, giving half a drachm of the aromatic spirits of ammonia three times a day in camphor mixture, a grain of the sulphate of quina as often, and one ounce more wine. The blister to the chest was repeated, and afterwards dressed with mercurial and savine ointment. Though otherwise a little better, she continued to pass noisy, restless nights. The rubbing sound softened down, and nearly disappeared, making a loud systolic bellows-sound more plainly heard.

On the 16th there was a fresh accession of præcordial pain, and a loud to-and-fro rubbing returned. The quina was doubled, and given every four hours with ten minims of tincture of opium. The record of the 18th is as follows:—"She only slept two hours last night, and not at all for the twenty-four hours preceding. Pulse feeble, fluttering, 140; skin cool." At night she was delirious, chattering and singing, and got no sleep. On the 19th her pulse was 140, small and weak; her pupils contracted. Five ounces of brandy and some arrow-root were now given in the course of the day, and, to humour her fancy, a little fish was allowed; a grain of the muriate of morphia was given at night, and the quina was increased to four grains. In the evening the foot of the bed was raised, that her head might be lower, and a quieter night followed. The next day brandy was regularly administered every half hour in arrow-root. The last note was made on the 21st, after her death:—"She was wandering all night; her pulse weak, quick, and fluttering. At seven o'clock this morning she changed for the worse; the rhonchus of the dying was heard; and, despite of brandy, administered at intervals through the night, she died at half-past ten this morning. Nitric and sulphuric æther were also given, in camphor mixture, through the night."

The examination of the body was made twenty-seven hours

after death. The brain was found healthy, but the gray substance pale; there was no effusion on the surface or within the ventricles.

The lungs were congested, but otherwise healthy.

The opposed surfaces of the pericardium were adherent throughout by a layer of lymph, a quarter of an inch thick, but soft behind, and containing some fluid. The pleura, where in contact with the pericardium, was adherent, but there were no adhesions elsewhere. Warty excrescences of lymph were found on the margins of the aortic and mitral valves.

Here was a case which exhibited very clearly the natural course of the acute rheumatic disease with its complications. It was not likely that any part of the treatment would have materially modified the phenomena. And while we found unequivocal marks of intense pericardial inflammation with abundant plastic deposits, there was not a sign of anything to indicate inflammation either of the substance or of the membranes of the brain. That organ was pale, poorly supplied with blood, and resembled the brain of an animal bled to death. Again, let me remind you that there was none of that serous effusion on the surface of the brain to which many attribute so prominent a part in the production of the phenomena of delirium and coma. These effusions, indeed, we now know, are *results* of the diminished size of the brain which follows its imperfect supply of blood, and its impaired nutrition; and as they do not exert any undue pressure on the brain, or any part of it, they produce no symptoms during life.

Case XIV (vol. xii, p. 57).—The second case is that of a single woman, Martha Mitchell, thirty-four years of age, who was admitted into King's College Hospital on the 18th of June, 1844, with her third attack of rheumatic fever. She stated that her general health for many years had been far from good; that she had suffered, she believed, from attacks of acute inflammation of the liver, with pain in the right hypochondrium and shoulders, and dyspeptic symptoms; that since her last attack of rheumatism, seven years previously, she had suffered from palpitation and dyspnœa on slight exertion.

One evening, more than a week before her admission, she went to bed unwell, with pain and stiffness in the right hip, and a feeling of chilliness, and awoke in the morning with pain in all her joints, especially in the knees and ankles, which subsequently became red and swollen; her appetite had completely failed, she had great thirst, and towards evening shivered violently. Two days afterwards she was suddenly seized with palpitation and dyspnœa. The articular affection continued up to the time of her admission; she also perspired much towards night; the bowels became confined; the urine scanty, and very dark.

When admitted, the joints of the arms, as well as the knees and ankles, were affected. There was pain in the præcordial region, and a systolic bellows sound was heard over the base of the heart, and in the course of the aorta. Dover's powder, with nitrate of potash, was given every four hours, and the joints were wrapped in cotton wool.

The next day, the 19th, the joints were less painful, her tongue covered with a yellowish brown fur, and red at the tip and edges, her pulse 120, and respirations 34. As there was still præcordial pain, a blister was applied, and two grains of calomel added to each powder.

The case goes on as follows :—"11 p.m., June 19th. Having continued up to this hour in the same state, complaining of little or no pain, the physician's assistant was called to her. He found her delirious, talking incoherently, and the delirium accompanied with hallucinations; pulse somewhat increased in frequency, 128, weak and compressible; her skin hot and perspiring.

"She was ordered thirty minims of the liquor opii sedativus immediately. She slept after taking the opium, but at two o'clock a.m. the physician's assistant was again sent for in consequence of her having had a convulsive fit affecting all her extremities. He found her lying on her back, her pupils very much contracted and insensible to strong light; pulse 132, weak but regular; her head hot, but the forehead perspiring; the respirations were 30, and of a croupy character. She was quite comatose. She had a return of the convulsions, screamed out, and died."

The body was examined thirteen hours after death. The following is the account from the case book :—"The body was very exsanguine externally ; the lips blue ; the skin of the face and arms much freckled. The head, chest, and abdomen were examined.

"The vessels of the pia mater were not more than ordinarily injected except on the left side—(hypostatic?). There was no fluid in the arachnoid or subarachnoid cavities; the surface of the arachnoid membrane was, however, moist. On dividing the hemispheres horizontally, the vascular pink points were rather numerous, but no trace of disease was discovered.

"There was no fluid in the pleura, but adhesions were found around the apex of each lung, where also there were one or two caseous tubercles: the lungs were elsewhere healthy.

"The pericardium was almost universally adherent, and the adhesions were organised. Besides these, however, there were traces throughout of fresh inflammation—there were many flakes of soft lymph, and collections of a few drops of fluid here and there, with the surface much injected. The edges of the mitral valve were much thickened; all the other valves were healthy.

"The liver was large, and broke down readily under pressure; its convex upper surface was covered with a distinct, though thin, layer of apparently condensed cellular membrane, with one small patch of lymph. The remaining abdominal viscera were healthy."

In the first of these cases, there was no evidence of any abnormal condition of the brain or its membranes, excepting, indeed, a slightly anæmic one. In the second, the congestion of one side of the pia mater was probably mechanical, and due to the gravitation of the fluid to the most depending part after death. The increase in the number of vascular pink points observed in the substance of the brain was probably connected with the mode of death,—in convulsions, which we well know congest the brain.

The next case is worthy your attention, illustrating as it does all the phenomena of rheumatic fever, the accompanying

delirium, and the benefit likely to result in such cases from the early and liberal use of opium and stimulants, as I have recommended to you.

Case XV* (vol. xxxiv, p. 166).—George Gough, a footman, nineteen years of age, of temperate habits, and usually enjoying excellent health, was taken ill, five days before his admission on the 10th of February, 1852, with general lassitude and aching of the limbs. The pains soon became localized in the joints with swelling; he also had pain in the left side of the chest.

On admission, the ankles, knees, and wrists, were swollen and painful, and the hip and shoulders also; perspiration was profuse and acid; the urine acid, high-coloured, and full of urates; his pulse was 104, and respirations 30; his tongue white and furred; he had lost his appetite, and could not sleep. On examining the chest, there was a slight systolic bellows-sound heard at the apex of the heart. He was ordered some of the usual mixture, containing fifteen grains of the bicarbonate with five of the nitrate of potash, and five minims of laudanum, every four hours. The joints were wrapped in cotton wool and oiled silk, and a mustard plaster was applied over the heart.

During the next two days there was not much alteration, and he passed sleepless nights, although the tincture of opium in the medicine had been increased to ten minims. The urine continued loaded with urates and phosphates, and its specific gravity was about 1030. A blister was applied to the left ankle, and mustard plasters to each hip. The medicine was now given every four hours, and the laudanum increased to twenty minims. As his bowels were confined, he was ordered five grains of calomel, followed by a dose of hospital white mixture.

On the 14th he was in less pain. Sudamina were observed scattered over his chest. That evening a to-and-fro rubbing sound was first heard over the base of the heart; this continued, and the next day a blister was applied, and strong mercurial ointment used in the dressing; his pulse remained as high as 126, and the respirations 40. On the night of the 15th he was

* For the record of this case I am indebted to my clinical clerk, Mr. Pearl.

for the first time delirious. On the 17th the whole body was covered with sudamina, and as the restlessness continued, he was ordered a night draught, containing half a drachm of tincture of opium; three grains of carbonate of ammonia were added to each dose of the mixture, and he began taking one ounce of wine every three hours (a quantity which, I think, was not adequate to the demands made upon the nervous power). That night, however, he was very delirious, trying to get out of bed and leave the ward, without any corresponding increase in the severity of the other symptoms, there being, on the contrary, a general improvement, excepting that the pulse remained 120. On the following day he was ordered a pill, containing two grains of calomel and a quarter of a grain of opium, with each dose of the mixture. On the 19th the delirium was not confined to the night, but he continued muttering to himself in the day, unless aroused or spoken to. The opium in his medicine was increased by five minims, and he took it every three hours; mercury also was rubbed into the axillæ.

On the 20th the delirium still continued, and he seemed unconscious of what was passing around him; his pupils were contracted; pulse 116, respirations 36. The mixture was omitted, and five grains of carbonate of ammonia were given every two hours, together with half an ounce of brandy in beef-tea every hour; the pill was continued every six hours. The favorable effect of this active stimulation was almost directly apparent: that same night he was much quieter, and slept a little, and on the morning of the 21st his pulse had fallen to 104, and the respirations to 26; some swelling of the knee-joints, and a pericardial rubbing sound, still remained. He again had a comfortable night, and the next day, the 22d, his pulse was 96, and respirations 34. On the 23d he was going on most favorably: as his urine was alkaline, twenty minim doses of chloric æther were substituted for the ammonia, and the brandy was reduced. On the 24th he was free from pain and progressing favorably; it was then just a fortnight from his admission, and about eighteen or nineteen days from the commencement of his illness.

After this he remained in the hospital about a month, regaining his strength under a course of tonics and good feeding;

a rubbing sound, and also a faint bellows sound, continued to be heard for some time over the heart; there was also some chronic swelling of the knee.

I have already told you that you must be careful to carry out this general plan of elimination with the closest attention and regard to the powers of your patient's constitution. I allude to this subject again, for the purpose of mentioning to you a sign which has, over and over again, proved most valuable to me, in leading me to pursue an altered course of treatment. When the patient has begun to pass pale urine, in good quantity, either without precipitate, or with a greater or less quantity of pale urates, you will almost invariably find that he will be the better for a more generous treatment, even although the articular affection still continue troublesome. You may give him ammonia, or quinine and sulphuric acid, and in many instances you may give wine or brandy; and I have been astonished at the rapidity of the progress of cases under this altered treatment; patients, whose symptoms had been stationary for two or three days, have, under the circumstances and treatment I have described, become convalescent in little more than forty-eight hours.

The plan of treatment which I have now recommended to you, does not contain any new remedy, nor does it profess to point to any summary method of treating rheumatic fever; it is merely the application of old and well-appreciated remedies to the treatment of this formidable malady, in furtherance of a certain determinate object—that of eliminating morbid matter, at various points and through different channels, from the current of the circulation. Since I have adopted this mode of treatment I have much more rarely met with those accidents of the disease —pneumonia, pericarditis, delirium, &c., which are so formidable to both the patient and practitioner, in the same severe form in which I used to meet with them under a more depleting treatment; and when such severe cases do occur in the hospital, they are generally persons who have suffered from a depleting treatment prior to their admission, or who have been thrown into a very reduced state from other causes. Again, I find that under this treatment the duration of the disease does not exceed from ten days to three or four weeks, and that relapses, which

were very frequent under the treatment by bleeding, are of rare occurrence under this plan. Now, it was formerly the dictum of an eminent physician, "that the only cure for rheumatic fever is *six weeks*." By this he meant that the disease would take its course, that time was its only cure, and that this time was not less than six weeks. But I should not attach much importance to a plan of treatment which failed to get patients into a good state in a much shorter time than that. Our patient, Elizabeth Stocking, whose case had been a severe one, and who had pericarditis and slight delirium, has been in the hospital now just eleven days, and had been ill three days prior to her admission, and you see that she is convalescent already. She has lost every rheumatic symptom; all the pains in her joints have ceased; her tongue is clean, and I have no doubt that in two or three days more she will be struck off the sick list altogether. And, as the last, though not least, advantage of this treatment, there is no fear of those unpleasant consequences which are so prone to follow in the wake of this disease; there is no fear of a tardy anæmic convalescence, for her blood has been spared; nor of a state of chronic rheumatism, for there is every indication that the whole of the morbid material has been eliminated from her system.*

* The last note made before this patient left the hospital is dated June 17th, and is as follows:—"She is improving in health and strength: appetite good: sleeps well."

I must add here an allusion to the plan of administering acetate of potash, as suggested by the late Dr. Golding Bird, and large and very frequent doses of bicarbonate of potash, as put in practice by Dr. Garrod. Both these physicians aim at making and keeping the urine alkaline. There is nothing in this treatment which militates against that which I have advocated in the foregoing lectures, and I may add that the free administration of alkalies is usually a very valuable practice, according to my experience.

LECTURE VI.

ON CERTAIN ACUTE DISEASES.

CONTINUED FEVER.

GENTLEMEN,—I wish to-day to call your attention to a case of common continued fever, with enteric disease, also called typhoid fever, which we have had lately in Rose Ward, and which we have watched with great interest, and not a little anxiety, for some days past. The case ended fatally, and for this reason I am the more desirous not to let it pass without some observations upon it. And I shall take this opportunity of giving you the following piece of advice: never shrink from analysing and carefully thinking over the cases which prove fatal under your care, with a view to inquire, whether by a little more care you might not have been more precise in your diagnosis, and whether you might not have been more watchful in your treatment, or have adopted a more promising course. Such an inquiry, if faithfully pursued, involves an amount of self-examination which, in course of time, cannot but redound most beneficially to the character of the practitioner.

It is a doctrine supported by our best physicians and highest authorities, that you cannot cure a fever, that is, that you cannot cut it short; you can guide the patient through the several stages of the disease, you can support his strength, uphold his vital powers, until the influence of the poison is worn out, and combat any accidental affections which may arise in the course of the treatment, such as diarrhœa, pneumonia, &c. By such careful management you may save the patient, by preventing him from dying by exhaustion, and you may shorten his convalescence considerably.

This is a doctrine to the truth of which I have for many years given my full assent, not only as regards typhus and typhoid fever, but also with respect to other fevers,—those, for instance,

connected with the exanthemata. And although many, from time to time, have professed by some heroic method, adopted very early, to cut the fever short, and thus to convert what would otherwise have been a tedious and painful illness of three or four weeks, into a short attack of a few days, yet I have failed to convince myself, either by experience or reading, that any such important discovery has as yet been vouchsafed to us, as one calculated to destroy the venom of the typhus poison, and to check its ravages.

All the cases in which it has been said that typhus has been cut short, as by a very large bleeding at the outset, or by free vomiting, or by some other means, are fairly open to the strong suspicion, if not the charge, of erroneous diagnosis. It is plain, if you think on the subject but for a moment, that, without an exact diagnosis, this question of the early curability of typhus cannot be settled. Now, those who have seen most of this and other maladies know best how difficult, nay, how impossible it often is, in the first week or ten days, to predicate with certainty of this or that case, that it is typhus fever. And, therefore, if you deal candidly with yourselves and others, you must not affirm that you can cut short and cure typhus, unless you have the most unequivocal evidence that the cases in question have been examples of that disease.

If these views be correct, you will perceive the necessity, when you come to treat a case of this nature, of not wasting time in trying this expedient and that medicine, but you will apply yourselves to provide for the due care and watching of your patient, and the careful administering to his wants and necessities. In this respect the poor, who are inmates of our public hospitals, have often a great advantage over the patients we have to treat in private practice; for here we have trained attendants, always ready, experienced in the management of cases of this kind, and accustomed to obey orders. In private practice, we are too often obliged to trust to the timid and inexperienced nursing of relatives and friends, or perhaps of servants already over-burdened with other duties; or, if we do succeed in overcoming prejudices, and in inducing the friends to procure the assistance of a nurse, it is too often the case that she is accustomed only to act as a lying-in nurse, and has no experience in

fever cases. I would gladly read for you here the remarks of the late Dr. Graves, one of the greatest authorities on the subject of the pathology and treatment of fevers, on the choice of a nurse in cases of this kind; but I must content myself with referring you to the first volume of his valuable work on clinical medicine, where you will find them in the ninth lecture.

Case XVI. (vol. xxxii. p. 75).* And now for the particulars of the case. The patient was a man named John Gavin, thirty-two years of age, a large bony man, of strong build. He lay in Rose Ward. He is a printer, and had just come from Edinburgh to look for work in London. His illness probably commenced in Edinburgh, and developed itself immediately on his arrival in London. It is often extremely difficult to fix precisely the day on which a fever began, partly from the imperfect recollection of patients, and partly because the symptoms often develop themselves so insidiously and gradually, that the patient cannot note exactly the time when he really began to be ill; he feels for many days languid and out of sorts, but is still able to get about, and, unless some such prominent symptom as rigor has occurred, it is impossible to name one day more than another on which the fever began. Now, what we gather is this, —that on or about the 9th, as he was leaving Edinburgh, he caught cold, of which he has no other evidence than the existence of great languor and weakness, with a strong sense of fatigue upon the slightest exertion. On his arrival in London, he found himself quite unequal to the task of looking out for work, and unable to follow his business if he had succeeded in securing employment.

All this looks very much as if he had caught the infection in Edinburgh, where, we know, fever is always more or less rife among the lower orders; its period of incubation being the day or two before he left that city, and the first few days after his arrival in London. During the first week of his arrival in town the sense of languor increased, and he felt very ill. On the 16th of January, 1851, sore throat came on, and he was attacked with several severe rigors, succeeded, on the 17th, by increased

* Reported by my clinical clerk, Mr. J. H. Sylvester.

debility, vomiting, headache, and tinnitus aurium. On the 18th these symptoms had increased in severity, and his friends stated that he became stupid, and appeared as if drunk, and at times he wandered a little. It was, then, on the 16th, that the more decided symptoms of fever had developed themselves, although we cannot doubt that the poison had already begun to work in his system at least seven or eight days before that date.

He was admitted into the hospital on the evening of the 19th of January. On the 20th, the following report of his condition that day was entered in the case-book:—"The patient is very thin and weak; has a dull vacant look; is delirious, incoherent, and it is not without great difficulty that answers to questions can be elicited from him; he is, however, very quiet and lies chiefly on his back; respiration hurried; crepitation audible all over the posterior surface of both lungs, especially at their bases; the tongue is dry, but not coated; slight sordes on the lips and teeth; the abdomen slightly prominent and tympanitic; has had one loose motion in the night; no spots are observable; pulse 130, very compressible; respirations 44. He was ordered half an ounce of brandy with beef-tea every two hours, and five grains of the sesqui-carbonate of ammonia, with half a drachm of chloric æther, in an ounce and a half of water, every six hours, and turpentine stupes to be applied freely to the back."

On the 21st, his symptoms had not changed, and the pulse was 128; the respirations 40. Reckoning from the occurrence of the rigors on the 16th, our patient must have been, at the earliest, in the fifth day of the fever,—it might be the seventh or eighth. The description I have just read to you portrays, very accurately, the condition and the symptoms of a patient labouring under the most common form of continued fever now met with in London and our other great towns, about that period of the disease,—that is, not earlier than the fifth day. Now, from this time, the symptoms usually continue of much the same character, with more or less of exacerbation, till the seventeenth or eighteenth, or to the twenty-first or even the twenty-eighth day: the most important being those referrible to the nervous system—coma or delirium; to the lungs, congestion, or even pneumonia and pleurisy, which are less frequent; and to the bowels, the diarrhœa.

When a case is about to terminate favorably, these symptoms gradually give way;—the pulse exhibits no tendency to quicken, but rather to fall in frequency; the bowel affection appears easily controllable; the tongue begins to clean at the tip and edges; the patient becomes less stupid; the comatose or delirious state diminishes; the pulse improves in quality, and the general powers of the patient experience a gradual but manifest change for the better. These changes commence generally in or about the third week.

But if the case is not about to end favorably, we shall find an aggravation of some of these symptoms about this period. The pulse will increase in frequency, and its power will be much diminished; the delirium and other head symptoms will become more alarming; or the symptoms referrible to the lungs may become more severe—the breathing more rapid and feeble, and the bronchial tubes impeded by mucus, which the patient has not sufficient power to expel, and, in consequence, death may result from a slow asphyxia; or he may be run down by the constancy or profuseness of the diarrhœa, and perhaps by hæmorrhage from the bowels.

Now let us see what was the further course of the symptoms in John Gavin's case.

On the 23d of January he had in some degree recovered the exhaustion caused by his removal to the hospital. His pulse had fallen to 112, but the respirations continued at 48. He was purged four times in the day; the chest signs remained the same.

An enema of starch and opium was ordered at night to counteract the diarrhœa, and his brandy, ammonia, and beef-tea were continued as before. The motions became less frequent, and he remained without any change till the 27th.

On this day we found the bowels with a tendency to be loose again; three motions in the day; abdomen tympanitic; pulse 120, and respirations 52. Many of you will remember, that I pointed out to you on this occasion a good mode of estimating the real power of the pulse in fever and other asthenic states, namely, by causing the patient to sit up in bed, and comparing the condition of the pulse in this semi-erect posture with its state in the horizontal position. It was not accelerated by the

change from the horizontal position, but its strength and volume became most strikingly diminished; it became very small, and much more compressible, but immediately he returned to the horizontal position it recovered itself.

There cannot, I apprehend, be a more palpable or unequivocal sign of an enfeebled circulation, than this marked deterioration in the quality of the pulse, on the patient's assuming the semi-erect from the horizontal posture. It indicates very clearly how dangerous it is to remove patients in fever, or other low diseases, from one place to another, or to allow them to move themselves; and how necessary for them it is that they should be constantly attended upon, that every, even the slightest, exertion on their parts should be prevented as much as possible.

It was now evident that what we had chiefly to deal with was the extreme debility, and the looseness of the bowels. The state of debility was the more fearful, inasmuch as it had come on notwithstanding the free use of stimulants; for since the 21st he had been taking brandy, at the rate of half an ounce every hour. I now doubled the quantity of brandy, and ordered the ammonia and chloric æther to be taken in an ounce and a half of infusion of rhatany every fourth hour.

For the two days (28th and 29th) following this increase of the stimulants, he continued much in *statu quo*—the pulse 129; respirations 50; the purging diminished, so that he had only one stool in twenty-four hours. The rhonchus in the chest had increased, however, and the heart sounds were very feeble, so that I felt it needful to increase the stimulant to five drachms every half-hour, or thirty ounces in the day.

On the 30th there was some improvement: he was more conscious, the breathing was more free although still rapid, 50; the rhonchus somewhat less, but the pulse was still 120; he had one loose stool, and the belly was soft.

On the 31st, a still more sensible improvement had taken place. He was much more conscious; the rhonchus was less; vesicular breathing became much more distinctly audible in the lungs; the pulse had fallen to 112, and beat at this rate in the semi-erect as well as in the horizontal posture, although in the former it became reduced in power; the respirations were 46, and the heart's action stronger. No movement of the bowels.

On the following day, the 1st of February, the pulse was down to 100; respirations 45. The tongue was evidently cleaning; the heart's action was stronger; he coughed a good deal, and was rather drowsy. One loose stool.

On the 2d matters were much the same; pulse 100.

On the 3d a much more decided improvement had taken place than had yet been observed. The pulse was only 84, and the respirations 38; he was more conscious; the rhonchus was less, and he breathed more freely; the tongue was clean, the abdomen soft, and the bowels quiet.

So far, then, we were in excellent spirits respecting our patient. All the most important symptoms had improved under the high degree of stimulation to which he was subjected; and of these improvements, none was more important than the reduction of the pulse in frequency at the same time that it acquired more power. The least change for the better was found in his consciousness: although he took more notice than before, and was less deaf, and answered questions more readily, he was still very heavy and stupid.

The continuance of this state of stupor led me, on the 3d, to reduce the quantity of his stimulants by six ounces, so that he now took an ounce every hour instead of ten drachms. The chloric æther was omitted. From this time, I regret to say, "a downward tendency" (to borrow a mercantile phrase) became evident; the crepitation in the lungs increased, and he began to expectorate a large quantity of thick purulent fluid; his stupor did not diminish; and the pulse and respirations became each day more rapid than the previous one.

On the 4th the pulse was 116; the respirations 46. On the 5th, pulse 120; respirations 52. On the 6th, pulse 138; respirations 52. On the 7th, pulse 140; respirations, 52. And these changes took place, notwithstanding that the largest quantity of stimulants was again administered, and that the infusion of serpentary was substituted for rhatany, with increased quantities of ammonia and chloric æther.

On the 7th a very serious symptom showed itself, which in part explained the rapid declension of his powers. This was hæmorrhage from the bowels. He passed on that day a large quantity of blood by stool, which evidently exhausted him very

much. Turpentine was now administered in small and frequent doses, but on the 8th he passed some more blood. He was now evidently sinking, with an extremely rapid pulse and very quick breathing, and he died on the morning of the 9th, which must have been the thirtieth day of the fever.

Here, then, was a case in which no pains were spared to save life, so far as diligent treatment and careful nursing could accomplish that object; it terminated, however, unsuccessfully, and the patient died evidently in a state of extreme exhaustion. The treatment consisted in the early and free administration of support and stimulants, and in the use of counter-irritation over the chest and abdomen; turpentine stupes were used daily to the front and back of the chest for some time, and afterwards large blisters were applied, and the abdomen was occasionally stuped with turpentine. Close attention was paid to the state of the bowels; astringents were given constantly; and, on one occasion, when the diarrhœa appeared most threatening, an opiate enema was administered; thus the tendency to looseness of the bowels was kept so completely under control, that his weak state could not have been attributed to this. He was supported by a full allowance of strong beef-tea, besides milk and arrow-root, and stimulants were given in large quantity, as I have already described.

Now, it behoves us to inquire, why did this patient die? Was there here the *nimia medici diligentia?* Were the quantities of food and stimulants too much for him? Was there any other treatment which we did not use, but which we ought to have had recourse to? Or did death result from causes clearly beyond the control of all medical interference?

The *post-mortem* inspection showed that the morbid changes were limited to the chest and abdomen. In the former there was congestion of the lungs; but to an extent decidedly less than we had expected. The bronchial tubes, however, contained a considerable quantity of the thick yellow purulent matter which he was expectorating during the last few days of his life.

But the most serious lesion was in the intestines. The lower part of the ileum contained numerous deep ulcers, some of which had eaten through the coats of the intestine so as almost to perforate. These ulcers were placed on the free margin of

the intestine, and occupied the position of Peyer's patches. In the lower three feet of the ileum we counted as many as seventeen ulcers, some of which were larger than a shilling. The floors of some consisted only of peritoneum and a little lymph. One very large ulcer existed on the iliac side of the ilio-cæcal valve. In addition, several of the solitary glands were enlarged, and some ulcerated, and the mesenteric glands were enlarged.

I need hardly say that, from our experience of cases of this kind, and from the diarrhœa, controllable although it was, and the tympanitis, and the hæmorrhage ultimately, we were quite prepared to find ulcerative disease in the intestine; although, owing to the mildness of the symptoms referrible to the bowels, we might well be surprised to find such large ulcers, and so many of them.

This extensive lesion of the mucous membrane of a part of the intestinal canal so important to nutrition as the ileum must have contributed mainly to the state of prostration of this patient, which persisted for so long a time, notwithstanding the abundant supplies of nourishment which were given him. And yet it is difficult to explain precisely how these ulcerations could have occasioned all this debility, inasmuch as there was no excessive diarrhœa, no great drain from his system, nor did they interfere with the due digestion and absorption of his food; for the quantity of the fæces formed was not unusually great, nor out of proportion to the amount of food taken. It is plain enough that, notwithstanding the disease in the ileum, gastric and duodenal digestion, and chylous absorption in the jejunum, must have gone on sufficiently to admit of the appropriation of the greatest part of the food given.

It cannot, then, be said that this patient had too much food; if he had, surely we should have found in the bowels large quantities of fæces and portions of undigested food, and during life there undoubtedly would have been flatulence and distress, referrible to the stomach, and other signs of indigestion, none of which existed. Nor can it be said that he had too much stimulant; for we had this most striking fact, that with the increase of stimulants the pulse on successive days fell from 120 to 84, and that with their diminution it rose again to 120 and

130. Under the highest stimulation, all the symptoms improved; the chest became more free, the head clearer, the fever less, the tongue cleaner. It was quite evident that both the food and brandy were fully digested and absorbed. We cannot, therefore, plead guilty to the charge of *nimia medici diligentia*. And, on the other hand, I am not aware that anything else could have been done for him besides that which was done. I know of no medicine or remedy more applicable to his symptoms and morbid condition than those which we used. There are those who place great confidence in the powers of mercury to promote the healing of such ulcers as this man had in his ileum. I confess my faith does not carry me so far; and I think most practical men now-a-days would eschew the use of mercury, where they had reason to believe that the small intestine was ulcerated, or likely to become so.

The rapid change for the worse which followed the hæmorrhage from the bowels indicated sufficiently that that was the *immediate* cause of death. If the hæmorrhage had not taken place, there can be no doubt his life might have been prolonged a few days. But the small quantity of blood lost was quite insufficient to cause death, if there had not previously existed a state of great depression. I have frequently seen much more blood passed by patients who have afterwards perfectly recovered.

I repeat, that were it not for our experience of the constant accompaniment of a state of prostration with a few ulcers of the small intestine, it would be impossible to believe that so grave an effect would follow such a cause. It is true that in this patient the ulcers were not few, but they were found in but a small portion of the intestine, namely, in a space three feet in length, leaving twenty-seven feet of the highest part of the bowel intact. I have, however, seen a state of as great, if not greater, prostration, where there were not more than four ulcers. What seems most essential to the production of this state of prostration is, that the sloughing and ulcerative process should be quick, and that it should be perforative in its tendency; that is, that it should eat quickly through the tunics of the bowel, as was the case with Gavin, in whom we found, that at several points the coats of the bowel had been so eaten through as to leave only a little lymph and a thin film of peritoneum as their floors.

But the ulcers are not the only mischief existing in connexion with the bowels in these cases: the mesenteric glands are likewise diseased, swollen, and evidently irritated by some abnormal matter passing through them. No doubt the state of these glands interferes with due chylous absorption, but still scarcely sufficiently so to account for the prostration, for the food is freely absorbed in the upper portion of the bowel, and a good deal of it is of a nature (as the oily matter of milk) which must assume the state of chyle, before it can be absorbed.

It seems to me that the production of this state is due not so much to imperfect appropriation of food, as to the absorption of a matter from the ulcerated surfaces, which, circulating with the blood, exercises a poisonous and depressing influence on the system: a matter of the nature of, if not identical with pus, which is absorbed by the lacteals, and perhaps also by the blood-vessels, but probably chiefly by the former, by which route it quickly reaches the lungs, without passing through the liver, where it may contribute to the increase of the bronchial congestion and irritation which so constantly accompany this typhoid state. This view I have often broached to you already at the bedside of patients suffering in this way.

I show you here a preparation which was put up for me some time ago by Dr. Lionel Beale.* It exhibits a few well-marked deeply-perforating ulcers of the ileum, having much the appearance, from the thick, swollen, and red margins, that the process of sloughing and ulceration was a quick one. In this case (the patient was a young woman), the fever ran its course in about three weeks, the diarrhœa was almost none, and the chief symptoms were a tympanitic abdomen, stupor (in fact coma), bronchial congestion, and extreme prostration. A short time ago you may remember a woman of the name of Lock, who went off very quickly likewise with similar symptoms, the stupor being so great that I was afraid that a few drops of laudanum, administered with starch, to check diarrhœa, had narcotized her. There was in this case, in addition to the stupor, bronchial

* The patient's name was Ada Dacon, and the particulars of the case will be found in the next lecture, Case xxviii. This specimen was preserved in naphtha and creosote solution, in 1850, and it now retains its colour, and exhibits all the characters it had when recent [Ed.].

congestion and prostration, but the diarrhœa was very slight, and readily controllable.

Now, that the absorption of pus is capable of producing these depressing effects we have many proofs.

First, in puerperal fever. In some cases the absorption seems to take place rapidly, and in large quantity, and, under such circumstances, the patient succumbs in a few hours from rapid prostration and pulmonary congestion, with more or less stupor. In other cases, the absorption seems more gradual, the typhoid condition is induced more slowly but very completely, and, after a time, purulent deposits are found in the joints and muscles or elsewhere.

Secondly, in cases of erysipelas, in which the suppurative process is rapid, we have typhoid and comatose symptoms, which are out of proportion to the extent of lesion; in such cases doubtless pus finds admission into the circulation.

Thirdly, we sometimes have unequivocal evidence of the absorption of pus, as well as to the source whence it comes, as with respect to the secondary deposits. I remember attending a case in private practice, where the pus showed itself in the anterior chamber of the eye. This case presented all the symptoms of typhus fever; and for a day or two I viewed it as such. One day I was much surprised at observing pus in the anterior chamber, which increased in quantity very rapidly, and pus was afterwards found in the elbow and shoulder joints. When we came to examine this patient, we found an ulcer in the heart, at the base of one of the mitral valves. Some years ago, we had a case in the hospital of a woman who was suffering from chronic bronchitis; she suddenly became typhoid, and I looked upon it as a case of most aggravated character. She died in a few days, and we found an abscess in the septum of the heart, which had burst, and thus the pus had entered the very fountain of the circulation, producing symptoms nearly resembling those which come on in a case of low typhoid fever.

There seem, then, sufficient grounds for explaining the prostration and fatal termination in Galvin's case, without ascribing any ill effects to either what had been done for him, or to what had been left undone. The sloughing and ulcerative

process undoubtedly interferes, to a certain extent, with the function of the bowels, but it also furnishes a source of formation of a poisonous matter, which, we know by experience of analogous cases, when taken into the system, creates symptoms of the same character as those of these fatal instances of typhoid fever.

There is another mode of termination of these cases of typhoid or enteric fever for which you should yourselves be prepared, and for which you should prepare the friends of the patient when you may see sufficient reason to apprehend it: I mean, that by perforation. One of those films of peritoneum, which I have already alluded to as forming the floor of many of the ulcers, gives way, and the contents of the bowel pass into the peritoneal sac. In some cases of long duration, when the patient seems to have struggled, day after day, against the assaults of death, rapid sinking immediately follows the perforation, and, indeed, signalizes its occurrence. No new pain is felt, but the patient grows rapidly weaker; the pulse, too, fails, becomes rapid and fluttering, and death from exhaustion or fainting quickly ensues. In other cases the occurrence of the perforation is ushered in by severe pain in the abdomen; sometimes vomiting; tenderness and pain on pressure; tympanitis; with also increased prostration, all, signs of peritonitis induced by the irritating influence of the intestinal contents upon the peritoneum. When these latter symptoms make their appearance, the free exhibition of opium, in large and frequently repeated doses, is the only measure to which the practitioner can have recourse with any hope of success.

The following case will illustrate the last-mentioned mode of termination, although the direct evidence of perforation by a post-mortem examination was not obtainable.

Case XVII (vol. xxxvi, p. 97).—Robert Neek applied as an out-patient at King's College Hospital on the 24th of July, 1852. He was then very weak; his tongue tremulous, coated with a brown fur, and deeply fissured; he was also suffering from diarrhœa. He persisted in following his occupation as long as he could, and was therefore not admitted as an in-patient until the 31st; he was then deaf and completely

prostrate. Brandy and beef-tea were ordered, and some days later chloric æther and rhatany.

There was no great change in his condition for about a week; his pulse continued high, his bowels more or less relaxed, rhonchus and sibilus were heard over the chest, and on the 5th and 6th there was slight delirium.

On the 8th there was a considerable fall in the frequency of the pulse; and the record of the 9th is as follows:—"Tongue much cleaner; he feels altogether better; the cough has quite left him; the bowels are no longer relaxed."

On the 12th he was still doing very well, and was ordered a slice of mutton. He continued to make favorable progress until the night of the 18th, when he complained of severe abdominal pain. On the 19th all the symptoms of peritonitis were present,—he was lying with his legs drawn up, and the whole abdomen was exquisitely painful on pressure. A grain of opium was given, and ordered to be repeated.

He passed a delirious, restless night, the same symptoms continuing, and died on the 21st.

Had our patient Gavin not been carried off by the exhaustion consequent on hæmorrhage and purulent infection, it is very probable, from the state of the ulcers, that perforation must have taken place, of which he would have died in either of the two ways which I have described.

A third mode of termination is by colliquative diarrhœa. The patient may be going on well, and the practitioner may even be sanguine in his expectations of a favorable result, when the diarrhœa may suddenly become colliquative, and a few discharges of large watery evacuations will terminate the case.

But to return to the treatment of the patient Gavin. It may be said, surely the irritation of the bowels was kept up by all the stimulants (to say nothing of the food) which were given, and had they been more sparingly supplied, the ulcerative process in the ileum would not have gone so far.

This notion respecting the injurious effects of alcoholic stimulants, in cases where there is a tendency to bowel affection, is, I think, partly founded upon a vague supposition that the alcoholic fluid comes in direct contact with the irritable mucous

membrane. Now the reply to this is that we have the strongest reason to conclude, that fluids of this kind never, except when taken in very large quantity at one time, pass the pylorus, but are absorbed by the walls of the stomach. This is especially the case when they are administered in the way I recommend, —that in which they were given in Gavin's case,—namely, in small quantities, with intervals of not less than half an hour between each dose. Thus one dose is absorbed before the next is given.

But it may be urged, that the alcohol gets into the blood, circulates with it, and so increases the tendency to ulceration.

Upon this point we can only appeal to experience. The administration of alcohol to healthy persons does not prove injurious by any irritative effects it may produce on the bowels. Of all the ill consequences which the advocates of the teetotal system, in their most praiseworthy zeal, have summed up as likely to be caused by the use of alcohol, I do not find that diarrhœa or ulceration of the bowels is noticed; and were it a frequent effect, it certainly would not have escaped the scrutiny of these gentlemen. It is true that a debauch, in which a man may drink at one sitting as much, or considerably more than we should think of giving in twenty-four hours, may sometimes disturb the liver, and, through its increased secretion of bile, the bowels; but the looseness thus excited seldom or never proves otherwise than salutary.

Nor do we find that effects of this kind are apt to follow the liberal administration of alcoholic stimulants in other low diseases; in erysipelas; in the diffuse inflammation of the areolar tissue, whether traumatic or not; in puerperal cases; and we give it repeatedly in cases with threatened or actual ulceration of the bowels, without any increase, but, on the contrary, a marked diminution of the unfavorable symptoms. Such, indeed, was the case with our patient Gavin. On the first few days of his taking stimulants, a manifest improvement took place in all his symptoms, those affecting the bowels as well; so much so that until the post-mortem examination revealed the true state of matters, I blamed myself for diminishing his supply of stimulants on the third. Probably the good

effects continued until the puriform matter had entered the circulation in sufficient quantity to produce its poisonous effects.

I could enumerate many instances in which this mode of treatment, by free stimulation, was of great and signal advantage. But I must content myself with mentioning a few, referring particularly to some cases of this kind which have lately been treated in the hospital.

Case XVIII* (vol. xxxi, A. p. 54).—Many of you will recollect the case of Lucy Wood, aged fourteen, who was in the house about three months ago. She took as much as an ounce and a half of brandy every hour for three days together, and for the next fortnight half an ounce was hourly administered; this latter quantity, however, being sometimes much increased as occasion required. Under this large amount of stimulants, her symptoms gradually improved, and she was discharged quite well on the 4th of December, having been about nine weeks under treatment.

This girl laboured under the great disadvantage of heart disease. A loud systolic bellows-sound, heard most distinctly at the apex of the heart, was present when she came in, and resulted from an attack of rheumatic endocarditis, which occurred some time ago. She was admitted on September 26th, and on October the 4th her symptoms began to assume a very severe character, and she was evidently getting very low. At this time she was also suffering from diarrhœa, for which she was taking astringents with chloric æther, and on one occasion it was thought advisable to administer an opiate enema.

On November 1st, she was in a state to warrant us in diminishing the quantity of stimulants. The pulse had now fallen to 120; on the 4th it was 114. After she had recovered from the fever, a very painful node formed upon the anterior surface of the tibia, which ultimately did perfectly well.

Case XIX† (vol. xxxi, A. p. 57).—John Bigg, fifteen years of age, was admitted with fever, on the 3d of September, 1850.

* From the notes of my clinical clerk, Mr. Simpson.

† Reported by Mr. Simpson.

The attack had begun with shivering and the other usual symptoms five days before. He soon became delirious. A stimulating plan of treatment was commenced at once.

On the next day, the 4th, his nose bled and looseness of the bowels came on; the urine contained a little albumen; the pulse numbered 112. Chloric æther and krameria were given, and in the evening the wine was increased to between five and six drachms every hour.

On the 5th rose spots were developed: the purging continued, and about a pint of blood was passed at stool. Enemata of starch and opium were administered, and ten minim doses of turpentine were given every fourth hour. In the evening brandy was substituted for wine. There was then great prostration, muttering delirium, and cold feet; and as he had not slept since admission, ten grains of Dover's powder were given. The next day his pulse was 150. All the same symptoms continued, and there was still blood in his stools. During the 8th, 9th, and 10th the symptoms were the same, but less severe. He complained, however, of great abdominal pain and tenderness, for which turpentine stupes were repeatedly applied, and the brandy increased to an ounce every hour. The albumen disappeared from the urine.

On the 11th, a little blood again appeared in the motions.

On the 12th there was a slight sweating, apparently critical. On the 13th, he was much better, and from that day continued to improve. The brandy was reduced gradually, and on the 24th he was convalescent and on full diet.

Case XX* (vol. xxxi, A. p. 67).—Elizabeth Bevan, thirty-six years of age, an over-worked needle-woman, was attacked with the ordinary premonitory symptoms of fever, September 15th, 1850, and admitted in a semi-conscious state four days after. Half an ounce of brandy was given with strong beef-tea every hour, as well as chloric æther and ammonia; and turpentine fomentations were applied to her belly and chest.

The next day she lost about a pint of blood by stool, and became very pale and low. Pulse 122; respiration 33. Tur-

* Reported by Mr. Simpson.

On the 13th she seemed worse: her pulse was very feeble and thrilling to the finger, and had fallen to 84. The respiration was more embarrassed, and, on auscultation, large crepitation could be heard all over the chest. She had ceased to cough and expectorate—apparently from want of power.

On the 14th she appeared sinking: she could hardly speak, and her pulse was scarcely perceptible. A blister was ordered. She died in the evening.

At the post-mortem inspection, the lungs were found slightly congested and the tubes much choked with secretion, but otherwise healthy.

The heart was flabby and somewhat soft.

Petechial extravasations of blood were found among some of the muscles of the chest, more or less symmetrically situated on either side.

Neither Peyer's patches nor the solitary glands were found diseased.

The spleen was a good deal softened, presenting much the same appearance as in the patient Charles Andrews (Case xxxi).

Case XXIII* (vol. xlvii, p. 15).—George Rose was admitted May 10th, pale, exhausted, and only partially conscious, but with a rapid feeble pulse, quick respiration, and hot skin. He had felt languid and weak for a fortnight, but had given up work only five days. Blood had been passed both by stool and urine.

During the short time he survived his admission, two motions were passed approaching to a pitchy blackness, and one of a blood-red colour, and also bloody urine. He was restless, moaning, and delirious. Towards evening the exhaustion increased rapidly, and he died early on the morning of the 11th.

At the post-mortem examination, the patches of Peyer were found ulcerated, especially at the lowest part of the ileum. Many of them were covered with a red fungous mass; others simply enlarged and injected.

I have felt it a duty to make these remarks to you upon the

* Reported by Mr Hardwich.

subject of the treatment of fever by stimulants (and they apply no less to the treatment of other exhausting diseases—erysipelas, influenza, bronchitis, carbuncle, &c.), because I wish to caution you against the morbid fear of over-stimulation, which leads many to adopt an opposite or a vacillating course, and to allow their patients to die from exhaustion. This is the mode of death to which fever patients are peculiarly prone; and I hold that the lower you allow them to become at first, the more likely is the ulcerative process in the intestines to take head, just as it is apt to do in the bowels and in the cornæa of the eyes, in cases where there is an insufficient supply of properly nutritious food. At the same time, I must beg that you will not run away with the notion, that every patient in fever about whom you may be consulted is to be treated with thirty ounces of brandy a day. There are many cases in which no stimulant at all is necessary; others, again, in which it is not needful to give more than four or six ounces a day. You must bear in mind that we have two classes of cases of fever to deal with, the mild and severe; or those which have had a large, and those which have had a small dose of the peculiar poison on which the febrile state depends. Where a large dose of the poison has been received into the system, you will generally find it necessary to give large quantities of alcoholic food, or the patient will not have sufficient vital power to resist its depressing effects. Some few instances, indeed, there are, in which the dose of the poison is so large, that the patient never rallies from the state of almost complete paralysis which it induces; such cases run their course in twenty-four or forty-eight hours, or within a week. The case of S. A. Chandler (p. 115) was of this kind. But the mild cases—and fortunately in many epidemics these are the most numerous—do perfectly well on a very moderate amount of nourishment, with little or no alcohol.

The objections which some excellent practitioners have to the use of stimulants apply with more justice to the slovenly mode in which they are too often given. Generally left altogether to the discretion of a nurse, they are given in large doses at one time, or with other food, or without reference to the medicines which are being likewise administered; they consequently create

derangement of the primary, or stomach, digestion, flatulence and flushing. If you give alcohol, give it with due regard to its digestion by the stomach, and so as not to interfere with the other food or the medicines likewise being taken.

I am convinced that it is much better to err on the side of over-stimulation than not to give enough; for if we have over-stimulated a patient it is very easy to pull him down again; there are plenty of appliances and means for this purpose; but if the patient sink too low, nothing is more difficult than to restore him. If, by your feeding and stimulating, the thermometer of life has risen to too high a point, nothing is easier than to depress it; but if fallen below a certain point, then to raise it again, much more to restore it to the height from which it fell, "hic labor, hoc opus est."

In conclusion, let me say a word or two as to the treatment to be pursued when you have reason to fear that the bowels are ulcerated. It seems to me, that the great principle of treatment in such cases is to keep down peristaltic action, which is best done by opium and astringents containing tannic or gallic acid. Many attach great value to the use of sulphate of copper; but as it is generally given with opium, and does not always agree without opium, I think the latter drug has the largest claim to the good services often done by the combination. When hæmorrhage occurs, nothing is so effectual to restrain it as turpentine given in small doses, so as not to risk offending the stomach; even so small a dose as five minims is often sufficient; and I frequently apply it externally, as a stupe to the walls of the abdomen, with decided benefit. In dealing with these cases, you must not be timid as to allowing the bowels to remain inactive for even several days. I have never seen any bad consequence from their not acting even for four or six days; and when they are to be provoked to act, let that be done by some simple enema rather than by aperient medicine.

LECTURE VII.

ON CERTAIN ACUTE DISEASES.

CONTINUED FEVER.

In offering to you to-day some remarks on those forms of continued fever which are most likely to come before you, let me first say a word or two respecting the intimate nature of the most prominent clinical feature of the disease before us, from which indeed it derives its name—I mean *fever;* for it is of great practical importance to have something like definite views upon this point of pathology.

You all know that *fever* is marked by a hot, sometimes burning, often flushed, and generally dry skin; by a quickened pulse, loss of appetite, thirst, accelerated respiration, and more or less loaded urine; and these phenomena soon become accompanied by a manifest wasting of substance and loss of power.

We can best explain these symptoms by supposing that a poison, circulating in the system, interferes with and greatly modifies the processes of nutrition and secretion; what Dr. Prout has called the *secondary destructive assimilation* is exalted, and the elements of the tissues seem to undergo a rapid oxidation. The result is a rise in temperature throughout the systemic capillary circulation, general wasting, and more or less rapid exhaustion of vital power.

Now symptoms of this kind always follow the introduction of a poison into the system, and are indicative of a peculiar disturbance which the presence of that foreign matter in the blood establishes. You have every day the experiment, performed millions of times, of introducing into the blood a minute quantity of vaccine lymph, through a puncture in the arm. This, in a few days, establishes a definite form of fever, with certain local phenomena in the shape of one or more pustules at the seat of the wound. So if the analogous poison of small-pox gets into the system, as we call it, or more correctly into

the blood, a definite fever is established, with the local development of peculiar pustules on the skin and mucous membrane. The same may be said of all the fevers which we call *exanthemata*. In each there is a definite poison, and that poison produces definite febrile phenomena. If left to itself, the fever begins on a certain day and ends on a certain day, and affects the skin or mucous membrane, either gastro-intestinal or respiratory, or even genito-urinary, in a specific manner. Take, for instance, the poison of scarlatina: it quickly establishes an intense fever; it attacks the mucous membrane of the throat and that of the kidneys; it develops a peculiar rash on the skin, and more or less of irritation and swelling of the cervical glands. In such cases, although the great intensity of the fever is in the early stages, it nevertheless continues more or less in a chronic form until all local phenomena have disappeared.*

What the pathological significance of the local phenomena of fever may be, we are scarcely yet in a position to declare; but it seems very probable that they have at least much to do with the process of elimination of the poison. Each poison has apparently an elective affinity for some particular structure or organ, and through it makes for itself a channel of escape out of the system. Thus it seems very reasonable to suppose that the cutaneous desquamation, which so often occurs in scarlet fever, is one medium for the extrication of at least a part of the poison from the blood. In like manner the pustules of small-pox are, in all probability, due to a nisus of elimination, and each pustule is a point of exit of a certain quantity of poisonous matter.

When there is so great a diversity of symptoms, it is not too much to suppose that the poisons, upon which these various forms of fever depend, are also essentially different from each other. There are, in fact, as many poisons as there are fevers. And the greatest number agree in this, that they give rise to a febrile state which is continuous, or, according to the medical term, *continued*, until it exhausts the power of the patient, or if his strength will permit, until the poison is eliminated.

* Professor Parkes' invaluable Lumleian Lectures on Pyrexia deserve careful study by all who take an interest in the pathological phenomena of fever. —*Medical Times and Gazette*, March 17th, 1855.

One poison is distinguished by the extraordinary peculiarity that after infecting the system for a certain time, giving faint or no indications of disturbance, a form of fever is engendered which is distinguished by more or less complete remissions of the febrile state. These occur periodically, and form part of a peculiar train of phenomena, consisting of a cold or shivering state, a hot febrile state, and a sweating state or stage, out of which the patient gradually passes into a non-febrile state or one of apyrexia, and remains quite well, until an interval of twenty-four, or forty-eight, or seventy-two hours has passed by, when the same train of phenomena will be repeated.

It would almost seem as if the marsh or paludal poison, upon which this fever depends, underwent, with varying rapidity, some increased development, at the acme of which the peculiar three stage phenomena come on; these subside with the elimination of a certain portion of the poison from the system by the sweating process, to be renewed when in due course a fresh development of the poison takes place.

It seldom happens that the marsh poison, once admitted into the human system, ever becomes perfectly eliminated from it; and persons once infected are, for this reason, ever after liable to renewed attacks, under even the slightest malarious influences.

When that curious compound pus, a product of disintegrated tissue, enters the current of the circulation, it engenders a peculiar fever, of which the phenomena are increased heat of skin, accelerated pulse and respiration, and depression of nervous power, sometimes so great as to kill very quickly by sheer exhaustion. But in most cases the fever persists, and soon signs of elimination show themselves in local collections of pus in various parts of the body. After these have been evacuated, if the vital powers of the patient are sufficient to bear up against the trying and exhausting process, often of tedious duration, recovery takes place.

I must limit my remarks on this occasion to the subject of *continued fever*. Of this, it may now be fairly admitted that there are three varieties, as proved by the excellent researches of Stewart, Jenner, and others in this country, America, and on the continent. These are the typhoid, typhus, and relapsing

fever, each produced by a distinct, although doubtless very similar poison. As the relapsing fever is comparatively of rare occurrence, I shall confine myself to the *typhoid* and *typhus* varieties.

The term *typhoid* is applied to that kind of continued fever which is accompanied by catarrh, diarrhœa, or a tendency to it, and more or less abdominal tenderness and tympanitis; and in which, after death, we find a morbid condition of the solitary glands, and of Peyer's patches in the ileum, amounting sometimes to irritation and enlargement only, in other cases to sloughing and ulceration. Another feature, characteristic of typhoid fever, is the development of a peculiar eruption of circular, slightly elevated, rose-coloured spots, often of considerable size, which fade or vanish momentarily under pressure; these make their appearance from the fifth to the twelfth day, or even later, and are generally confined to the chest, belly, or back.

The appellation *Typhus*, on the other hand, is applied to those cases in which the symptoms of intestinal irritation are absent, and which are marked by a copious eruption, consisting of small, irregular, reddish or purplish spots, which generally run together so as to form irregular or crescentic patches, not confined to the chest and abdomen, but often to be found on the extremities, and indeed covering almost the entire surface, and on the whole, nearly resembling the eruption of measles.

The case of Gavin (Case XVI), which I detailed to you in my last lecture, is a good example of the Typhoid form of fever, excepting that the rose spots were not developed. The following case is, in some respects, a better illustration, as the catarrhal symptoms, the rose rash, and the diarrhœa were all well marked; it is also a good example of the treatment which I advocate in these cases.

Case XXIV* (vol. xxxix, p. 143).—M. A. Copstock, a nursemaid, eighteen years of age, was admitted into King's College Hospital, February 5th, 1860. Her illness commenced eight days before, with pains in her limbs, which she attributed

* Reported by Dr. Plowman.

to some trifling exposure to cold and night air. Carbonate of ammonia was administered in five-grain doses every third hour.

On the third day after admission, her condition was as follows :—

Her face looked puffed and heavy, her eyes suffused; she complained of headache, and seemed confused and drowsy; the skin felt hot and dry, and the tongue was coated with a whitish fur. There was a troublesome cough, with expectoration of a scanty viscid mucus of rusty tinge. On listening to the chest, slight rhonchus and crepitation were heard here and there, both in front and behind. There was considerable tenderness on making pressure over the belly, and she had passed three copious liquid evacuations during the night, and two that morning. No spots were then found on the skin. Her pulse was 108, and respirations 38.

An enema of starch and opium was directed to be given after every loose stool, and turpentine stupes to be applied to the belly.

On the next day, the 8th, beyond the check to the diarrhœa by the enemata, there was no distinct alteration, either for worse or better, in the general condition of our patient. Six drachms of brandy were ordered to be given every second hour.

On the 9th, she was extremely drowsy and unwilling to be disturbed. The brandy was increased to an ounce every hour.

On the 10th, the 14th day of the disease, all the same symptoms continued, but on the whole she seemed better. A number of scattered rose-coloured spots were observed, for the first time, on the chest and belly. She showed a great aversion to the brandy.

There was no material change on the 11th and 12th, but as the diarrhœa continued, the ammonia was given in decoction of logwood, and the starch and opium enemata were administered as before, with decided benefit.

On the evening of the 12th she seemed more prostrate, and the brandy was increased to an ounce every half hour.

Her condition on the 14th was scarcely better, the same symptoms continued—breathing urgent, 48 times a minute; pulse 116; cough frequent and hard, with expectoration of simple mucus; profuse liquid evacuations recurring from time

to time, but kept in check by enemata; the same drowsy condition and dislike to disturbance; the hot and dry skin, with scattered spots becoming fainter. She continued to exhibit the greatest aversion to the brandy and beef-tea, swallowing very imperfectly what was put in her mouth, so that the proper amount of food and stimulants was administered with great difficulty. This is not an uncommon feature of such severe fever cases as that of this patient; it demands great firmness on the part of the practitioner, and in no conjuncture will he more require the active co-operation of an experienced nurse; timid and anxious relatives and friends are not to be depended on in such emergencies.

On the 15th, as there was some increase in the catarrhal sounds heard over the front of the chest, turpentine stupes were ordered night and morning. In the evening she became more drowsy; her head was therefore shaved, and acetum cantharidis applied to the scalp.

On the 16th, which was about the 20th day of the disease, a very decided improvement took place: she was more lively, slept quietly, and a profuse perspiration, in all probability critical, burst forth; her pulse fell to 112, and the respirations to 40. This improvement continued the next day; her tongue and lips began to clean, and the eruption had disappeared from the skin. On visiting the hospital in the afternoon, I found her again more drowsy, and thinking that the drowsiness indicated over-stimulation, I reduced the brandy from an ounce to six drachms every half hour. After this the drowsiness passed off, but for many days the improvement was slow; her cough continued troublesome, the bowels relaxed, and the pulse and breathing high. A second attempt to reduce the stimulants led only to their renewal in the previous doses.

From the 25th (the twenty-eighth day of the fever) the pulse fell in frequency rapidly; the brandy was reduced. It was not, however, until the 4th of March that the cough had given way: she then felt well, and anxious to get up; her pulse was 80 and respirations 30; she slept well, and her appetite was good. She left the hospital on the 12th (forty-eighth day), quite recovered.

You will not often meet with so severe a case as this ending

in recovery. I cannot but believe that the favorable result was owing to the steady exhibition of support of all kinds, especially of stimulants, from the earliest period of the disease. Still it is curious to observe, how about the twentieth day a marked favorable change took place, and was accompanied by a profuse sweating, apparently of a critical nature.

As a good example of the *Typhus* form of fever, allow me to direct your attention to the particulars of a case which proved fatal in the hospital in September, 1853, and which I shall have to refer to again.

Case XXV* (vol. xliii, p. 103).—E. Church, a man, aged fifty-nine, was attacked with shivering and pains in his limbs. In the course of a few days he was too ill to remain up and about, and therefore took to his bed. He suffered chiefly from great pain in his head, and there was some delirium. He was admitted to the hospital, September 18th, 1853, about a week after the shivering. He was then not sufficiently conscious to understand what was said; his tongue was dry and coated with brownish black sordes; pulse 100; *a measly rash covered the whole front of the chest and abdomen.*

A blister was applied to the scalp, ten minims of chloric æther with five grains of carbonate of ammonia were given every four hours, and half an ounce of brandy with beef-tea every half hour. The brandy was increased to six drachms in the afternoon.

He continued delirious through the night, and in the morning seemed more insensible; he passed his urine unconsciously, and could with difficulty be made to take the brandy. There was continual hiccough; pulse 100. A large blister was applied so as to cover the lower part of the chest and the stomach.

On the 20th the delirium rapidly gave place to coma. The hiccough continued. Pulse 92. In the evening the urine was found to be albuminous and to contain blood casts; and on this account the physician's assistant discontinued the stimu-

* Reported by Mr. C. Macnamara.

lants. At ten o'clock his pulse was much weaker, and had risen to 110; at eleven, it was imperceptible. Convulsions and death followed shortly afterwards, the disease having existed only ten days.

On examining the body after death, a small quantity of serum was found under the arachnoid. The lungs were much congested and also the spleen. There was no evidence of kidney disease excepting a slightly granular appearance.

It may be suggested that this was a case of uræmic poisoning from renal disease, and not typhus at all. The measly rash was sufficient evidence of typhus, which in this case may have attacked a subject suffering from diseased kidney in an early stage. No doubt the influence of the poison in this case would embarrass the action of the kidneys more than it is well known to do even when those organs are healthy.

Admitting as I do the existence among cases of continued fever of two clinical varieties, the Typhoid and Typhus, I am nevertheless convinced that instances every now and then occur, in which the distinction cannot be made, unless the presence or absence of enteric symptoms alone, or of some other *single* symptom, be taken as diagnostic. The following cases will serve to explain my meaning.

Case XXVI* (vol. xl, p. 264).—Daniel Ragan, aged twenty-four. His illness began on the 2d of March, 1854, with headache, but no distinct shivering; and he was admitted on the 6th with a fever, a rapid pulse, a brown and dry tongue, and suffering great pain in his head. Severe headache continued throughout, as a prominent feature of the case.

On the evening of the 8th, he was twice purged, and a mixture of chloric æther and decoction of logwood was ordered; also half an ounce of brandy every two hours, and beef-tea. Some rose spots were observed thickly scattered over the belly and chest. Pulse 104, respirations 22.

The next day the purging had ceased, the other symptoms continued, and he coughed and expectorated some brownish

* Reported by my clinical clerk, Mr. Bird.

mucus. It was thought advisable to shave his head, on account of the pain.

On the 10th a measly eruption was fully developed on his chest, belly, and back. A blister was applied to the scalp.

On the 11th, the respiration was rapid and laboured, 44 times in a minute; the pulse continued the same; there were some drowsiness and delirium; the diarrhœa returned, but was controlled by an enema of starch and opium.

On the 15th, being the thirteenth day of the attack, the pulse had fallen to 96; there was great improvement in all the symptoms, and the enema and logwood were discontinued. The following day there was some return of diarrhœa, which was checked by a repetition of the enema. He expectorated, with difficulty, a viscid mucus, which seemed to choke up the lungs. General improvement continued; and by the 20th, his pulse had fallen to 76. On the 22d, he was pronounced quite convalescent. He remained some time longer in the hospital, with pain in his side.

This, then, was a case in which, with a well-developed rubeoloid eruption, enteric symptoms were nevertheless present; it had some features of typhus, and others more prominent of typhoid, and there was the occurrence of the two eruptions in one person.

Case XXVII* (vol. xliii, p. 39).—John Cahell, aged forty-two years, was attacked June 16th, 1853, with pain in his head and hips; but, although ill, he continued his work until the 19th, when he was seized with shivering, and increased pain in his head and limbs, accompanied by total prostration of strength and loss of appetite. He had been purged excessively by a dose of salts. One of his children and several people in the same street were suffering from fever. He was admitted on the 28th.

On the 29th he complained of cough and sore throat; his bowels had been twice moved; a number of deep rose-coloured spots were observed covering his body. Pulse 104, respirations

* Reported by Mr. C. Macnamara.

28. He was ordered a mixture of chloric æther and ammonia, to be taken every third hour, and half an ounce of brandy every two hours.

On the 30th the pulse and respirations were 100 and 24 respectively. Bowels open once.

By the 2d of July, the spots had almost died away. Pulse 100, respirations 23. On the 3d, the pulse and respirations were 104 and 24; on the 4th, 112 and 28. The bowels continued regular. On the 5th, the pulse had fallen to 96, the respirations remained 28. He still complained of sore throat, for which a blister was applied over the larynx.

On the 6th, about the twenty-first day of the disease, he was in every respect much better: his appetite good, pulse 90, respirations 24.

From that time he made a rapid recovery. On the 8th, the pulse and respirations were 80 and 20 respectively; on the 10th, 78 and 20; and on the 12th, 78 and 24. On the 24th, he was discharged well.

Cases of fever are occasionally met with, which run their course and prove fatal, without the occurrence of any serious diarrhœa, and yet, on making a post-mortem examination, extensive ulceration is found in the ileum. I well remember a case of this kind which proved fatal in the hospital as long ago as the beginning of 1850.

Case XXVIII (vol. xxviii, p. 32).—The patient's name was Ada Dacon; she was eighteen years of age. Her illness commenced in the ordinary way, with rigors and pain in her head, back, and limbs, and had lasted a fortnight when she first came under treatment, January 19th, 1850. She was then in a high state of fever, and complained of frontal pain, great depression, loss of sleep, with frightful dreams; there was some abdominal tenderness, but no relaxation of bowels. Her pulse and breathing were 120 and 30.

On the 22d, some of the aromatic spirit of ammonia was ordered to be given in camphor mixture every six hours, and six ounces of wine in the day.

On the 24th, there were bronchial râles heard pretty exten-

sively; she could not sleep, but lay drowsy and moaning. Pulse 130, respirations 32. A blister was applied to the back of the neck, and a mustard poultice to her chest.

On the 25th, or the twenty-first day of the fever, she was much worse; her pulse and breathing were increased in frequency to 144 and 44; she had passed two relaxed motions, for which an astringent and stimulating mixture was ordered. There was no return of diarrhœa, but she continued in much the same state until the 29th, when she became rapidly worse and insensible, and died the following morning.

At the post-mortem examination, we found numerous well-defined ulcers, situated in the lower part of the ileum, and one or two large ones, involving the ilio-cæcal valve.

Besides such well-marked exceptional cases as XXVI and XXVII, we meet with others, from time to time, in which the eruption is not either of the typhus or typhoid kind, but something intermediate; and in which abdominal symptoms may or may not be present. Others again, though well-defined and even fatal cases of continued fever, will exhibit throughout no eruption of any kind.*

Besides the specific eruptions, we frequently meet with dark, purplish specks of a variable size, called *petechiæ;* they are pro-

* I may here notice very briefly two other cases to illustrate these exceptions.

James Scott, aged 14, was admitted with fever, July 11th, 1855 (vol. xlviii p. 106]. He had then been ill about a fortnight. He was deaf, and his body covered with *rather large rose-coloured spots.* Pulse 120. *His bowels had been costive, and were still confined.* He was treated by moderate stimulation. On the 14th he was sweating freely; pulse 92. He continued improving daily, and by the 20th was pronounced convalescent. There had been no looseness of the bowels throughout.

Joseph Garland, aged 18 (vol. xlii. p. 85), was attacked with sickness, giddiness, pains in his limbs and bleeding from the nose; and some days after by shivering, followed by severe fever and *loose bowels.* He was admitted on the 19th May, 1854,—the seventh day. His eyes were bloodshot and suffused, his throat sore, his breathing rapid; *an indistinct diffused rash covered his chest and arms.* He continued very ill *and much purged* for five days. Brandy and beef-tea were regularly administered, and the diarrhœa restrained by opium enemata. On the 24th there was decided improvement, and by the 29th he was convalescent.

duced by little extravasions of blood beneath the cuticle, and of course do not disappear on pressure. They are not peculiar to the fevers we are considering, but are common to them and to other fevers and diseases of debility. They were present in the following low typhoid case, and the spots in the patient Selby (Case XXXIV) had very much the petechial character.

Case XXIX (vol. xxxvi, p. 24). Emma Turner, aged seventeen, was admitted a fortnight after shivering, with symptoms of low typhoid fever. Her pulse was then weak and rapid, 128; respirations hurried, 36; her tongue was dry and brown, and her teeth and lips were covered with sordes. She was passing loose, dark, and extremely fœtid motions, and complained of great abdominal tenderness. Petechiæ were present on the abdomen and back. On listening to the chest, rhonchus was heard both in front and behind. She was constantly moaning, occasionally screaming out, and delirious.

Half an ounce of brandy was given every two hours; also chloric æther and astringents, opiate enemata, and turpentine stupes to the belly and chest.

For three days she remained in much the same state: the bowels continued relaxed and the motions were passed unconsciously, the pulse ran high, food and stimulants were administered with great difficulty.

On the fourth day after admission, she sank into a state of stupor, and died at night.

There is yet another form of eruption, which you will have frequent opportunities of seeing in fevers of this class—an eruption of minute, pearly vesicles, scattered in profusion over the skin of the neck, chest, &c.; these have been called *miliaria* or *sudamina*, and, as I mentioned in my lectures on rheumatic fever, are not peculiar to any one disease, but common to many, and indicative generally of a sweating state; hence I prefer the term *sudamina* to *miliaria*. I may add that they do not require a general sweating for their development; a local sweating, such as may occur in a fissure between folds of the skin, is often sufficient to bring them out there.

I must now proceed to consider, briefly, the more common

complications of fever—those involving the lungs, brain, bowels or kidneys, which I have not yet mentioned; and the plan of treatment I usually pursue in each.

Whenever the natural interchange of material between the blood and the tissues is imperfectly performed, the capillary force of the circulation is deficient, and the circulation through the capillaries becomes sluggish and imperfect. This is what occurs when an unhealthy blood is circulated—blood, for example, charged with the poison of typhus, or with urea; and we consequently find, in all such poisoned conditions of the blood, a tendency to local congestions, often of vital organs. Hence it is that we meet with pulmonary congestion as a common complication of continued fever; it is a purely passive congestion, due to the altered quality of the blood, and it has no resemblance, except as regards the hyperæmia, to inflammatory congestion. When fairly established and persistent, it is marked by wheezing, and more or less of crepitation, with increased bronchial secretion of mucus, occasionally tinged with blood, which the patient coughs up. This condition was well marked in the patient Copstock (Case XXIV), also in Selby, to whom I have before referred, and whose case I will quote in detail presently (Case XXXIV). The case of Emma Turner (Case XXIX), just referred to, affords a third example.

Instances might undoubtedly be found of the occurrence of a true inflammatory bronchitis, and even of pneumonia, in the course of continued fever; but these must be looked upon as extraordinary complication. I could instance several fatal cases of fever, in which patches of lung have been found carnified after death. In these cases the congested state of the lungs was intense, and the solidification seemed due to the great engorgement and increased secretion rather than to plastic exudation. At the same time this exudation now and then takes place both into the air-cells and on the pleural surface, but I doubt not that it is then simply the result of the mechanical retardation of the blood in the finest blood-vessels.

Case XXX* (vol. xli, p. 58). Sarah Beeson, aged twenty-

* From the notes of my clinical clerk, Mr. Colston.

three, an artificial flower maker, was admitted into King's College Hospital, July 9th, 1853. She had been living badly and in the midst of bad smells, and working hard.

On admission she was scarcely able to stand; she complained of severe headache; her eyes were suffused, her tongue coated, dry, and brown, her skin covered with a copious rubeoloid eruption; her pulse numbered 130, and respirations 26.

She was ordered ammonia with chloric æther, and two drachms of brandy every hour.

At night, she became delirious; and the delirium lasted through the next day, becoming worse at night, so that she could with difficulty be kept in bed. Her head was shaved, and a blister applied. The other symptoms continued much the same as on admission, the pulse and respirations rather increasing in frequency.

On the 11th, as she continued wild and delirious, two doses of morphia were given, which procured her some sleep. The following day there was less active delirium and more drowsiness. She was slightly purged, for which an opiate enema was administered; and as exhaustion seemed increasing, an enema of quinine and beef-tea was ordered every two hours.

On the 13th she was no better, but remained in the same drowsy state; her breathing was hurried, and there was some recurrence of diarrhœa. Opium was added to each quinine enema, and an ounce of brandy given every half hour: turpentine stupes were also applied frequently to her belly and chest. On the 14th her pulse and respiration had risen to 140 and 44; and the next day she died.

On examining the body after death, the lungs were found to be much congested, and the lower lobe of the right lung solidified, exhibiting a carnified rather than a hepatized appearance.

The brain substance appeared healthy: there was a little fluid under the arachnoid.

The spleen was greatly congested, and broke down easily under pressure.

In the treatment of these local congestions, you will do well to keep in view their nature—that they are but symptomatic of the general disorder, the direct results of the vitiated state of the

blood; and that, with a return of the latter to its normal condition, a resolution of the congestion may be expected. I would not, therefore, advise you to be very anxious to adopt any specific measures beyond those which I have recommended for the treatment of fever cases generally—the due support of the patient by suitable food, and stimulants proportioned to the exigencies of the case.

The local treatment need not on this account be neglected. You will find as the most efficacious, and the least likely to be injurious, free counter-irritation by turpentine fomentations and occasional blisters of good size, and applied at various parts, such as we employed in the cases of Copstock, Church, Turner, and Selby.

The same cause which operates in the production of pulmonary congestion is often effectual in producing a congested state of the brain, though of this almost the only evidence we have is derived from the post-mortem examination of fatal cases; for the only symptom of a congested brain occurring during life, with which I am acquainted, is a soporose condition bordering on coma; but this might very well and generally does result directly from a poisoned condition of the brain itself, and not from a mere increase or stagnation of blood in the organ. It must not be lost sight of, that much of the congestion of the brain observed after death is due to the mode of dying. When the breathing is hard, when the moribund state is tedious, and above all, when the patient has been convulsed just before death, the greatest degree of congestion may be expected.

I will quote two cases illustrative of the greatest amount of morbid change which you are likely to find connected with the brain in those who have died of fever. In one, we found the congested state marked by some darkening of the grey matter, and the occurrence of numerous bloody points in the white; while in the other a similar state had resulted in effusion into the ventricles.

Case XXXI* (vol. xli, p. 6). Charles Andrews, a painter, of intemperate habits, but generally good health, was taken ill on

* Reported by Dr. Plowman.

the 1st of May, 1853, with shivering, followed by heat and perspiration. The following day he had what his wife described as a fainting fit, and was insensible for some minutes. From the commencement of his illness, until his admission to the hospital on May 7th, he vomited constantly, and was also much purged.

When admitted, he complained of feeling drowsy and confused; considerable muscular tremors were present; he had almost entirely lost his memory, and showed a tendency to delirium. These symptoms, with the dry lips, furred tongue, suffused eyes, hot and dry skin, covered with an eruption of light-coloured spots, resembling the eruption of measles, and the rapid pulse and respiration, told plainly enough the nature of his complaint, and the large dose of the poison which he must have received.

A mixture of chloric æther and ammonia, and half an ounce of brandy every hour, were prescribed.

The next day his pulse and respirations had fallen from 100, and 44, to 96 and 36, respectively; but the pulse was extremely feeble. There had been no recurrence of diarrhœa. At night, however, he passed five liquid evacuations, and an opiate enema was ordered. On the 10th, the brandy was doubled, his head shaved, and a mustard poultice applied to the scalp. His pulse rose to 116, and the respirations to 40.

During the 11th and 12th, the same symptoms continued and increased, the most prominent were restlessness and delirium at night, jerking of the limbs, general and excessive muscular tremors, and relaxed bowels, or ineffectual efforts to pass an evacuation.

The brandy was increased, and turpentine and catechu given, but he died on the morning of the 13th.

At the post-mortem examination "no effusion was found beneath the arachnoid, or in the ventricles; the membranes of the brain appeared perfectly healthy." "The brain itself was somewhat congested; the veins were turgid with dark blood; the gray matter of the convolutions was slightly deepened in colour, and very numerous bloody points were seen on slicing it so as to display the centrum ovale." "The brain substance was hard and firm." The spleen was very soft, almost of a creamy or pulpy consistence.

In this case, the symptoms were due, in my opinion, to the *poisoning of the nervous matter* of the brain; in other words, to its perverted nutrition; and the fatal result was much hastened by the diarrhœa which showed itself so early.

Case XXXII* (vol. xviii, p. 9).—The other case is that of James Davis, aged twenty-four, a man of temperate habits, who was admitted May 13th, 1846, with fever. The commencement had not been sudden or marked by any shivering, but gradual, with langour and pains in his limbs, head, and loins. He had been ill for more than a week before admission, and during the latter part of the time, delirious.

On the day after admission, he was still delirious, passed some watery evacuations, complained of much abdominal pain, and was unable to empty his bladder, which became rapidly distended and had to be emptied with a catheter. Pulse 104.

His head was shaved; five grains of carbonate of ammonia were ordered three times a day, and half an ounce of wine every two hours with beef-tea.

The next day there was no return of purging, but the other symptoms continued and increased; his pupils were dilated, and he lay continually on his back. One ounce of brandy was now given, alternately with an ounce of wine, every hour.

On the 16th and 17th, he continued much the same; some rhonchus was heard in the chest, and his belly became tympanitic, and the mucous membrane of the mouth very foul with sordes. He died on the 18th.

In this patient, as in Andrews, there was much subsultus tendinum.

At the examination of the body after death, the membranes of the brain were found much congested, as also the white substance of the hemispheres, which was thickly studded with red points; the whole brain was softer than natural; there was a large effusion of pale straw-coloured fluid into the ventricles.

I have no doubt that in this case, as in many others, the ventricular effusion was a passive dropsy consequent on the retarded cerebral circulation.

*. Reported by Mr. R. D. Mills.

There were evidences of tuberculous disease of the lungs and mesenteric glands.

Peyer's patches were very prominent, and increased in size, but not ulcerated.

The sub-arachnoid effusions which we meet with now and then after fever are not of an active kind. They are the result of a certain shrinking of the brain, fluid being poured out to fill up space. Do not fall into the mistake of supposing that an effusion of this kind is instrumental in causing comatose symptoms. It, in truth, exercises no more than the normal pressure which seems a necessary condition of the brain's nutrition.

Of the cerebral symptoms—delirium, coma, and convulsions—two of which, at least, are of frequent occurrence in continued fever, I can only repeat what I said when speaking of the same symptoms in my lectures on rheumatic fever—that we have no grounds at all for supposing them due to any inflammatory or congested condition of the brain or its membranes, but must rather consider them as the result of that perverted nutrition which is the necessary consequence of the poisoned condition of the nutrient fluid.

With respect to the treatment of these symptoms, what I said in speaking of the treatment of pulmonary complications is applicable here also; you must treat them as part of the general disorder, not as distinct diseases. An increase of these symptoms, especially of the delirium, usually indicates an increasing exhaustion, and therefore demands a larger supply of stimulants.

Of local remedies, I find the application of blisters to the scalp and back of the neck, and the employment of a cold affusion either to the head or over the whole body, the most efficient means of rousing a patient from a drowsy comatose state. The douche sometimes acts like a charm; it is most applicable to cases in which a lethargic state supervenes early, and before there is great exhaustion; and it should always be employed with as little distress to the patient as possible.

The three following cases are good examples of the occurrence of cerebral symptoms, and of the treatment I have recommended.

Case XXXIII* (vol. xli, p. 197). Zechariah Stilling, aged twenty-six, an Irish labourer, of irregular and intemperate habits, was brought to the hospital, February 11th, 1854, with unequivocal symptoms of fever,—a hot and dry skin, brown and furred tongue, some stupor, loose watery evacuations, numerous rose-coloured spots, disappearing or becoming pale on pressure, and harsh respiration heard over the front of the chest. The attack commenced about nine days before with shivering, headache, and considerable diarrhœa.

Half an ounce of brandy was given every three hours, and beef-tea; also some aromatic spirits of ammonia in decoction of logwood.

From the 11th to the 14th there was no change; his pulse continued 116, and the prominent symptoms were a noisy, restless delirium, with an obstinate determination to get up and leave the hospital, and great pain in his head.

On the 13th, the brandy was increased to half an ounce every two hours; and on the 14th his skin was moist and his pulse had fallen to 100, and from that day to the 20th it gradually declined to 50.

On the 15th and 16th he was still restless and wandering, with pain and noise in his head. Mustard poultices were applied to the scalp. All the bad symptoms, however, rapidly passed off; the brandy was gradually reduced; and by the 28th he was fairly convalescent. There had been no return of diarrhœa throughout.

In this case the active stimulation had not been commenced sufficiently early. Had it been otherwise, the delirium would have been less developed.

Case XXXIV† (vol. xxxix, p. 59). John Selby, aged twenty-seven, detective police officer, of temperate habits, and previous good health, was admitted to the hospital, December 11th, 1852, in a state of almost complete coma. It appeared, that about ten days previously he had been seized with severe rigors; he then grew rapidly ill, suffered from considerable purging, and, during the two days preceding his admission, was delirious. I will read you the report made by my clinical clerk of his condition

* Reported by Mr. Buzzard.

† Reported by Dr. Plowman.

on admission: "He appeared perfectly unconscious, and quite unable to swallow anything: when it was attempted to give him fluids to drink, they simply collected in the mouth, scarcely any passing into the œsophagus, and were slowly ejected by each expiration. His abdomen was covered by a great number of small, circular, somewhat purplish or mulberry-coloured spots, having much the character of petechiæ, not raised, and not disappearing, though slightly fading at their circumferences, under pressure. There was no marked tenderness of the abdomen. Pulse 140, respirations 30."

Beef-tea and half an ounce of brandy were administered every half hour, a mustard poultice was applied to the back of his neck, and a turpentine stupe between the shoulders. His head was shaved and a cold douch applied. The bladder was emptied by a catheter.

Great benefit seemed to result immediately from this free counter-irritation and stimulation; he was completely roused from his stupor, and appeared altogether much better. On the following day, the 12th, the improvement continued; his pulse and respiration had fallen to 112 and 24 respectively; he had passed his water freely, though involuntarily, and his bowels had moved, but the motions were not relaxed. Some of the acetum cantharidis was rubbed on the scalp.

He passed a quiet night and slept well; but we were disappointed in the morning to find him decidedly weaker, his pulse risen to 120 and the respirations to 30, his tongue dry, with a thick brown coat. This was explained by the discovery that he had been neglected by the night nurse, who had omitted to give him the brandy and beef-tea regularly. He passed a quantity of dark-coloured acid urine and some dark liquid fæces, voluntarily. The brandy was doubled, and a blister applied to the scalp.

On the 14th, after a comfortable night, he was again better, but his pulse remained the same, and he complained of some slight abdominal pain.

On the 15th, as there was some cough with a dark sanguineous expectoration, a turpentine stupe was ordered. By the evening his pulse and respiration had fallen considerably, numbering 100 and 22 respectively.

The following note was made on the morning of the 16th :—
'He remains much the same. Has still a little cough, but the sputum is clear; breathing natural; the spots are becoming faded, and much fewer. Pulse 96; respirations 24. The tongue is tremulous but much cleaner; there is still no purging, and he does not complain of any particular pain in his stomach."
The brandy was reduced again to an ounce every hour.

From this time there was steady improvement. The respiration and pulse declined; the latter was 92 on the morning of the 17th, and 88 in the evening; 84 on the morning of the 18th. His tongue became cleaner, his urine natural, and he passed good nights. An equal amount of wine was substituted for the brandy: that is to say, twenty-four ounces of wine were given him daily, and under this the pulse came down in the manner detailed.

There was something like a critical sweating on the 18th, which was also the eighteenth day of the disease: the perspiration was profuse, and continued through the 19th and 20th.

On the 21st he felt himself to be much stronger. His pulse was 80, full, and fairly strong, though still compressible; respirations 20. He only complained of some confusion of thought, and of a swimming sensation in his head, which disappeared in the course of a week or ten days, as his strength returned.

The amount of wine was gradually reduced, and on the 28th a quinine mixture was ordered, and on this treatment, with a liberal diet, he made an excellent recovery. He remained in the hospital until January the 22d, when he was discharged quite well and in almost his former strength.

Case XXXV* (vol. xxxix, p. 213).—Thomas Keen, aged thirty-three was admitted March 26th, 1853.

Ten days before, he had been seized with shivering, and for a week afterwards suffered from relaxed bowels; but, from the time of his admission until he left the hospital, he was quite free from any recurrence of looseness or abdominal symptoms.

He was ordered some carbonate of ammonia with henbane in

* Reported by Dr. Plowman.

effervescing mixture, morphia at night, and an ounce of wine every two hours, with beef-tea.

During the night of the 26th he was delirious, and had not recovered his consciousness by the morning of the 28th. His face was then flushed and his eyes suffused, his skin hot and dry, and the tongue presented a brown band in the centre. Pulse 92. On his back and abdomen were scattered a few distinct, non-elevated, rose-coloured spots, rather smaller than a split pea, and some of them entirely disappeared on pressure. A great number of sudamina was also present on the belly at the upper part. He coughed frequently, and rhonchus and sibilus were heard in front.

On the 29th, there was more wandering, with drowsiness and slightly stertorous breathing. Pulse 120; respirations 28. The other symptoms were but little altered.

On the 30th, brandy was substituted for wine. He appeared to derive benefit from the change, for he slept better and was less delirious and more conscious.

On April 1st, the brandy was doubled, *i. e.*, an ounce was given every hour, his head was shaved and a mustard poultice applied. On the 2d a cold affusion was administered. On the 3d, there was a fall in the pulse from about 100 to 86, notwithstanding twenty-four ounces of brandy daily; and on the 4th, which was the twenty-first day of the fever, it did not exceed 72. He was then sweating profusely, the moisture running off his forehead.

On the 5th, the pulse was as low as 52. The brandy was reduced to four ounces, and porter and quinine given. On the 12th, he was convalescent.

In a former lecture, I spoke at length of the exhausting diarrhœa or hæmorrhage which is apt to accompany cases of typhoid fever, and also of the morbid appearances found after death in the intestine. I shall have to allude to this intestinal hæmorrhage again, as one of the critical discharges by which the fever sometimes terminates.

There is another abdominal symptom for which you must be prepared. The affection to which I allude is a form of tympanitis, or meteorism, as it is called. It consists in an inflation

of the intestinal canal by gas more or less rapidly generated within it. In consequence of this inflation, the belly becomes very prominent, tense, and drummy, and is highly resonant on percussion at every point. There is no sense of fluctuation present excepting when there may have been liquid diarrhœa, and a considerable quantity of fluid remains in the bowels. Under these circumstances an obscure fluctuation is perceptible, which you must not allow yourself to be misled into supposing to arise from fluid in the peritoneum. That the fluid is within the bowels along with the air, is proved by the borborygmi and other metallic sounds, which are audible under the influence of the peristaltic action of the bowels, or under strong pressure or succussion of the abdominal walls.

This distended condition of the bowel is due to a secretion of air from the mucous membrane, partly also, possibly, to a generation of gas from decomposition of the contents of the gut. There is no doubt a very defective nervous influence, which regulates imperfectly both the secretions and the muscular motion of the bowel.

This *meteorism* occurs, so far as my experience teaches me, in all the forms of continued fever, and does not, as one might suppose *à priori*, especially belong to that in which the bowels are so apt to be irritated—namely, the Typhoid. On the contrary, I should say, it is of more frequent occurrence in Typhus. I may remark that this symptom has not been nearly so often met with in my own practice, since I have adopted the plan of thoroughly upholding my patients from the commencement.

This tympanitic state is by no means peculiar to typhus or typhoid fever. It occurs in other diseases of defective nervous influence: in severe diseases of the spinal cord; in affections of the brain, such as acute meningitis, and in peritonitis. In all such cases the influence of the intestinal nerves must be impaired; the muscular coat of the bowel must in great degree lose its tone, and allow the bowel to become full and distended, through the want of the resistance which a strong muscular coat would oppose to the accumulation of gas.

The treatment which we adopt for this condition in typhoid cases consists in the external application of turpentine, in the

form of hot fomentations to the belly, the frequent use of enemata with confection of rue and turpentine, sometimes a mild warm aperient, and in extreme cases, galvanism.

Another incidental feature of fever cases, allied to the last, is a more or less perfect paralysis of the bladder, so that when it becomes full no active contraction of the detrusor muscle occurs, and the sphincter remaining closed, the urine is retained, and the bladder becomes distended. If this is allowed to continue, the urine will dribble away, and it may be long before the bladder recovers its muscular power.

We had illustrations of this condition in the patients Selby and Davis. Let me advise you when attending a case of fever to make frequent inquiry respecting the condition of the bladder, and if you can feel it forming a tumour above the pubis, to draw off the water with a catheter as soon as you can. I can say of this symptom, as of tympanitis, that under good support and stimulation from the commencement, it is of far less frequent occurrence.

An albuminous condition of the urine is occasionally found in cases of continued fever. When it occurs, we must consider it owing to a congested condition of the kidneys, arising not only from the general tendency to capillary congestion, but possibly also from a functional effort on their part to eliminate some of the poison from the system. The urine of the patient Church (Case XXV) was very albuminous, and revealed *blood casts* under the microscope. When you find albumen in the urine, you will naturally suggest to yourselves, is this due to diseased kidney or to a temporary congestion? The answer cannot be given at once: to discover any morbid change you had better wait till the fever has gone off. Meantime no harm is experienced by the passage of the albumen, and if there be evidence of morbid change, it must be dealt with when the patient has thrown off the fever.

I have yet a few remarks to make on the manner in which cases of fever terminate. You have all heard of the *turn* of a fever: the idea is an old one, as old as Hippocrates, that fevers are wont to change suddenly for better or worse on certain days called critical days. In later times the notion was discarded as little better than an old wife's fable; but more recent

observations go far to establish the truth of it. The *crisis* of a fever is frequently marked by the occurrence of some copious evacuation, either of a natural kind, such as free sweating, which is by far the most common, or by some unnatural one, such as a profuse bronchial secretion of watery mucus, or the passage of a quantity of blood from the bowels. This is followed or accompanied by a rapid diminution of the febrile symptoms, and in favorable cases by speedy general amendment. This is not, however, invariably the case: a well-marked crisis may occur, and perhaps be followed by an abatement of fever, and yet from that time the patient, instead of recovering, may grow more comatose or exhausted, and the case terminate fatally.

I will give you three examples of well-marked crises, one of which was unfavorable.

Case XXXVI (vol. xxvii, p. 164).—William Brown, a painter, twenty-nine years of age, who had been living badly for some time, towards the close of a week of unusually hard work, felt weary and indisposed, and on Saturday night was attacked with a fit of shivering, feeling alternately hot and cold; he continued chilly and shivering all Sunday, and was admitted into the hospital on the next day, Monday, May 28th, 1849, suffering severe pain in his head, back, and extremities, and with all the symptoms of fever—heat, thirst, loss of appetite, furred tongue. After having a warm bath, there was some perspiration, and a copious eruption of spots, called in the record of the case *petechial* (?), was observed. His pulse numbered 100.

A saline mixture of citrate of ammonia and nitrate of potash was ordered, and a very small allowance of wine with plenty of beef-tea.

The fever continued high for four or five days; he became deaf, stupid, light-headed; he coughed a good deal, and the eruption remained fully developed. The wine was increased to half an ounce every two hours.

On the 2d of June (the seventh day of the fever) the bowels became relaxed, and he passed a watery evacuation; this was followed, the next day, by a remarkable cessation of fever, the pulse falling to 60. He seemed, nevertheless, so extremely

low, that eight ounces of brandy were ordered to be given in the next twenty-four hours; an enema was also given to check the bowels, and a large mustard poultice applied to the belly. From this time, however, the amendment was rapid, and he was discharged on the 14th of June, the twentieth day from the shivering.

In this case the purging seemed to mark the crisis.

Case XXXVII (vol. xl, p. 47).—Jane Green, thirty-eight years of age, from the parish of St. Giles, was admitted to the hospital, May 24th, 1853, with fever. She stated that she had never had a serious illness, but that her habit had been to live badly and drink hard.

Her illness began, May 14th, with shiverings, perspirations, numbness of the hands and feet, deafness, relaxed bowels, and nausea; with a hard cough and sense of weight in her chest.

She first applied at the hospital as an out-patient, but becoming much worse she was taken in.

The following was her condition on admission: "Her pulse was 140, her skin very hot, her arms and chest dotted all over with red spots, and her tongue and teeth were covered with some blackish blood which she had vomited just before. Her bowels had been recently moved, and the motion was dark-coloured and offensive."

She was ordered five grains of carbonate of ammonia and fifteen minims of chloric æther every three hours, an ounce of brandy every hour, and a morphine draught at night. Her head was shaved, and a mustard plaster applied to the chest.

On the next day, the 25th, an enema of ten grains of quina in two ounces of beef-tea was ordered every two hours, and the morphine draught to be repeated at night.

On the 26th, she appeared much better, complaining only of thirst. She slept a good deal, but wandered at times; pulse 110; her urine was found to be albuminous, and the skin on the buttock seemed threatening to slough.

Acetum cantharidis was applied to the scalp, so as to produce vesication, and the enema of quina and beef-tea continued every four hours.

In the evening *she was purged three times, passing very dark*

and offensive motions, for which a starch and opium enema was prescribed.

On the 27th, she seemed better; her pulse was 108, her tongue red and moist, the quinine enema was discontinued.

On the 28th, the fifteenth day of the fever, the pulse had fallen to 90, and the patient was sweating profusely.

The brandy was reduced to half an ounce every hour.

On the 30th, the pulse was 84; and on the 3d of June, 75. Her tongue was then clean, and a chop was ordered and wine instead of brandy.

In this case, although great care had been used, a sore had formed in the gluteal region, which required poultices and afterwards stimulating dressings, and detained her some weeks in the hospital.

Case XXXVIII (vol. xliv, p. 81).—Sophia Bruce was admitted with fever July 26th, 1854. There had been two or three cases of fever in the house from which she came. Her illness had commenced with shivering, and the usual symptoms, seventeen days before her admission. Shortness of breath had occurred very early, and formed a prominent symptom when she was admitted; crepitation and rhonchus were then also heard both in front and behind; the usual febrile symptoms were present, and there was an eruption of scattered rose spots on the abdomen.

Half an ounce of brandy was given every hour, also chloric æther and ammonia, and enemas of salicin and beef-tea; and turpentine stupes were applied to the chest.

She continued much the same for some days. Her breathing was extremely rapid, exceeding 50; her pulse about 100; but the heart's action on the 29th was nearly twice as rapid as the pulse. The brandy was doubled, and quinine substituted for salicin.

On the 30th, the twenty-first day from the shivering, *she was sweating very profusely;* but from that day she became worse, and on the 31st was semicomatose; some purging also occurred. These symptoms continued, the diarrhœa with some intermissions, until her death on the 9th.

After the sweating on the 30th, the fever seems to have abated; for on the 3d of August, we have the following note,

"Her skin is cool and moist; the fever seems to have abated." It appears to me that the cause of the fatal termination was the excess of the critical discharges, which with the diarrhœa exhausted the patient. No doubt the attempt at relief by the natural process may occur, unsuccessfully. And herein we learn the importance of upholding patients from the commencement, that these critical evacuations may not produce fatal exhaustion. Sometimes a crisis, though favorable, is marked by a temporary exacerbation of the symptoms, as in the following case:—

Case XXXIX (vol. xli, p. 213). Daniel Shea, aged twenty-six, a labourer, of intemperate habits, was attacked with violent pains in his head and limbs. He continued to have headache for a week, and passed disturbed and restless nights until his admission to the hospital, March 15th, 1854. He was then suffering from fever; his pulse was 106, and respiration 30; his tongue furred; his whole body, but especially the back and belly, was covered with a papular rose-coloured eruption, the spots being aggregated into patches of variable magnitude, and disappearing on pressure. Half an ounce of brandy was given with beef-tea every two hours, also ammonia and chloric æther.

For about a week he continued very ill, with little change in the symptoms; his pulse continued feeble and rapid, 112—104, his tongue dry and furred, and lips black with sordes. For several nights he was delirious; the brandy was doubled, and ten minims of tincture of opium were added to each dose of his mixture. Subsultus tendinum, cough, and relaxed bowels occurred, but did not continue.

About the 21st and 22d, delirium gave place to deafness and drowsiness, the eruption disappeared, and his pulse fell to 100. At night, however, he became very delirious, and on the morning of the 23d, the twentieth day of the fever, he was unconscious: he lay on his back with his eyes nearly closed, and his face covered with a profuse sweat. His head was shaved and blistered.

This condition of the patient might naturally enough have lead to an unfavorable prognosis; but from this time he began

to amend, the pulse steadily falling, so that by the end of a month from the commencement of the attack he was quite convalescent.

A crisis most frequently occurs in this country, I think, speaking roughly, at the end either of the second or third week from the shivering, more commonly the latter, not often much earlier or later, but sometimes on intermediate days.

The case of Copstock (XXIV, p. 138) affords a good illustration of crisis by free sweating.

Dr. L. Traube, of Berlin, has published the results of some elaborate researches, made by himself, in an essay on crises and critical days, of which you will find a review by Dr. E. H. Weber in the 'Medico-Chirurg. Review,' January, 1853. The investigations were made with special reference to the temperature of the body in febrile affections, that being taken as the index of fever. He finds that crises, marked by a fall in temperature and such critical evacuations as I have mentioned above, occur, in continued fever, most frequently on the fifth, seventh, ninth, or eleventh days; that is, on the odd days only, and considerably earlier than usually happens in this country. The days are successive intervals of twenty-four hours, commencing from the first accession. But the difficulty of determining the exact period of accession is so great, that the assignment of the crises to the odd days with accuracy must, in many cases, be quite impossible.

Very often these febrile symptoms subside gradually without the observation of any critical evacuation. This is what has been designated resolution of the fever by Lysis. The occurrence of such cases in no degree invalidates the notion that the fever may be quickly terminated by a critical discharge, for it is quite possible that that, which under other circumstances would be limited to the skin or the kidneys or the bowels, in such cases is distributed among these emunctories, so that the increased discharge from each becomes comparatively insignificant, and escapes detection.

I shall conclude this too long lecture by a few instructions to you, which for conciseness I have put into an aphorismic form, touching the management of the kind of cases upon which I

have been commenting. Let me, however, first make this remark, that however important it may be for clinical purpose, and for prognosis, and for satisfying the inquiries of friends and relatives, to make the distinction between typhus and typhoid, there is no essential difference in the treatment, excepting that in the latter you must be always on the look out for bowel disturbance, and take measures to prevent or to check it.

My first rule is this—never give up a fever patient until he is plainly *in articulo mortis*. Patience, perseverance, and steady adherence to a well-devised plan of treatment are, in fever cases, often crowned with success under the most discouraging circumstances (case of Shea, No. XXXIX, vol. xli, p. 213).

2. When you undertake a case of this kind, insist upon having a good nurse; give her full instructions, if possible in writing, and require her to keep a note of all food and medicine administered by her. Do not trust to the nursing by relatives.

3. It is not advisable that you should see your patient too often; once, or at most twice a day, will be, in general, quite sufficient.

4. Do not be anxious to account or provide for every new symptom or change that may arise. Do not treat symptoms too much, but look to the general condition of your patient. Diarrhœa, delirium, coma, hæmorrhage, are the symptoms which you should look out for, and be prepared instantly to meet.

5. Watch the pulse closely, both as to quality and frequency. Always keep in view that increase of quickness is a sure sign of increasing debility, and that diminution of quickness (when not referable to any cerebral affection) is the reverse.

6. Never allow a day to pass without carefully examining the abdomen of your patient, and especially the region of the bladder.

7. Do not be anxious about the action of the bowels, and the so-called secretions. Many a patient in fever has fallen a victim to the diligentia medici nimia in improving "the secretions." You may make them perfect in colour and consistence, and yet your patient will die.

8. Restrain diarrhœa and hæmorrhage, and when, in typhoid fever, you have fairly locked up the bowels, keep them so.

Patients will go for four or six days, or even longer, without suffering inconvenience from this state of constipation.

9. From the first moment of your attendance let it be your constant and anxious effort to uphold the vital power of your patient, by nitrogenous food given as broths, and carbonaceous food, selected from farinaceous substances, and from alcoholic fluids, such as wine, brandy, or other fermented liquors.

10. Increasing delirium and coma are signs of increasing debility; both indicate the necessity for additional support: coma is often benefited by freely blistering the nucha and scalp, and even the region of the heart.

LECTURE VIII.

ON CERTAIN ACUTE DISEASES.

ERYSIPELAS.

I TAKE as the subject of my lecture to-day, gentlemen, two cases of erysipelas which have lately been under treatment in the hospital. This disease deserves your most diligent watching, inasmuch as it is of frequent occurrence in this town, and one which it is very important you should be well prepared to treat. One of the cases to which I shall refer is a good example of that form of erysipelas which is most commonly met with in the medical wards,—erysipelas of the head and face; the other, of that form which comes more immediately under the notice of the surgeon, though in both instances the disease is essentially one and the same.

CALE XL (vol. xliv, p. 189).—The first case is that of John Child, who was in No. 4 ward, but who has now left the hospital; this patient presented a good example of an average case of erysipelas of the head and face, coming on, as it very often does, after exposure to wet and cold. His history is as follows:—He is a labourer, twenty years of age, and has always enjoyed good health until a few days before his admission into the hospital, when, after considerable exposure to wet and cold, he was seized with general febrile symptoms of a severe character, and these were accompanied with swelling and redness of the nose, which gradually spread, until they involved the whole of the face and head.

Unfortunately, our notes of the case at this period are not so full as could be wished, and we are, therefore, quite in the dark with respect to certain points in the early history of the attack,

with which it is important that we should be acquainted. My clinical clerk, for example, has omitted to mention whether vomiting or rigors were present among the first symptoms. Erysipelas generally begins with rigors, and the attack is also often ushered in by vomiting; indeed, vomiting is a common symptom at the onset of the disease; and to such an extent does this obtain, that whenever I meet with a patient who has been suddenly taken with vomiting, and this vomiting accompanied with, or preceded by, rigors, I deem it expedient to watch carefully for erysipelas.

Sore-throat, too, is often one of the earliest symptoms of this malady; the disease sometimes appears to begin at the fauces, which under these circumstances are generally much redder than natural; and from this part it seems to spread outwards through the nose, affecting the alæ nasi, the face, the eyelids, the forehead, and lastly, the scalp,—following, in fact, a regular course; and this was very much what occurred in the case under consideration. At other times the throat and face become affected simultaneously.

The swelling of the face is frequently so great that the eyes are, to use a vulgar expression, "bunged up," and the patient is quite unable to open them; and so they generally remain for some days,—a copious secretion, poured out by the conjunctiva or Meibomian glands, concreting, and gluing the eyelids together. In this instance, the swelling rapidly spread, the face became greatly puffed up, and the eyelids completely closed; and in this condition the patient was brought into the hospital. Under the treatment adopted, and which I am anxious to recommend to your particular attention, the fever diminished in the course of four or five days, the swelling of the head and face subsided, the patient was able to open his eyes, and the pulse fell in frequency, and in six days after his admission he was fairly convalescent, "sitting up, and able to move about the ward."

The duration of the fever in erysipelas is not long, usually varying from seven to fourteen days; but the phenomena of the disease naturally divide themselves into two classes,— the *primary* and the *secondary*. The former of these comprises the rigors, the vomiting, the general febrile symptoms, the rapid

pulse, the peculiar redness and swelling of the skin, &c.; and if due attention be given to uphold the powers of the patient from the commencement of the attack, it rarely happens that the secondary phenomena manifest themselves at all. The poison of erysipelas, whatever its nature, appears to fall chiefly upon the tegumentary system, expending its virulence upon the true skin and the mucous membranes; yet it frequently happens, when the march of the disease has not been successfully opposed by well-directed measures, that it spreads from the true skin and mucous membranes to the areolar tissue beneath these structures, and it then gives rise to the secondary phenomena of the malady, namely, fever of the hectic kind, and the formation of collections of pus, either at the seat of the primary disease or in other parts of the body. It is very important that you should keep in view this tendency of erysipelas to involve the cellular tissue beneath the skin, and there induce the formation of pus; for it should be your aim to cure the disease without allowing it to bring about these secondary consequences, which are always exceedingly wearying and debilitating to the constitution, and sometimes so exhausting as to terminate the patient's life. In the case which I have just related to you, the cure was speedily effected without the occurrence of any of these secondary results; and this I attribute, in great measure, to the plan of treatment which was adopted.

Case XLI* (vol. xlviii, p. 22).—In the second example of erysipelas which I wish to bring under your notice to-day, the result was by no means so satisfactory; indeed, the case forms a good contrast to that of John Child, which you have just heard, illustrating, as it does, the less favorable course of the disease, under a very different plan of early treatment from that pursued in his case, as you will presently see.

The patient is a lad named John Walker, who was admitted into Fisk ward, February 17th, 1855, with erysipelas of the right leg and foot; and, I am sorry to add, he is still under treatment in the hospital, and likely to be so for some time to come. There was no history in this case of the patient's

* The record of this case was kept by my clinical clerk, Mr. Jenkins.

having received any injury, and nothing whatever to explain the occurrence of the attack; but it was attributed by the child's parents to his having lived very badly for some time, and to his having been of late much exposed to wet and cold. The short account which we have of the case is this:—About a fortnight before the lad's admission into the hospital, it was noticed that his right leg, all the way down to the foot, was exceedingly red and somewhat swollen, and this was accompanied with general febrile symptoms; but neither his parents nor himself could remember whether he had any shivering or not. He was kept in bed, and the only treatment which was adopted consisted in the application of hot fomentations to the limb, and, from the necessitous circumstances of the parents, in the administration of the poorest kind of food, and very little of that.

Upon his admission to the hospital on February 17th, about fourteen days after the commencement of the attack, it was found that the primary phenomena of the disease had run their course, and that the secondary phenomena had supervened. For this we cannot attach blame to the medical treatment adopted, for, properly speaking, he had none; but it is rather to be ascribed to the extremely bad living and poor food upon which the patient had subsisted during the whole of the winter, and to his having been deprived, during the first fortnight of his illness, of all those comforts which are so necessary during a severe attack of this disease. And here let me call your attention to the fact—that if low diet, and that general course of treatment which is commonly summed up in the word antiphlogistic, be essential to the cure of erysipelas, surely this poor lad had enough of it! Nevertheless, it did not succeed in preventing the secondary phenomena of the disease from manifesting themselves; for, upon the patient's admission, there was distinct evidence of suppuration beneath the integument of the leg, for the relief of which it was necessary, after a day or two, to make two incisions through the skin, one along the outer, and the other along the inner side of the ankle; and the following day it was thought expedient to make a third incision along the dorsum of the foot, for the escape of a considerable quantity of pus which had collected in that situation. The weakness of the patient on his admission was

extreme, and his pulse was 120, small and very compressible; the treatment upon which he was at once put consisted in the free exhibition of beef-tea and brandy, ammonia and chloric ether.

The further history of the case is as follows :—The erysipelatous swelling of the leg and foot subsided, but a very free discharge of pus continued, and still continues, to take place; the rapidity of the pulse kept up, and the exhaustion became, if possible, still greater than on the patient's admission, so that it was found necessary to adopt every means which could be resorted to for the purpose of upholding his strength. To this end, beef-tea enemata, each containing ten grains of quinine, were administered, regularly, every fourth hour, half an ounce of brandy every half hour, and as much beef-tea, and milk thickened with flour, as the patient could take. Notwithstanding all this, the pulse kept up, being 134, 137, 140, &c., and never below 120; the suppuration continued to progress, and, a day or two ago, a large sinus was found to have formed between some of the muscles of the leg, so that it is even now exceedingly doubtful whether this lad will ever recover; for, when suppuration has taken place to this extent, it often not merely spreads along the areolar tissue between layers of muscles, but also, when in the neighbourhood of joints, it may so extend as to involve these also. It is by no means improbable that some of the joints of the tarsus may here become affected, and render the amputation of the leg necessary; and such a measure as this, I need not say, would be attended with great hazard to life in a patient so thoroughly depressed and exhausted as this poor boy is.

Before dismissing the subject, let me observe, that in all these cases, the rapidity of the pulse from day to day is a valuable index of the progress which the suppurative process is making; for if you find that the rate of the pulse keeps up, despite of free supplies of nourishment, you may infer that more mischief is about to ensue, and that further suppuration will occur; and under these circumstances, it will seldom happen that the pulse will come down, until a free discharge of purulent matter shall have taken place.

Such then, gentlemen, are the two cases which will serve as

the basis of my remarks to-day. The one, an ordinary case of erysipelas of the head and face; the other, a case of that form of the disease which is most frequently met with in the surgical wards—phlegmonoid erysipelas, and which is precisely analogous to traumatic erysipelas, or to that which follows a surgical operation.

Now, as I have already remarked, the most common form of erysipelas which comes under the care of the physician is that of the head and face. Let us trace the clinical history of this disease. It very frequently begins in the throat, and it is important that you should be acquainted with the various courses which it may take. It is one of the features of this disease that it has a remarkable tendency to spread, sometimes wandering all over the body, from face to neck, and neck to trunk, being then termed *erratic* erysipelas. When, then, it has commenced in the throat, it seldom finishes its course there, but immediately begins to spread, and its ordinary course is upwards from the throat, through the nose, to the face and head; sometimes it pursues the opposite direction, and wanders down, over the respiratory mucous membrane. In following this latter course, it generally passes very quickly (most fortunately for the patient) over the laryngeal mucous membrane, and then, affecting the mucous lining of the air-passages below the larynx, produces *erysipelatous bronchitis*—a complaint of more common occurrence than is generally supposed, of very fatal tendency, accompanied with great prostration, and which often leads rapidly to purulent expectoration. When this malady terminates fatally, it generally does so by inducing that condition which, since the death of the Emperor Nicholas of Russia, has been so much talked about as paralysis of the lungs—a bad term, intended to express that state of things which occurs when the air passages become choked with muco-purulent secretions, which the patient is too much exhausted to expectorate. This accumulation of viscid secretion in the air-tubes tends, of course, to produce suffocation, by interfering with the proper aeration of the blood in the lungs; and the consequent retention of carbonic acid in the circulating fluid poisons the nerves and nervous centres, diminishing their excitability, and rendering less free those reflex actions on which expectoration in some degree depends.

Thus the accumulation of the secretion in the air passages is still further promoted, and so these two conditions, narcotized nerves and loaded air-tubes, go on mutually acting and reacting upon each other, until at length death takes place.

I shall illustrate these different courses which erysipelas takes by two or three examples from my hospital case-book. I have said that erysipelas of the head and face frequently begins in the throat; the following case commenced in that way, but the throat affection was more than usually severe, and attended with redness and difficulty of swallowing.

Case XLII* (vol. xxx, p. 126).—John Lawrence, fifty-one years of age, of rather intemperate habits, after suffering from a bad sore throat for more than a week, with great restlessness, was attacked with vomiting, shivering, and headache; and these were followed, the next day, by erysipelas of the face.

He was admitted on the 2d of April, 1850, the fourth day from the shivering. His nose was then of a deep crimson red, and this extended to the eyelids; there was much œdematous swelling and great pain, and he suffered from faintness and sickness. His pulse was 86. A dose of the hospital magnesian aperient mixture was ordered, and liquor ammoniæ acetatis with excess of carbonate of ammonia.

On the 3d, the day after admission, the erysipelas had not extended on the face, but an erysipelatous blush was observed over the fauces; he had been unable to sleep in consequence of the pain in his head, which continued very severe.

Ten grains of the compound ipecacuan powder were ordered.

On the 4th, after a sleepless night, his throat was extremely painful, and he was unable even to swallow his saliva. Chloric ether was now given, and between two and three drachms of wine every hour. After this the erysipelas subsided in his face, but he still complained of acute pain in his head, and this, and the throat affection, remained the most prominent features of the case. His pulse throughout had not exceeded 88. A stimulating gargle was ordered for his throat.

On the 9th he commenced taking two grains of quinine every

* Reported by Mr. Dickinson.

four hours, and on the 12th some morphia at night. At the end of the first fortnight a very great improvement had taken place, and he left the hospital, about three weeks after his admission, quite well.

The next case is an example of erysipelas of a very erratic type, beginning in the trunk and wandering at length to the neck and head.

Case XLIII* (vol. xvii, p. 100).—Maria Marshall, a married woman, aged thirty-seven, was admitted into the hospital in a state of general ill health: she was pale, weak, and low, with some enlargement of liver and œdema of the lower extremities.

She was doing well until leeches were repeatedly applied over the liver. After the last application, July 17th, 1846, she was attacked with shivering; she had several rigors during the day, and towards evening became delirious. On the following morning an erysipelatous redness was observed extending from the leech-bites up to the right mamma and shoulder: she had a hot, dry skin, much thirst, a thickly furred tongue, a high pulse (100), and an anxious face.

The erysipelas continued to extend. On the fourth day some carbonate of ammonia was ordered, also a little wine, and repeated hot fomentations. The next day the wine was increased to half an ounce every two hours.

The inflammation now extended to the patient's back, and assumed an erratic character. From the back it spread to the neck, and thence to the face, accompanied with great redness and swelling. There were some delirium and continued fever.

By the 3d of August, about the eighteenth day, she was very much better; free from fever and pain, and the erysipelas was rapidly subsiding. This improvement continued; but she was left very deaf and weak. She was, however, greatly benefited by a tonic regimen, and left for a convalescent institution on the 31st.

A third case I shall bring before you was one of traumatic

* The notes of this case were kept by my former clinical clerk, Dr. Tanner.

erysipelas, in which severe dyspnœa and bronchitis suddenly supervened, and terminated the patient's life in a few hours.

Case XLIV* (vol. xxxiii, p. 27).—Edward Gresley, aged fifty, was admitted into King's College Hospital, under Mr. Fergusson's care, for hæmatocele. An operation was performed on the 26th of December, 1851; and the patient continued to do very well until the 8th of January, when he became very low, delirious, and generally ill. Opium and brandy were given very freely. Erysipelas soon became apparent about the wound, and rapidly spread over the groins and thighs, and some way up the back.

On the 17th, the redness was less, and he seemed generally relieved; on the 20th, it had nearly disappeared. He continued to progress favorably until the night of the 26th, when he was attacked with severe dyspnœa, and intense catarrhal sounds were heard in the bronchial tubes on listening to the chest. For this condition he was placed in Rose ward, under my care, on the 27th; his pulse was then 148 and the respirations 28; intense rhonchus and crepitus were still heard throughout the chest.

The treatment consisted in giving two drachms of brandy every half hour, with strong beef-tea, also chloric ether and ammonia every hour; and in the application of blisters to the front, and turpentine stupes to the back of the chest.

On the morning of the 28th the same symptoms of severe bronchitis continued: his pulse and respirations were 128 and 32; the breathing had become even more difficult since the night, and the prostration was extreme. The turpentine stupes were continued, and the brandy increased to three drachms every half hour, and hot-water bottles were applied to the feet and sides.

Our patient soon became insensible, and shortly after breathed his last.

On making a post-mortem examination, we found great congestion and œdema of both lungs, their surfaces being impressed by the ribs. There were some pleuritic adhesions. The bronchi

* Reported by Dr. E. Liddon, now Physician to the Taunton Infirmary.

were choked up with mucus, and the whole lung tissue was very friable, breaking down with but little pressure: this we attributed to a post-mortem change.

Incipient atheromatous disease was observed in the heart and great vessels.

This intense capillary bronchitis is a most intractable disease. I know of no treatment which affects it in the slightest degree. If you reduce your patient by giving tartar emetic, or by bleeding, or both, you intensify the œdematous condition of the lungs and accelerate the fatal termination. If, on the other hand, you administer stimulants freely, and nutritious broths, you may prolong life for a very brief period, but this is all. This capillary bronchitis occurs independently of erysipelas, and is often found in connexion with an attack of influenza, the poison of which is probably similar to, though not identical with, that of erysipelas. It occurs sometimes in gout, and likewise in connexion with the rapid development of tubercles in the lungs.

In some instances, the erysipelas commencing in the fauces spreads no further than the laryngeal mucous membrane, and it then produces what is termed *œdema glottidis*—one of the most formidable affections to which the human frame is liable. I cannot too strongly insist on the importance of keeping in mind the true pathology of this most alarming malady; for if you thoroughly understand the true nature of this complaint, and act up to that knowledge, you may, I believe, save many lives; whereas, if you are shilly-shally in your treatment, either from ignorance of the pathology of the disease, or from prejudice in not acting up to your knowledge of it, you will certainly lose your patient.

The disease consists in an erysipelatous inflammation of the laryngeal mucous membrane, which rapidly leads to swelling of the glottis, and thus to such a degree of narrowing and occlusion of the rima glottidis, as induces the most intense dyspnœa, which is quickly destructive to life. For this condition there is but one way of affording relief, and that is by immediately making an artificial opening into the trachea, below the chink of the glottis; the further below this point the opening can be

conveniently made, the better, though, as you are doubtless aware, the lower down the aperture into the trachea, the more formidable is the operation. I would especially impress upon you that you should lose no time in resorting to this step, after having become satisfied as to the nature of the attack; the sooner the operation is performed, the greater probability will there be of your saving the patient's life. Nevertheless, I would remark, that although the first step in the treatment, the providing an artificial inlet for air into the lungs, which has to be performed by the surgeon, is a very important one, still the most important part of the treatment belongs to the physician; and any one who has witnessed the proceeding must have felt convinced, that although the relief afforded by the operation is instantaneous as regards the breathing, yet the patient will still die, unless proper means are adopted to combat the local affection, and the constitutional state which accompanies it.

I had long been convinced that the ordinary so-called antiphlogistic means are not only powerless as regards the œdematous state, but tend to depress the vital powers of the patient already much lowered by the necessary surgical interference, and I determined, when opportunity offered, to pursue, from the moment of the operation, a different line of treatment.

Case XLV (vol. xlii, p. 141).—There are many present who will remember to have witnessed a case of this disease which was in Rose ward last summer. The patient, Henry Wallis, a very fine, strongly built young man, twenty-two years of age, was brought to the hospital with marked symptoms of acute œdema of the glottis. The urgency of inspiration soon became extreme. Mr. Edwards, of Edinburgh, who was then house surgeon, with great promptitude and tact, performed the operation of tracheotomy. I determined, with the assistance of my clinical clerks, Mr. Teale and Mr. Holberton, and of many of the students, to whom I am greatly indebted, that nothing should be left undone to uphold the strength of the patient, and to keep a close watch upon him through the kind co-operation of relays of men who sat by his bedside by two at a time; and I am happy to say, I have a most accurate deta

of the case from the date of the operation to that of complete convalescence. He was fed upon strong beef-tea and brandy, of which half an ounce was given every hour; ammonia and chloric æther were also freely exhibited; and as at first his power of swallowing was not good, owing to the œdematous state involving the epiglottis, injections of beef-tea, containing a considerable quantity of quinine, were thrown, at stated intervals, into the rectum. In short, this patient was well supplied with food and stimulants, but in small doses at short intervals; and, you will hardly believe it when I tell you, that from the beginning of this treatment he had scarcely any fever; his pulse from 110 in a minute on the day of the operation steadily came down to 99 on the first, 86 on the second, and 75 on the third day. On the evening of the second day, the tube was taken out of the trachea, as he was found to cough up mucus freely through the glottis; in a fortnight the wound was healing up kindly; and in a month from the date of the operation, the patient was discharged quite well, the wound being by this time perfectly healed.

Let me relate to you another highly interesting case, illustrative not only of the efficacy of this mode of treatment, but also of the evil effects of not continuing it steadily, and showing in a very marked manner how depressing to vital power is the influence of the disease itself.

Case XLVI.—A gentleman, sixty years of age, of spare habit, who had come to town for a short time, had a shivering, followed by hoarseness, amounting nearly to complete loss of voice with difficult stridulous inspiration, soon followed by great difficulty of swallowing—each attempt at deglutition giving rise to a severe spasmodic cough. I saw him for the first time late at night. The voice affection had commenced, and the breathing was slightly stridulous at that time. On visiting him the next morning between eight and nine o'clock, I found the breathing and deglutition much more difficult, and the voice worse. I now felt it necessary to ask for surgical assistance, and Mr. Bowman was requested to see him. We agreed to pursue the treatment already commenced—namely, quinine injections

every fourth hour, brandy half an ounce every hour, and such nourishing food as he could be got to swallow. We thought the operation might prudently be postponed until two o'clock in the afternoon, when it was arranged that we should meet again and avail ourselves of the invaluable judgment and experience of Sir Benjamin Brodie. At this meeting we found the patient still breathing with much difficulty, but deglutition was easier, the pulse was falling, and fever was less. Further postponement of operation was agreed to, and a third meeting appointed for seven o'clock in the evening. At this time, finding the symptoms still improving, pulse less frequent, deglutition better, and breathing easier, we began to hope that surgical means might not be necessary, and this hope was confirmed by the still improved aspect of the symptoms at a fourth visit late at night.

On the following morning (the third day of the disease) all the symptoms were still better—deglutition had become comparatively easy, and the breathing sufficiently free to remove all anxiety as regards the risk of suffocation. The treatment had been steadily pursued through the night, notwithstanding much opposition from the patient. It was continued throughout that day, omitting the enemata, and at the evening visit the symptoms were still improving; but the patient was much opposed to the frequent administration of food, and the consequent interruption of sleep, which I deemed necessary. Nevertheless, I left positive instructions that he should not be allowed to sleep more than two hours without food, until my morning visit. Immediately after I had left the house, he called the nurse to his bedside, and directed her in the most peremptory terms not to come near his bed during the night, saying that he would not submit to "doctors'" orders, and was determined to have a good long sleep. Unhappily the nurse obeyed; exhausted nature was not restored by sleep: on the contrary, he sank rapidly in the course of the night, and next morning I found him hopelessly prostrate. Nothing now seemed of any avail to arouse the failing powers of life, and in the course of the afternoon he became comatose, and died late at night.

You will, I am sure, allow me to add another case illustrati

of the good effects of the treatment I have been advocating in œdema glottidis.

Case XLVII* (vol. xlviii, p. 126).—Hugh le Fevre, aged thirty-six, was admitted with symptoms of œdema glottidis, July 24, 1855. His occupation, that of a showman, had obliged him to make undue use of his voice, and he had repeatedly suffered from sore throat. About two years before, an usually severe attack had occurred: his throat was then red and swollen, and he suffered from difficulty of breathing and pain in swallowing. The more acute symptoms passed off, but a chronic affection remained, and he continued subject to relapses. The attack which brought him to the hospital had commenced about a week before. No history of syphilis or other illness could be made out.

When admitted, he was breathing with great difficulty, thirty-two times a minute; his face was dusky and swollen, and he was evidently nearly suffocated.

Tracheotomy was performed by my advice: the breathing was instantly relieved, and the patient restored to comparative ease. After the operation the pulse and respirations were 80 and 28. Small quantities of brandy (ʒj) and beef-tea were administered every quarter of an hour, and the tracheal tube was frequently cleared of mucus.

In the evening of that day his pulse rose to 90, but the breathing remained 28. He passed a comfortable night, and was only disturbed by occasional fits of coughing. Towards the next morning (July 25th) he had some sleep, and the pulse and breathing afterwards were 84 and 26. The brandy was reduced to half an ounce every two hours. In the evening the pulse and respirations were 86 and 24 respectively.

During the two following days, the 26th and 27th (third and fourth days), he continued remarkably well, disturbed only at times by cough or sickness. On the 26th the pulse was 90, and the respirations 24: on the 27th they were 90 and 22.

By the 28th no unfavorable symptom had occurred; his pulse and breathing had fallen to 81 and 20, and the brandy

* From Mr. Wharton Hood's notes.

was further reduced to half an ounce every four hours. Some redness surrounded the wound. The epiglottis, which had been swollen and stiff, felt softer.

On the 29th he was still doing well: the pulse and breathing were 85 and 24. On the 30th the pulse was 80, the respirations 29; on the 31st they were 70 and 24. He was now more troubled by cough, and there was increased mucous secretion. On the 30th five grains of iodide of potassium were ordered three times a day.

By the 3d of August (tenth day from the operation) he felt quite well: his cough was much better and his appetite good. On the 8th he sat up for three hours, and could eat perfectly.

On the 13th the tube was removed. His breathing became difficult in the afternoon, and in the evening the tube was returned, although with some trouble. The iodide of potassium was discontinued on the 16th. On the 27th the tube was permanently removed, and no further difficulty in breathing experienced. The brandy was discontinued.

He remained in the hospital until the 8th of September; by which time the aperture in the trachea had closed; he was quite well, and had recovered his voice.

Now in looking at the statistics of these cases, you will find that the deaths are but little under a hundred per cent.; indeed it is a rare thing for one to hear of a case of recovery from œdema glottidis after the operation of tracheotomy has been performed; and it seems to me very questionable, whether similar treatment to that which was pursued in these instances ought not to be adopted in all cases of tracheotomy, for the operation is in itself a very severe one, and accompanied with great shock to the nervous system, while there is invariably great distress of the respiratory organs during its performance.

I gave you just now an instance of erysipelas of the face accompanied throughout by redness and soreness of the throat with difficulty of swallowing. There is yet another form of erysipelas which commencing in the throat confines itself entirely to the faucial region, inducing paralysis of the pha-

ryngeal muscles with utter inability to swallow and great prostration; these cases are rare and of a very fatal tendency; far more formidable indeed than those in which the throat is only affected transiently, or in common with the skin of the face and head. I need not now enter into the particular consideration of these cases, as I intend to devote a lecture exclusively to the subject.

Thus, then, you see that erysipelas not only affects the external integument of the body, but also the mucous membranes, both alimentary and respiratory. And in addition to those parts which I have already mentioned as so peculiarly liable to be fixed upon by the poison of this disease, there is one other tissue which, I suspect, it not unfrequently invades, namely, the peritoneum, producing what is known as puerperal fever, or puerperal peritonitis; a disease which I believe is really of an erysipelatous nature, and which certainly in most, if not in all instances, requires to be treated upon a thoroughly supporting plan.

When erysipelas terminates the life of a patient, it most frequently does so by inducing a state of extreme prostration, and the patient sinks exhausted. This was what occurred in the man J. H. Collins (Case XLIX, p. 170), whose history I will presently read you. A second mode of fatal termination, by the occurrence of erysipelatous bronchitis and pulmonary congestion, I have already spoken of, and illustrated by the case of Edward Gresley (Case XLIV, p. 158). In a third class of cases the patients die delirious or comatose. When this occurs, some authors would tell you that the erysipelas has induced inflammation of the membranes of the brain. These notions are now, however, almost entirely exploded, and there is ample evidence, that if death takes place while the patient is in either of these conditions, the cerebral meninges are found, upon post-mortem examination, to all appearance perfectly healthy, nor can any of the products of an inflammatory process be detected; or, if there be anything amiss with them, it is that the vessels of the pia-mater contain rather less blood than they ought to do. These formidable symptoms are rather to be attributed to the circulation of a noxious blood through the brain, and to a consequent defective and depraved nutrition of that organ, than to inflammation and its products.

Let me repeat here that the amount of congestion of the vessels of the brain is very much influenced by the mode of dying of the patient. A prolonged moribund state, with heavy breathing, and especially with convulsions, increases congestion by retarding the flow through the veins; a rapid death, with much exhaustion and faintness, as I have repeatedly remarked to you, leaves the vessels with very little blood.

I shall conclude with some remarks upon the treatment of erysipelas generally; and with especial reference to this subject, I must beg your attention to the following classification of cases, based on a due consideration of the clinical history of the disease, and the issue of different examples.

I think we may fairly arrange the various cases of erysipelas into the five following classes:—

1. There are certain cases of erysipelas which get well of themselves, and these are generally examples of the disease in a slight form, affecting the head and face only, or some other limited portion of the skin. Usually in the course of two or three days, especially if they are kept in a comfortable place, and have a little beef-tea or other light nourishment, patients suffering from attacks of this intensity recover, and this, too, in some cases, despite of a certain amount of lowering treatment, and of the use of such remedies as tartar emetic.

2. In a second series of cases of this disease, the very opposite of the first, the patient dies downright, if I may use the expression: he sinks rapidly, do what you will. This mode of termination is common to erysipelas, with most of the other diseases which are due to the influence of a poison, whether it be generated in the human body, or be of atmospheric origin. Thus, in a considerable proportion of cases of cholera, it is vain to attach very much importance to anything in the way of treatment, for the patient is dead almost before the case comes fairly under observation; and, in such instances, I doubt if any plan of treatment ever will avail, because the accession of morbid phenomena is so very rapid. The same is also sometimes observed in typhus fever, the patient being killed within a very few days, or even hours, of the commencement of the attack; and so likewise, in all the exanthemata,—small-pox, measles, scarlet fever, &c. In these extreme cases treatment

is often utterly unavailing, and death occurs during the first twenty-four or forty-eight hours of the illness.

Hence, then, it becomes necessary, in all endeavours to estimate the value of any particular plan of treatment in erysipelas, carefully to exclude from the data upon which conclusions are to be founded these two classes of cases—first, those which would get well of themselves; and, second, those which defy every attempt at treatment.

3. The third class comprises those cases which recover under a suitable plan of treatment, but in which is a marked tendency to death, and which there is good reason to believe would terminate fatally if left to themselves.

4. The fourth group consists of those cases which pass through the early stages of the malady more or less favorably, but which then exhibit the secondary phenomena of the disease. In this class complete recovery may take place, or death may result from the exhaustion which is frequently induced by the extension of the suppurative process, and by its duration.

5. In the fifth and last class, not only do the secondary phenomena of the disease manifest themselves, but, by some means a morbid material finds its way into the circulation, and as a result of this, formations of pus take place in various parts of the body, and the patient dies of purulent infection, or pyæmia, as it is called.

The treatment of erysipelas which I have for many years past adopted, is the supporting plan; and this I would, from a long experience, recommend to you, under the conviction that it is the best adapted to save life, and check the progress of the disease; and that under it, if begun early and with decision, you will seldom have to deal with the secondary phenomena of the malady.

The treatment consists in the free administration of food and stimulants, such as beef-tea, and some form of alcohol—brandy by preference—in precisely regulated quantities, at stated and short intervals; if drugs are needed, ammonia, bark, and chloric æther, in forms most agreeable or least offensive to the stomach, may be given. The beef-tea, or other animal broth or soup, and brandy, should be given at

stated times, in small doses, two or three ounces of the former, and from two drachms to half an ounce or an ounce of the the latter slightly diluted with water. Two different forms of alcoholic fluid should not be given at the same time, such as wine and brandy, or beer and brandy, or gin and brandy; and for other nourishment it is desirable to observe the same rule, as far as possible. You must attend closely to the digestive power of your patient, and be careful to avoid exciting dyspeptic symptoms, such as nausea, sickness, hiccough, flatulence, by giving too much at one time, or by too great a variety of stimulants or food.

Sometimes in the course of an attack of erysipelas, the patient may become delirious, or he may fall into a state of coma. It is during the first fourteen days of the illness that these formidable symptoms are most apt to occur; hence the necessity of beginning early, from the first, with support and stimulants, which you will find preventive both of delirium and coma. The lower you keep your patient, the greater will be the tendency to delirium or coma, and the more violent or profound will either be, and the development of either is an indication for pressing the treatment in the same or greater doses. Sometimes you will find that the coma persists, notwithstanding all the support you can give; and then you may generally conclude with certainty that the blood has become poisoned by pus, or some other morbid agent, and that death from pyæmia is about to occur, or that local formations of pus are about to be developed in various parts of the body.

In those cases in which the disease responds to the stimulating treatment, the delirium subsides, and speedily altogether disappears; the redness and swelling diminish; the pulse becomes softer, fuller, and less frequent; the fever decreases, and the state of convalescence is rapidly established.

Sometimes, through feeble powers of digestion in the patient or injudicious zeal on the part of the attendants, you may find that you are over-stimulating. What are the indications of this? They show themselves in sickness, in flatulence, in a sense of oppression, perhaps also in derangement of bowels. When such symptoms occur, nothing can be easier than to suspend the treatment for a few hours, to give only a little co

water, and afterwards to resume it cautiously in diminished quantities.

Case XLVIII* (vol. xlvii, p. 13).—This case serves as a good illustration of the effects of over-stimulation. Ellen Sutcliffe, aged thirty-four, admitted into King's College Hospital, on April 25th, 1855, with a severe attack of erysipelas of the head and face. Pulse 112; respiration 28. This patient was at once ordered plenty of beef-tea, and the following draught every hour:—Ammon. carb. gr. ij, sp. æther. chlor. ℥x, sp. vini gallici ʒiv, aquæ ℥ij. For the three next days the erysipelatous inflammation gradually spread, and the pulse maintained its frequency, and on the 29th the draught was ordered to be given every half hour. On the 30th, however, retching and vomiting manifested themselves, and the patient became extremely prostrate, and it appeared as though the vomiting had arisen from the excessive amount of stimulants, or, perhaps, rather of ammonia and chloric ether, for the brandy had been mixed with the physic, in order to ensure its being administered. She was now, therefore, ordered simply half an ounce of brandy every half hour, and a little bark and ammonia in effervescence every third hour; and from this time the case proceeded in the most favorable way. The stimulants were gradually diminished as the pulse fell, and the erysipelas subsided; and, without the occurrence of any secondary abscess, this patient was discharged from the hospital quite well, after a stay of little more than three weeks.

The following case may serve to warn you of the danger likely to arise from delaying the administration of nourishment and stimulants until the worst symptoms have showed themselves. When this patient was first admitted, his pulse was only 88; but twenty-four hours afterwards it was 112, and he was hopelessly prostrate: the time for treatment was then well-nigh passed. Had that day been spent in the steady employment of the treatment I have recommended, I think it very probable that the patient, who was a favorable subject for the treatment of acute disease, might have been saved.

* This case was recorded by Mr. Hardwich.

Case XLIX (vol. xxxi, B. p. 36). J. Howard Collins, aged twenty-seven, of temperate habits and general good health, was admitted with erysipelas of the face, May 3d, 1850. He stated that he had suffered from a similar attack two years previously; that the present one had commenced, five days before, with a small boil on his nose, which rapidly increased in size; at the same time he suffered from shivering, which he attributed to exposure to cold. The inflammation rapidly extended from his nose over the greater part of his face, which was of a dark rose red when he came to the hospital. He then complained of loss of appetite and want of sleep, headache, thirst, and pain in his right side; his pulse was then only 88. A little carbonate of ammonia formed the whole treatment at first.

By the 4th, the inflammation had extended to the scalp, the pulse became feeble and rapid—112, and the prostration great. His nose was of a livid hue and covered with minute pustules, and it was thought advisable to make a small incision into it. Two drachms of brandy were given every half-hour, and half a drachm of chloric ether with each dose of the carbonate of ammonia every four hours. In the afternoon the brandy was doubled, and fifteen minims of Battley's liquor cinchonæ given every two hours. The prostration, however, increased, and difficult respiration came on; the brandy was again doubled, an ounce being given every half-hour; but even this active treatment was utterly powerless against the state of hopeless exhaustion into which he had so rapidly fallen. The difficulty of breathing increased, a cold perspiration broke out, and he died early on the second day after admission.

Almost the only morbid condition found at the post-mortem was the evidence of recent pleurisy on the right side. The transverse colon lay in front of the liver, and had made a depression on the left lobe.

The upshot, then, of all I have to tell with respect to the treatment of erysipelas is to give stimulants and nourishing food freely, and from the very commencement of the attack. Don't trouble yourselves with too much attention to the secretions, as some are apt to do, who imagine that the alteration of these by gray-powder, black draught, *et hoc genus omne*, is

necessary to the favorable issue of the case, but who, by the time they have got the secretions into what they conceive to be a correct condition, find that their patient is fairly slipping through their fingers, and is dying, worn out and exhausted. As soon as you are satisfied that the patient to whom you are called is labouring under erysipelas, at once begin to administer stimulants and nourishing food, using the precautions I have mentioned; and what I wish above all things to impress upon you is, that this stimulating treatment should be employed from the very beginning of the attack. With respect to the bowels, you must be guided by circumstances; if they are confined, you may open them by an enema, or by a dose of castor oil, or some other medicine, which will neither irritate the mucous membrane of the alimentary canal, nor exhaust the patient's strength; always keeping in view that the poison of erysipelas is exceedingly depressing in its action, and that the object of all your treatment should be, first, to antagonise the poison, and, secondly, to uphold the patient's powers, to enable him to bear up against one of the most lowering and debilitating diseases to which the human frame is liable.

Now of all the stimulants, I believe, as I have already said, the alcoholic are the best, and I have witnessed such remarkable effects, in such a variety of cases, produced by their free exhibition, that I am inclined to consider them as *antidotes* to the erysipelatous poison. If I were to be restricted to any one remedy in the treatment of this disease, I should, assuredly, choose brandy. With a commissariat well supplied with brandy and simple means to keep the bowels open, I think I could engage to keep erysipelas at a minimum among the wounded in our army in the Crimea.

Some attach great importance to the use of the tincture of sesquichloride of iron in this disease. I have no doubt many cases, such as those which I have placed in my first group, will get well under that drug, partly and mainly because it excludes depressing treatment, partly, perhaps, from some tonic power in the medicine; but I would as soon think of trusting to it in the treatment of the third or fourth group of cases, as I would to the billionth of a grain of aconite, or arnica, or sulphur, or any other homœopathic absurdity. The remedy, so far as I

know, is unobjectional in itself, but its power to do good is small; and if you try it, let me advise you not to trust to it alone, but merely to use it as an adjunct to the treatment which I have endeavoured to impress upon you to-day. For, as I before said, there is a large class of cases of erysipelas which will get well without any treatment whatever, and, indeed, in spite of depressing treatment, either because the dose of the poison which these patients have imbibed has been very small, or because their powers of resisting acute disease are very great. In such cases you may, if you like, amuse yourselves with giving a remedy of the nature of sesquichloride of iron. But in all severe examples of the malady, place your trust in food and brandy, freely given under careful regulation, and adopted from the very commencement of the attack.

The following cases are worthy of your attention as good illustrations of the effects of the treatment I have recommended in this lecture.

Case L* (vol. xlviii, p. 80).—Thomas Lane, aged fifty-three, a copper-smith, of intemperate habits, admitted into King's College Hospital on June 1st, 1855, in a state of violent delirium, with erysipelatous inflammation of the right cheek and with signs of slight suppuration in the right upper eyelid. It appeared that this man had been formerly in the habit of working in distilleries, where he was constantly exposed to the fumes of spirits, and to counteract the intoxicating effects of the inhalation of these, he used to drink a very large quantity of beer; but that he had latterly changed his occupation for that of a plumber, and while following this employment he had been continually exposed to wet from the escape of water from pipes. The attack of erysipelas, for which he was brought to the hospital, commenced about a fortnight prior to his admission, with pain, redness, and swelling over the right eye, which symptoms gradually increased in severity until he was brought under observation on June 1st. By this time delirium had manifested itself, and he was so violent, and created so much disturbance that it was found necessary to place him in a ward apart from

* From the record of the case by my clinical clerk, Mr. Wharton Hood.

the other patients. Pulse 86; respirations 30. The treatment upon which he was put consisted in the free supply of beef-tea, milk, arrow-root, together with half an ounce of brandy every two hours, and twenty drops of laudanum every four hours. The following day an incision was made into the right eyelid, and a small quantity of pus escaped, and the delirium and other symptoms still continuing unabated, the brandy was increased to half an ounce every hour, and bark, chloric æther, and ammonia in effervescence, were substituted for the laudanum. Pulse 76; respirations 24. The day succeeding this, the 3d, the delirium had considerably diminished. Pulse 74; respirations 22. On the 4th, the pulse was 74, and the respirations 20; and on the 5th, the erysipelas was rapidly disappearing, the delirium had entirely ceased, the pulse was 56, and the respirations 22. The stimulants were now diminished gradually from day to day, meat and porter being substituted for brandy and beef-tea, and on the 9th the patient was convalescent.

Case LI* (vol. xlviii, p. 54).—J. Scarfe, aged forty-seven, a relieving officer, of temperate habits and good general health, was admitted into King's College Hospital on May 17th, 1855, with a severe attack of erysipelas of the face and head. Pulse 110; respirations 38. He was at once put upon half-ounce doses of brandy every two hours, with a plentiful supply of beef-tea, &c. A week previous to this man's admission to the hospital he was seized with shivering and vomiting, and this was speedily followed by redness and swelling of the nose. The erysipelatous inflammation rapidly spread, and involved the whole of the face and temples, the eyes being completely closed. The day following his admission, the pulse was 104, and the respirations 42; and the day after this, the 19th, the report of him is this:—"He is rather worse to-day: pulse 112; respirations 38: he had no sleep last night, and is now very restless, and wanders occasionally: tongue thickly coated with brown fur; the swelling of the face is slightly diminished." The brandy was now increased to half an ounce every hour, and on the 20th, the pulse had fallen to 96; and on the 21st, this report is entered

* Reported by Mr. Wharton Hood.

in the book :—"The redness and swelling have entirely subsided except under the right eye: pulse 88; respirations 35: he sleeps well, the tongue is cleaning, and he is asking for meat." On the 22d, the brandy was diminished to half an ounce every two hours, and the convalescence was now rapidly established, the only indication of suppuration, anywhere, being the existence of a very small collection of pus in the right upper lid, which was opened on the 23d. On the 26th, all brandy was withdrawn and the patient put on porter and quinine; and a few days subsequently he was discharged quite well.

Case LII* (vol. xlviii, p. 70).—Martha Tasker, aged twenty-one, a healthy-looking girl, with a marked hysterical countenance, admitted into the hospital on May 23d, 1855, for pain in her left side,—which appeared to be hysterical,—and occasional headache. For this she was put on steel and a pretty liberal diet, and on June 8th was ordered to leave off all medicine, as she was reported to be "nearly well." Three days after this, however, it was found that she was attacked with erysipelatous inflammation in the right eye and side of the face, which was red and swollen. She now complained much of pain in the head and giddiness, and she was at once put upon half-ounce doses of brandy every third hour. It almost appeared as though this treatment put a stop to the progress of the disease, for the erysipelas ceased to spread and in three or four days the face was almost well. On the 14th, however, the right arm became the seat of the erysipelatous inflammation, which, treated in a similar way, rapidly disappeared.

Case LIII.—M. E., aged twenty-two, a needle-woman, greatly over-worked, was admitted into King's College Hospital on May 9th, 1855, with a severe attack of erysipelas, involving the whole of the face, which had commenced four days prior to her admission into the hospital. It appeared that this patient had had two previous attacks of the disease, one nine and another six years before the present.

On admission her pulse was 116, and her respirations 26, and she was very restless and quite unable to sleep

* Reported by Mr. Wharton Hood.

Plentiful supplies of beef-tea were ordered her, together with half an ounce of brandy every hour. On the 11th the pulse was 120 and the respirations 30; and on the 12th the erysipelas exhibited a tendency to spread to the scalp, but the pulse had fallen to 118, and the respiration to 22.

On the 14th an immense improvement had taken place: the swelling had very much diminished, desquamation was beginning to be established, and the pulse had come down to 89, while the respirations were 22. On the 15th the report is as follows:—"The swelling has almost entirely subsided; the cuticle is peeling; the pain in the head is quite gone; the pulse 87; respirations 22."

The stimulants were now gradually diminished, and on the 16th the pulse was 72 and the respirations 20; on the 17th, pulse 66, respirations 18; and on the 18th she was fairly convalescent, the pulse being 62, and the respirations 16, while the only appearance of a secondary abscess was a small pustule on the margin of the left upper eyelid. Full diet, porter, and quinine were now gradually substituted for the brandy and beef tea; and the patient was discharged quite well on May 26th, 1855.

Case LIV* (vol. xliv, p. 215).—Elizabeth Knight, aged fifty-six, a married woman, who had had fourteen children, was brought into King's College Hospital on March 15th, 1855, with a curious chorea-like shaking of the right arm, and erysipelatous inflammation of the right cheek. About four weeks previous to her admission to the hospital, she suffered a great fright from waking up one morning and finding a niece, who was sleeping with her, lying dead by her side, and a few hours afterwards she was seized with chorea-like jerkings of the right arm, which have continued ever since, but which cease entirely during sleep. From that time she remained in a very weak and low state, fainting occasionally, until March 11th, when she was taken with pain, redness, and swelling in the right cheek, and violent shivering; and on the 15th she was brought to the hospital. Her pulse was then 68, exceedingly weak, and her respirations 48, loud rhonchus being audible all over the

* From the notes of my clinical clerk, Mr. Dunn.

chest. The treatment to which she was subjected consisted in the exhibition of half an ounce of brandy every two hours, five grains of carbonate of ammonia every four hours, with an opiate draught at night, together with free supplies of beef-tea. On the 16th, the erysipelas was evidently spreading, the pulse was 86, and the respirations 46; the brandy was now increased to half an ounce every half-hour. On the 17th, the whole of the right side of the face was involved, and violent retching had come on, all the brandy taken being almost immediately returned. The brandy was therefore at once left off, and an ounce of wine was given her in an effervescing water every half-hour. On the 19th, though the erysipelas had involved the whole of the face, yet the patient appeared decidedly better, the convulsive movements of the arm and the vomiting having entirely ceased. On the 20th, the erysipelatous inflammation was rapidly subsiding, and desquamation of the cuticle was commencing. The pulse had now fallen to 94, and the respirations to 20. From this time the patient gradually improved, the pulse coming down a few beats daily, while, as in the other cases, the stimulants were by degrees lessened and lessened until, on the 30th, all erysipelas had gone, and the patient was put upon porter, quinine, and a generous diet. The only suppuration which occurred in this case consisted of a little pustule on the edge of the right lower lid, and a small abscess in the right armpit, both of which speedily got well. The convulsive movements of the arm never returned while this patient was under observation after their first disappearance on the 19th.

Case LV* (vol. xxi. p. 168).—James Webster, a temperate man, but past middle age, and therefore not the most favorable subject for acute disease, was brought into the hospital on the 23d of October, 1847, in a state of high delirium and quite unconscious, apparently suffering from severe erysipelas of his head and face, which were red and covered with a branny scurf; there was also a large slough over the sacrum.

For some time after his admission he appeared in a dying state, and passed his evacuations unconsciously. He was

* Reported by Mr. Lakin.

ordered a full dose of opium, strong beef-tea, half an ounce of wine every two hours, and carbonate of ammonia; under this treatment he rallied, and partially recovered his consciousness by the afternoon of the 24th, but could remember nothing about the commencement of his illness.

He continued for some days in a drowsy, wandering state, but could be roused, and then answered questions rationally. There were very considerable muscular tremors, such as we see in bad fever cases and drunkards. His pulse was 108, small and feeble. An equal quantity of brandy was ordered in place of the wine, and he was put on a water-bed.

On the 28th, he was much better,—quite conscious and rational: pulse 96, but still very feeble: the redness had quite disappeared: the slough on the sacrum seemed alone to prevent a rapid recovery. On November 3d, he was ordered a mixture of quina and sulphuric acid; also a pint of porter and six ounces of wine instead of the brandy. On the 10th, the slough separated. On the 18th he was much better: his tongue clean, and appetite good. His convalescence was rather slow; but taking into consideration his age, and the severity of the attack, it was, on the whole, most favorable, and he was discharged, well, on the 18th of December.

Case LVI* (vol. xxxi, B. p. 121).—John Jones, aged forty-four, a man of intemperate habits, after a fall while intoxicated, was attacked with erysipelas of the face, for which he was admitted into the hospital, April 27th, 1850.

His face was then much swollen, and of a deep-red hue from the inflammation. The usual constitutional symptoms were present—hot and dry skin, thirst, loss of appetite, pulse 84. He was ordered a quart of strong beef-tea, a small quantity of brandy and chloric æther. The brandy was increased the next day to half an ounce every two hours. On the 30th, he became very delirious and would not stay in bed; his pulse was feeble and had risen to 100; the brandy was increased to half an ounce every hour. By the following day the erysipelas had somewhat extended, but the delirium abated, and his pulse

* Reported by Mr. J. C. Dickinson.

attention to the peculiar characters manifested by it, as instances may occasionally come under your own notice, and the disease being of a very fatal tendency if not at once treated with great decision, it is of the utmost importance that you should immediately recognise it. The cases are, if I may so speak, of the kill or cure class: the symptoms last but a short time, and the disease runs its course, whether for life or death, in a brief period. If the treatment be vacillating, you will probably lose your patient; but if you have decided upon the right line, and pursue it promptly and steadily, you need scarcely ever lose a case. For these reasons, gentlemen, I shall beg your attention to-day to the particulars of some extremely interesting examples of this affection, one of which is still in the hospital; but first let me give you, very briefly, a connected outline of the characteristic features of the malady, and of the treatment I have found effective for its cure.

The peculiarity, then, of the disease consists in this:—that the force of the poison seems to fall upon the pharynx and to paralyse it; and it must do this, either by benumbing the sensitive nerves, through which the muscular contractions are usually excited by the contact of food, or by extending to the muscles themselves and paralysing them directly, or, it may be, in both these ways. If you look into the throat of a patient labouring under this affection, you will find the pharyngeal mucous membrane exhibiting a peculiar dusky-red colour, the fauces will be perfectly open, and you will be unable to discover any mechanical impediment to free deglutition; and if, now, with your finger, or a pen or probe, you touch the back of the pharynx, you will find that none of the pharyngeal muscles are thrown into action, as they invariably are in a state of health; in other words, you cannot excite the reflex actions necessary for deglutition; and if you give the patient something to swallow, as soon as he gets the liquid or solid, whichever it be, upon the back of the tongue, instead of its being grasped by the contraction of the muscles of deglutition, and guided, as it were, into the œsophagus, in consequence of the

inflammation spreads externally, and I gave an instance (Case XLII, p. 156) in which the throat affection was throughout the most prominent feature: I think it must be to such cases as these that Dr. Elliotson alludes.

complete palsy of these muscles, it falls by its own gravity into the larynx, and is thence immediately ejected, by a powerful expulsive effort, through the mouth and nostrils.

I have seen, and have notes of several of these cases which have occurred both in hospital and in private practice, and all those which were treated upon the antiphlogistic plan died; but for some years past now, from being better acquainted with the true pathology of the malady, I have invariably resorted to an opposite mode of treatment,—an actively supporting and stimulating one, precisely that, in fact, which you have heard me advocate as most beneficial in œdema glottidis—except that we can get on here without calling in the aid of the surgeon—and since I have pursued this practice, I have scarcely met with an instance of this affection which has not recovered.

In these cases, the patient is apt to die, not from want of air, as when the erysipelatous poison attacks the mucous membrane of the larynx, but from want of food, the inability to swallow being even greater than in cynanche tonsillaris, which creates a mechanical obstacle. The difficulty therefore in the treatment is to get sufficient nourishment into the patient; but if a person cannot gain admittance into a house through the front door, the first thing to which he would most probably resort, if it were very important that he should gain entrance at all, would be to try at the back door. So here, since there is an almost insurmountable difficulty in introducing food into the stomach through the mouth, the only resource left us is to throw beef-tea injections, containing large doses of quinine, into the rectum, and feed the patient in this way. Then the back of the fauces should be lightly touched with the solid nitrate of silver, or freely washed with a strong solution of it; and as soon as the power of swallowing begins to return, which it generally does, under this plan of treatment, in the course of from twenty-four to forty-eight hours, frequent and large doses of brandy, ammonia, chloric æther, and beef-tea, should be exhibited by the mouth. If, however, from any cause, the plan of feeding by the rectum fails to restore the power of deglutition, you must then have recourse to feeding by the stomach-tube; but this mode of proceeding is generally unsatisfactory, and must only be resorted to when all the

other means, which I have just mentioned, have been fairly tried and found unsuccessful. Very patient spoon-feeding, carrying the food as low down in the fauces as possible, will often be found successful. A skilful and practised nurse will succeed in this where a more scientific person would fail.

Case LVII* (vol. xxxiii, p. 191).—The first case I shall speak of is that of the man George King, in Sutherland ward, aged sixty-four; he is tall, and of spare build, and he looks much older than the age he gives, and appears as if he had been suffering for some time from illness. This condition, he tells us, resulted from a severe attack of rheumatic fever, under which he suffered about a year and a half ago, and which was accompanied with cardiac affection.

Since that attack, he has been more or less an invalid, from a succession of catarrhal affections, up to Friday, the 26th of April, 1851, on which day, while he was crossing St. James's Park, under a sharp north-east wind, he was suddenly seized with a sensation as if something had forcibly closed his jaws, and he found, to his alarm, that he could not open his mouth with the most violent effort. The affection at the same time seemed to attack the throat, and completely to prevent his swallowing. When he arrived at Pimlico he could not eat his dinner, and was unable to swallow some gin and water which he attempted to take. He could get it into his mouth, and it even reached the pharynx, but would go no further; it seemed, to use his own expression, "to stick in his throat," and was soon returned. He made frequent and ineffectual attempts to swallow, but, from this time till the following Tuesday, he remained without taking a particle of nourishment.

When he was admitted into the hospital, attempts were made to examine his throat, but, in consequence of the state of trismus which prevailed, the teeth could not be separated sufficiently far apart to admit of any examination being made. I will read you the following extract from notes made at the time of his admission by Dr. Salter, who then ably filled the office of house-physician: "On attempting to look into his mouth, I found that I could not separate his jaws more than about one

* The record of this case was kept by Dr. Bridgwater.

eighth of an inch,—just a chink. I gave him some water, which he attempted to swallow. He performed the buccal and lingual, and, to a certain extent, the pharyngeal part of deglutition, but then, with a spasmodic effort to get it further, he choked, his eyes looked as if they would dart from his head, and up it came. Repeated attempts merely led to repeated failures. I could neither look into his throat, nor insert my finger to feel it."

At my own visit, in the afternoon of the same day, just the same state of things continued to exist,—deglutition was quite impossible, the attempt to swallow was followed by the forcible ejection of the food through the mouth, and in some degree through the nostrils, by the agency of a spasmodic expiratory effort.

In considering what might be the affection calculated to give rise to these symptoms, I first thought of stricture of the œsophagus; but, upon inquiry into the history of the case, it seemed very unlikely that the difficulty of deglutition could arise from this cause. The affection came on suddenly, which is not the case in ordinary stricture of the œsophagus. Stricture of the œsophagus generally results from cicatrization of ulcers and contraction of the tissues adjacent, or from some malignant formation round the tube, and such strictures are most apt to occur in the lower part of the tube not far from the cardiac orifice. Did such a stricture exist, the dysphagia would have been of a different kind. Here matters were suddenly and forcibly rejected, while in the former condition the food is swallowed to a certain point, and seems to encounter an obstacle at a certain situation, to which the patient points with his finger; then it is either stopped completely, and afterwards ejected, but not with the force and rapidity which we observed in this case, or, by a considerable effort, it is made to overcome the obstacle. A spasmodic state of the œsophagus might create a dysphagia like that noticed in this case, but the symptoms would scarcely have come on so rapidly, nor would the danger of choking have been so imminent as in our patient. It seemed to me, that the seat of obstruction or difficulty was not so low as the œsophagus, but rather in the pharynx, and that the food encountered it immediately on passing from the mouth.

Difficulty of deglutition sometimes results from an affection of the medulla oblongata; in such cases the dysphagia does not come on suddenly, and is not accompanied by the acute symptoms present in this case. The attempt at deglutition is in both cases quite alike, and its failure is from a similar cause, namely, spasm of the glottis, excited by the food coming into contact with the mucous membrane in this region, instead of being guided downwards into the œsophagus by the contraction of the pharyngeal muscles; but in the one the affection is acute, in the other it is chronic.

Aneurisms, again, frequently give rise to dysphagia: difficulty of deglutition is often a most important diagnostic mark of thoracic aneurism; but this, of course, comes on gradually, as the dimensions of the aneurismal tumour increase, and the dysphagia is by no means of the complete kind which occurs in the affection under our consideration.

Another cause to which we might refer these symptoms is inflammation of the epiglottis, which is capable of producing great difficulty of deglutition; and, if we bear in mind the position of the epiglottis, we shall not be at a loss to conceive how this takes place. If the epiglottis be enlarged, it lies as a tumour between the rima glottidis and the base of the tongue, and would not only offer some degree of obstruction to the passage of the food down the pharynx, but it would prevent that complete apposition of the root of the tongue to the rima glottidis which is necessary for the perfect closure of that chink. When the epiglottis is diseased, the difficulty of swallowing arises from the tendency of the food to pass into the larynx, the effect of which is, a violent spasmodic action of all the muscles of the part, and forcible projection of the food upwards into the posterior nares. Thus this peculiar mode of regurgitation of the food becomes a most valuable point in the diagnosis of a diseased state of the epiglottis; for you will always find that, where difficulty of deglutition arises from an inflamed or ulcerated state of the epiglottis, the food is thrown upwards into the nose, and is frequently forced out of the nostrils with considerable violence. The dysphagia in both these cases is very similar, although not exactly alike; in our case the food was chiefly rejected through the mouth; only a small portion of it

passed through the nostrils. Moreover, in disease of the epiglottis, there is more or less affection of the voice; in our patient that function was not impaired. But conclusive evidence is obtained from feeling the epiglottis with the finger: in our patient it was soft and flexible; in inflammation it would have been swollen and stiff. Hence I was led to exclude epiglottidean disease from my diagnosis.

Again, it might have been a case of common cynanche tonsillaris. If we could have opened his mouth, this point might have been decided at once; but the complication of trismus hindered our efforts at diagnosis very much. However, I decided against cynanche tonsillaris upon the suddenness and force of the rejection of the food. In cynanche the deglutition is extremely difficult and painful, and made with great effort and suffering; but it is not impossible, nor is rejection of the food a constant symptom; and when it does occur, it is not made in that violent sudden way which we observed in this case, nor with the signs of choking.

If, then, there was no cynanche, no stricture, no laryngeal or epiglottidean disease, no aneurismal or cerebral affection, what could have caused this remarkable dysphagia?

When I had examined the patient, some other cases were brought forcibly before my mind, and I felt certain that the peculiar symptoms in the present instance resembled those which I had observed on previous occasions with great interest. The first occurred to me many years ago in private practice.

Case LVIII.—I was called to see a lady who had for some days been suffering from influenza. The principal affection, however, was great difficulty of deglutition. When she took any food, it evidently reached the isthmus faucium, but there it seemed to excite choking in consequence of a portion apparently passing into the larynx, whence it was repelled with considerable force; so urgent was this difficulty of swallowing, that it was impossible to give her food, for fear of producing suffocation. When I looked into her throat, I saw that there was no mechanical impediment to the passage of the food into the pharynx; and all that I could observe was slight redness of the velum, and a dusky hue of the mucous membrane of the pharynx.

I found, also, that no amount of stimulation of the mucous membrane of the velum would excite contraction of the palatine or pharyngeal muscles. Believing that the phenomena depended upon an inflamed state of the mucous membrane of the throat, and not having yet learned that the proper antiphlogistic in such cases is support and nourishment, I was content to trust in the application of blisters and leeches to the throat. But my patient became speedily more exhausted, and rapidly sank.

Case LIX.—The second case occurred in the person of a middle-aged woman, the wife of a respectable tradesman in Westminster. I had no difficulty in immediately recognising the similarity to the former case; the symptoms were just the same: if fluids were given, they ran down into the larynx, and were expelled with cough; and any mechanical stimulus failed to excite contraction of the velum. There was dusky redness of the velum and pillars of the palate. Profiting by former experience, I determined to exclude all depressing treatment in this case. I ordered injections of strong beef-tea with ten grains of quinine to be given every four hours, and I freely applied the solid nitrate of silver to the fauces. The injections were given regularly, and the next morning I found my patient much better: she could swallow a little, and appeared stronger. I now ordered her to take beef-tea and wine cautiously by the mouth, in small quantities at a time, and the quinine was also given by the mouth. In less than forty-eight hours more she had completely recovered.

Case LX.—The third case was that of a gentleman of fortune whom I attended at one of the hotels in my neighbourhood, in the month of March, when erysipelas was rather prevalent about town. The symptoms under which he laboured were very similar to those which I have just detailed in relating the other cases. There was great difficulty of deglutition, but he had some power of swallowing; there was also the same tendency for fluids to pass into the larynx; the muscles of the palate showed the same want of contractile power; the mucous membrane was of the same dusky-red hue. This patient was treated by support and stimulants, and the local application of nitrate o

silver. As he was in affluent circumstances, I gave him turtle-soup and port wine, and quinine. He got quite well in a few days.

Case LXI.—Not long since I was called to see a woman who was suffering from symptoms of the same character. In this, as in the other cases, it was impossible, by any amount of stimulation, to excite contraction of the muscles of the soft palate; and if we administered a small quantity of fluid, it ran down into the glottis, causing violent irritation and choking. She had been ill some days, and had been treated by leeching, blisters, and mercury, as in my first case, and was in a state of extreme exhaustion when I saw her. The isthmus was quite open, and there was no impediment to deglutition except the paralytic state of the muscles. This patient died after an illness of two or three days: she died, indeed, while I was in the house.

You see that these were all very serious cases, and that I am justified in calling them kill or cure cases, so brief is the period of their duration whether for weal or woe; and, although they are rare cases, you will, I am sure, appreciate the importance of being prepared for them, so that you may not be at a loss if perchance one should fall to your care. Though these are formidable cases to witness, and run their course very rapidly, terminating in death or recovery within forty-eight hours, they are, I believe, if taken soon enough, perfectly amenable to treatment, and I feel persuaded that the first case which I described would have recovered, if she had been treated on a different plan. At the time that it occurred to me, I was not familiar with the symptoms—I was not "up to the disease," if I may so express myself; and I hold the opinion, that such cases would almost always recover if subjected to proper treatment before extreme exhaustion had come on.

Now, believing that our patient upstairs was suffering in the same way as those others whose cases have been related, I put him on the same treatment as that which I had followed in the successful cases: ten grains of quinine diffused in two or three ounces of strong beef-tea were administered every four hours in the form of an enema. I should have also ordered the nitrate

of silver to be applied to the throat, but that the state of trismu prevented his mouth from being opened.

On the following day he was very much better: his mouth could be opened to the extent of half an inch, and he could swallow a little liquid.

On the 30th, he could open the mouth sufficiently to enable Dr. Salter to examine his throat; and then, although considerable progress had been made towards recovery, the mucous membrane of the upper and back part of the pharynx was found of a purplish, dirty-red colour, indicating, as I thought, the existence of a low erysipelatous inflammation. There was no swelling of the mucous membrane, and no mechanical impediment to the passage of food into the pharynx; but the velum did not contract freely, as it does in health, upon the application of a mechanical stimulant.

The case of our patient King differed remarkably from the others, in being complicated with a state of trismus; which condition, however, I think, admits of explanation on the supposition that the principal source of irritation was the throat. If you call to mind the presence of the extensive nervous plexus lying outside the tonsils and isthmus of the fauces, which is called the pharyngeal plexus, and consider how this is formed, you will not be at a loss to account for this symptom. The pharyngeal plexus, as you know, is made up of fibres from the vagus, glosso-pharyngeal and sympathetic nerves. Irritation of the ramifications of the two former nerves may be readily propagated to the medulla oblongata, so as to affect the motor portion of the fifth nerve, which is implanted there, and by which a convulsive state of the muscles of mastication may be excited and maintained.

This man, as I have said, recovered to a slight extent his power of swallowing, the day after his admission into the hospital, and was able to open his mouth slightly. We then discontinued the quinine injections, and gave him wine and nutritious food and quinine by the mouth. Under this plan, his power of deglutition was completely restored in two days; but there remained a catarrhal state of the mucous membrane of the trachea and large bronchial tubes, for which he still remains a patient in the hospital.

Now, that this affection of the pharyngeal membrane is of the erysipelatous kind, I think I am justified in affirming from the following considerations:—First, from the rapid invasion of the attack, and the great constitutional disturbance with which it is accompanied. Secondly, from the local redness; and, thirdly, from the great prostration with which the attack was rapidly followed, which was sufficient to kill two out of five cases. Our patient King, as well as all the others whose cases I related to you, seemed to succumb at once under the influence of some powerfully depressing poison, just as patients attacked with external erysipelas do. Then, it is well known, that cases of erysipelas of the head and face often commence with sore throat; or that a soreness and redness of the fauces are developed simultaneously with the appearance of the first patch on the face.

The marked difference in the treatment of the successful and of the fatal cases likewise favours the opinion, that the affection was erysipelatous in its nature. The two fatal cases were treated by a depletory and depressing plan; the three successful ones by a supporting plan, which consisted in, first, the careful avoidance of everything tending to produce fatigue, or exhaustion, or depression; secondly, in the frequent administration of large doses of quinine, with beef-tea in the shape of enemata, with or without brandy, and afterwards, when the power of deglutition returned, the exhibition of bark, ammonia, wine, &c., according to the circumstances of the case; thirdly, in the local application of the nitrate of silver to the throat.

The erysipelatous character of the affection is further indicated by its extension to the trachea and bronchial tubes, giving rise to the bronchitis under which the patient subsequently suffered, and from which he is now recovering, having been subjected to a similar course of treatment to that first adopted.

The other sore throats which bear the closest analogy and resemblance to this are that from influenza, that connected with diphtheritis, and also that of scarlet fever. The last is very readily distinguished by its ulceration and sloughing character; the diphtheritic throat is characterised especially by the plastic exudation which covers it, which again you will distinguish from the aphthous throat which occurs in phthisis, in carbuncle, and

after other low diseases, and which has a non-plastic exudatio in which the *oidium albicans* abounds. Influenza produces state of throat very difficult to distinguish from this which have been describing. I cannot say, however, that I have eve seen under influenza the dysphagia go to the same extent as i the erysipelatous throat. To the eye both states of throat ar alike; the mucous membrane of a dingy dusky red, and th fauces quite open. In the influenza throat the mucous mem brane has generally a more lax appearance, and the submucou glands of the velum are more prominent; the uvula is sometime quite œdematous. In the erysipelatous throat there is mor tendency to swelling in the tonsillitic region, but the swelling according to my experience, is never such as to create mechanical impediment to deglutition as in quinsy.

I shall conclude this lecture with the history of two othe cases, both interesting examples of the disease we have bee considering; one of them is especially deserving your attentio as from the case having terminated fatally, you can have befor you the whole clinical history of the disease.

Case LXII (vol. xxxviii, p. 77).—John Covey was admitte into King's College Hospital, November 2d, 1852. He was sixt years of age, and therefore not a good subject for acute diseas From the notes of the case we learn the following history:- His health had been pretty good until October 27th, when h was seized with shivering, which was followed by fever and loss o appetite; at the same time he experienced some difficulty i swallowing. The last symptom gradually increased up to th day of his admission.

I shall read you the description of his condition on Novemb 2d, immediately after his admission, as entered in the case-book —"The patient breathes with some difficulty, as if there were collection of mucus in the larynx and trachea. He suffers good deal of pain, increased by pressure beneath the angles the jaw, but not much in front over the anterior surface of th larynx. There is no enlargement of the glands of the nec apparent externally. His chief complaint is of difficulty swallowing: when he attempts to swallow anything, it seems t

go the wrong way, and appears as if it would suffocate him. He can swallow a little arrow-root, but even that with considerable difficulty. When the food or the fluid which he attempts to swallow gets to the back of the tongue, instead of being guided by the action of the faucial muscles into the pharynx, it seems to fall towards the glottis, and then excites a spasmodic state, producing a feeling of suffocation, and is forcibly ejected, partly through the mouth, partly through the nose. There is no actual impediment to the passage of the food into the pharynx: the tonsils are not at all enlarged, and the pharyngeal mucous membrane looks red but very slightly swollen, and there is a good deal of mucus upon it. When touched with the finger or spatula, the pharynx is not, as in health, thrown into action, apparently in consequence of paralysis of the pharyngeal muscles. The peculiar state of the mucous membrane extends to the larynx; for the epiglottis feels slightly swollen, and he spits up a good deal of mucus. He is very restless, and sleeps badly at night. Bowels confined: pulse 96: respirations 30." Urine acid, containing blood in considerable quantity: blood corpuscles in abundance were seen under the microscope, and numerous casts of kidney tubes, apparently consisting mainly of blood-cells, depending in all probability upon the rupture of one or more Malpighian capillaries.

You are aware that it is not uncommon to meet with slighter cases of erysipelatous inflammation of the fauces in connexion with erysipelas of the head and face; that affection, indeed, not unfrequently begins in the throat and thence spreads outwards through the nose and mouth. In this case, as in the whole class to which it belongs, so far as my experience goes, no tendency of the morbid process to spread outwards has been manifested. In Covey's case, unlike the others, there was some tendency to spread to the laryngeal and bronchial membranes. Many fatal cases of œdema of the glottis originate in this way by the extension of an erysipelatous inflammation to the laryngeal mucous membrane.

Our patient was promptly treated in the manner I have recommended in this lecture: the throat was freely washed with a solution of twenty grains of the nitrate of silver in an

ounce of distilled water; a mustard poultice was applied to the throat externally; he was ordered two drachms of brandy in arrow-root every three hours, and carbonate of ammonia with chloric æther was also freely given.

On the 3d, there was no improvement: prostration very great; pulse 90, and very compressible: he could scarcely swallow anything; the attempt to do so nearly suffocated him, and he had taken hardly any of the brandy and arrow-root. The mucous membrane of the throat was red, and secreting a quantity of muco-purulent fluid; the uvula was slightly swollen.

The solid nitrate of silver was now applied freely to the mucous membrane of the fauces, and he was ordered to have an enema, consisting of ten grains of quinine in three ounces of strong beef-tea, every three hours, the rectum having been first cleared out by an enema of warm water. He was allowed brandy, if he could swallow it.

The next day, November 4th, the report was satisfactory; the pulse was better; he had had the enemata regularly, and retained them all; he was able to swallow much better, and to take at least nine tenths of what was offered him.

On November 5th, he was still improving; the throat was less sore externally; the secretion much diminished; he was able to swallow all that was brought him; pulse 80. The brandy and the enemata had been administered regularly up to that time, but the latter were then discontinued, and he was ordered chloric æther, ammonia, and bark. From this date our patient rapidly recovered. On the 6th, his pulse had fallen to 70, and all difficulty of deglutition had disappeared; the urine assumed its natural condition; but the patient continued weak for a long time, and did not leave the hospital until the 27th.

Case LXIII* (vol. xxxiv, A. p. 50).—The last case I shall mention is that of M. A. Ayres, aged thirty-six, a poor married woman, who had been some time in ill health and subject to cough. She was confined with her third child on the 24th of September, 1851. There had been several cases of fever in her house; she was therefore not surprised when on the third day

* Recorded by Dr. Maurice Davis.

she was attacked with symptoms of fever—thirst, parched mouth, hot skin. The following day these symptoms increased; her thirst became excessive, and she found herself unable to swallow.

She obtained admission to the hospital on the 30th, and was then suffering from great dyspnœa and difficulty of swallowing. On examining her throat, however, no obstacle could be observed; the interval between the pillars of the fauces was not at all contracted, but the mucous membrane looked purple, and the pharyngeal muscles refused to respond to any stimulus applied to them. When food was taken, it seemed to fall upon the glottis, causing spasm and symptoms of choking.

She was ordered strong beef-tea with half an ounce of brandy every hour, besides carbonate of ammonia and chloric ether.

On the 31st her pulse and respirations were 120 and 40 respectively; they rose in the evening to 126 and 46, and the next morning they were 128 and 44.

On the 2d of September, the eighth day from her delivery, there was less difficulty in swallowing: the secretions of milk and the lochia continued. On the 3d and 4th her breathing and power of swallowing improved still further, and she was able to eat with comparative ease. The pulse and respirations continued very high—120 and 42, and the brandy was increased to an ounce every hour.

On the night of the 6th, she was seized, while swallowing some fluid, with a convulsive attack, and gasped for breath. The dyspnœa remained for three quarters of an hour afterwards, but was relieved by hot fomentations.

She continued, during the 8th, 9th, and 10th, in very much her former condition, with rapid pulse and breathing and a flushed face, complaining chiefly of her throat feeling parched; but she was able to swallow, breathe, and speak very fairly, and her cough was trifling. The brandy was reduced to six drachms every hour, and finally to rather less, with a little wine.

On the 11th, after being removed from her bed and washed, she was seized with rigors, never rallied, and died in about twenty minutes.

On making a post-mortem examination, the pharynx and

larynx appeared healthy, and the pharyngeal mucous membrane which during life had a purple hue, now looked anæmic. The upper parts of both lungs were congested, the rest anæmic. The lining membrane of the uterus was purple. All other parts exhibited a remarkably anæmic appearance.

LECTURE X.

ON CERTAIN ACUTE DISEASES.

ON THE TREATMENT OF ACUTE INTERNAL INFLAMMATIONS.*

Gentlemen,—We have lately been watching with great interest the patient Jane Cook, aged nineteen, who is still in the hospital, Case LXIV† (vol. lvii, p. 17). Her case has afforded us a good illustration of the phenomena of disease in its most acute form: she has had pericarditis in connexion with rheumatic fever, some degree of endocarditis, and pneumonia, with consolidation of about a fourth of the posterior part of each lung. I shall take this opportunity of making a few remarks upon the treatment of acute internal inflammations generally.

This patient is rapidly recovering, and, indeed, in an illness of unusual severity, she has had no serious drawback. On the 2d of July, rheumatic symptoms showed themselves in pain and swelling of the lower joints. On the 6th of July, a pericardial friction sound was heard over the base of the heart, which soon became distinctly audible over its whole anterior surface. On the 7th, bronchial breathing became audible at the posterior part of the lower third of the left lung; and on the 10th, the right lung was similarly affected, and to an equal extent. On the 12th, vesicular breathing began to be audible in both lungs, and the bronchial breathing to disappear.

Now this patient was treated in the manner in which (with but slight modification) I have been for some years in the habit of dealing with similar internal inflammations, especially those of the lungs and heart. Although my practice in such cases is now pretty well known, and I am proud to think is adopted by

* Delivered in July, 1857, and reprinted from Nos. I and II of the 'Archives of Medicine,' edited by Dr. Beale.

† Reported by my clinical clerk, Mr. Mason.

very many of my pupils in various parts of this city, and of th country, it may be useful if I take this opportunity of explainin to you the principles upon which it is based.

And first let me describe to you in detail, as a good instanc of this treatment, that to which this girl Cook has been sub jected.

On admission, while yet it was uncertain how far the rheu matic symptoms would extend, she was treated with alkalies an mild saline purgatives. Bicarbonate of potass, in doses of fro twenty to thirty grains, was given every four or six hours, an very soon opium was freely given, when the cardiac affectio manifested itself. As much as one grain of opium was give every fourth hour. Care was taken to keep the bowels open b giving an aperient draught daily of sulphate and carbonate magnesia. Counter-irritation was employed over the situatic of the inflamed lungs by means of stupes of flannel soaked i turpentine; these were applied twice or thrice a day, and th region of the heart was freely blistered.

A principal and very important part of the treatment which, as most of you know, I pay very special attention, is th which I may call the dietetic portion. The object of this is support the vital powers of the patient, and to promote gener nutrition, during the time when those changes are taking pla in the frame which tend to check or to alter the morbid proces and to convert it into a healing process.

When a patient suffers from pneumonia, the tendency is f the lung to become solid, then for pus to be generated, and last for the pus-infiltrated lung-structure to be broken dow and dissolved. Such are the changes when matters take an u favorable course. On the other hand, recovery takes plac either through the non-completion of the solidifying process, by the rapid removal, either through absorption or a process solution and discharge, of the new material, which had ma the lung solid.

It will scarcely be affirmed, even by the most ardent believ in the powers of the therapeutic art, that any of the measur which are ordinarily within our reach, such as the administr tion of certain drugs, or the abstraction of blood, or the applic tion of blisters, exercise a *direct* influence in effecting the

changes. Save in the case of antidotes, which directly antagonise the proximate cause of the morbid state, medicines promote the cure of acute disease by assisting and quickening some natural curative process. And he is the wisest practitioner, and will be the most successful therapeutist, who watches carefully the natural processes of cure; in other words, who studies the phenomena, both anatomical and physiological, which accompany them, and of which, indeed, they consist.

Let me, therefore, exhort you to look very carefully to this as a part of your clinical study. If you will be on the look-out, you may often meet with cases of acute disease, which recover with little or no medical treatment, and you may observe and note the clinical phenomena which they exhibit.

Allow me to anticipate your observation on this head, and to point out what you may look for in cases of pneumonia, and what you will certainly find in almost every instance.

First, the hot, often burning skin, which is so generally present in the first stages of pneumonia, will be exchanged for one bedewed with moisture, generally to the extent of free sweating.

Secondly, along with this sweating process, there will be one of increased flow of urine, and very often a free precipitate of brick-dust sediment, urate of soda, more or less deeply coloured.

Thirdly, not unfrequently, expectoration will become freer, the sputa more easily discharged, they will lose their characteristic reddish, rusty colour, and often become very profuse and even purulent. Now and then the purulent sputa are so abundant that it is difficult to imagine that they can have come from any other source than an abscess.

Fourthly, the chemical characters of the pneumonic sputa exhibit an interesting contrast with those of the urine. In the height of the inflammatory state, the sputa contain common salt (chloride of sodium) in abundance, and the urine is entirely devoid of it. As the inflammation becomes resolved, the salt returns to the urine and leaves the sputa.*

* On the "Diminution of the Chlorides in the Urine in cases of Pneumonia," by Lionel Beale, M.B. F.R.S. 'Med.-Chir. Trans.,' vol. xxxv, 1852.

Lastly, when all these changes are going on, the physiologic functions which have been disturbed by the local malady gradually approach their normal state. The quickened breath ing, the accelerated pulse, the unnatural generation of hea gradually subside. As all these admit of being measured b numbers, you should tabulate them in your records of cases, an you will find on each succeeding day (under such circumstance as I am now referring to) the figure assignable to each functio gradually become lower until you arrive at the normal.

Now is it not plain from all this that the process of resolutic of pneumonia is a distinct natural process effected by the variou physical agencies which are concerned in the nutrition of th lung? A material which clogs the air-cells and minute tub is removed, chemical changes of the most marked and obviou kind accompany the deposition and the removal of this materia and certain functions of excretion become strikingly augmente as if for the purpose of getting rid of some noxious matter ou of the circulation. A more exact and minute analytic chemistr than we have at present will at some future time, beyon doubt, detect more minute changes in the blood, and determin the exact nature of the discharged matters.

One other remark I must make in connexion with th subject. These acute internal inflammations are very often— suspect always—connected with the prominence of son peculiar diathesis—the gouty or the rheumatic, for instance- sometimes the scrofulous. Of these diatheses, the main chara teristic is the generation of some peculiar morbid matter whic when accumulated in quantity in this or that organ, gives ri to inflammation in it. And the determination of the morb matter to the lung or the pleura, to a joint or a muscle, w often depend on the direct influence of cold, or of an unwonte amount of exercise, or of some mechanical injury. The evil to be remedied by diminishing the intensity of the diathesi This is done naturally, and is to be imitated artificially, by th elimination of the morbid element through the channels augmented excretions, such as the sweat, the urine, and th secretions of the alimentary canal.

You will perceive, then, that my argument may be th summed up. Internal inflammations are cured, not by th

ingesta administered, nor by the egesta promoted by the drugs of the physician, but by a natural process as distinct and definite as that process itself of abnormal nutrition to which we give the name of inflammation. Our interference either may aid, promote, and even accelerate this natural tendency to get well; or it may very seriously impair and retard, and even altogether stop, that salutary process.

If, then, this view of the nature of the means by which inflammation is resolved in internal organs be correct, it is not unreasonable to assume that a very depressed state of vital power is unfavorable to the healing process; for how can an important vital process go on satisfactorily where there is a lack of vital power? Indeed, if you watch those cases in which nothing at all has been done, or in which nothing has been done to lower the vital powers, you will find that the mere inflammatory process itself, especially in an organ so important as the lung, depresses the strength of the patient each day more and more.

Look, indeed, at our patient Cook, whose case has led me to this subject! See how depressed she is by the mere force of the inflammatory affection of both lungs! Her pulse small, weak, compressible, and at 120; her heart's action feeble and rapid; her surface pale and exsangueous; and yet this girl was well upheld from the commencement; she took no remedy which has any depressing influence. What must have happened in her case had she been largely bled to twelve or sixteen ounces, and taken tartar emetic freely? My impression is, that she would not have had power to go through the healing process, or that her vital powers would have been so diminished that the healing process would have stopped. The hepatized lungs would have remained hepatized (as I have often seen where blood has been freely taken), and the pericardial effusion, which did undoubtedly take place, would have been so much more considerable in quantity, as to have materially interfered with the heart's action, and added another cause of depression of vital power to those already in existence.

You will perceive, then, that, according to these views, there are strong *à priori* reasons in favour of the policy of upholding our patients, even in the earliest stages of acute disease, by such

food as may be best suited to their digestive organs, such as is most readily assimilated, and calls for the least effort, the smallest expenditure of vital force, for its primary digestion. Nutritive matter in a state of solution—broths, soups, farinaceous matters—answers this purpose best, and also alcohol, which is directly absorbed without any previous change, and tends to feed the calorifacient process, and to diminish the waste of tissues, which would necessarily follow in order to maintain it.

Many people start with horror at the notion of giving alcohol in acute inflammatory disease. What! give brandy in inflammation of the lungs! it is only adding fuel to the fire, and cannot fail to keep up or to increase the morbid process.

Those who reason in this way take a narrow and, I must say, an incorrect view, both of the morbid process and of the healing process; they are led away by the name *inflammation*, which is likened in their imagination to an internal conflagration, to be quenched by some summary means, or to be starved out. Nothing is to be given but what is, in popular phrase, cooling; and blood, the great pabulum of animal heat, is especially to be diverted from the seat of inflammation, or to be abstracted in such ways as the peculiarities of the case will admit.

This reasoning is of the most purely fanciful kind. It rests upon a very imperfect view of the phenomena, both local and constitutional, which accompany the inflammatory process. In fact, it takes into account only two of the phenomena of this process, namely—the heat and the afflux of blood, leaving out of consideration both the exciting cause and the proximate cause of this heat and afflux of blood.

No doubt there is some analogy between an inflammation and a fire, and I might rest an argument in favour of my views upon the further prosecution of this metaphor; but I prefer to bring before you the real nature of the inflammatory process, and of the actual physiological changes which it involves.

Inflammation is a deranged nutrition. Like the normal nutrition, it involves supply and waste; and as the latter is considerable, the former will be proportionably needed. The tendency in inflammation is to the more or less rapid formation of abnormal products, such as lymph and pus; and the supplies for these formations must be drawn from the blood or from

the tissues, in both cases with the effect of more or less exhaustion of vital force, in the latter with more or less extensive organic disintegration. The active chemical process which accompanies all these changes engenders the great heat of the inflamed part.

The more this process of inflammation draws upon the blood, the greater will be the exhaustion of vital force, and the more the whole frame will suffer; the more it feeds on the tissues, the greater will be the difficulty of the reparative process. Is it not, then, important that adequate supplies should be conveyed to this process, abnormal though it be? And in what other way can the appropriate supplies be conveyed to it than through the blood, so that the waste of tissue may be stopped, and the tendency to abnormal formations be checked, at least from that direction?

And this, in truth, seems to me to be but the plain and simple fact;—you must feed inflammations as you would other active vital processes. You must, that is, feed them to prevent them from extending to, and preying on, healthy organic structures, and committing great destruction. Bear in mind, too, that you cannot stop an inflammation so long as the exciting cause of irritation is inherent in the inflamed part: you cannot cure an inflamed eye so long as the irritating particle of dust remains adherent to it. It is wise policy, then, to try and gain time, until by antidotal means, or by elimination, you can get rid of the local irritation, whatever that may be.

The physiological expression for what is commonly called *suppuration* is a more or less rapid waste of tissue or organic matter, and a conversion of the particles so wasted into what we designate *pus*. This conversion will, within certain limits, take place in greater quantity, and the more actively, the lower the vital power of the patient. Take two cases of erysipelas, involving the same parts, and in all respects alike, and place them in adjoining beds, feed one from the beginning of the symptoms, and give him stimulants, give the other milk and beef-tea; both patients will get well, but the first will have few or no secondary abscesses, the second will have them in greater or less number according as he may naturally have less or more power of vital resistance.

But to proceed to more practical points. Our patient, Jane Cook, exhibited an example of the acute inflammatory process, proceeding to a very high degree, and involving several important organs,— both lungs, the corresponding pleuræ, the pericardium, the endocardium. The tripod of life was assailed in this girl's case. It is, therefore, a highly valuable illustration of the extent to which you are likely to be called upon to proceed with the kind of treatment I have described. And on the other hand, I may remark, that being a young and healthy girl, not strumous, but clearly of rheumatic diathesis, she was as fair a subject as one is likely to meet with for the successful practice of the bleeding and lowering plan.

Yet what was our practice? Besides the drug treatment, which I have detailed at the commencement of this lecture, this girl was freely supplied with beef-tea, and she had half an ounce of brandy every hour. At first the quantity of stimulant was not so great, it did not, indeed, exceed half that amount; but very soon, when we saw the inflammation spreading, and the vital power evidently diminishing, the pulse showing a marked tendency to become rapid and weak, the patient suffering from profuse sweating, which within certain limits was salutary, we did not hesitate to increase the quantity of brandy largely. You have witnessed with what result. The pneumonia subsided quickly, so that on the fifth or sixth day the signs of hepatization had disappeared, and vesicular breathing returned in each lung. The pericarditis did not disappear so quickly. On the 16th of July, the patient was troubled with diarrhœa, notwithstanding which, on the 17th, all the signs of copious effusion into the pericardium were manifest; both the pulse and the breathing were greatly increased in frequency, and the patient suffered from orthopnœa. She was freely blistered over the heart; the brandy was increased to *six drachms* every hour; beef-tea was given frequently in small quantities. The opium was continued, and ammonia, with chloric ether, was also freely given.

During the spread of the pericardial inflammation the pulse rose from 104 to 120, and reached its highest point at 124. The breathing was excessively quick, but as the girl was of a highly nervous temperament, much of that rapidity was due to

her extreme nervousness, which became much augmented when she was under examination: she panted rather than breathed. But it was satisfactory to find in our daily examination of the lungs, that no cause for quick breathing existed in them at this period. It was due primarily to the cardiac disturbance, but was greatly aggravated by the hysterical state, which so often complicates and gives a peculiar complexion to the symptoms of more serious disease in women.

It was very remarkable, that, notwithstanding extensive pericarditis and some endocarditis, our patient never exhibited any marked delirium. This is uniformly the case in acute diseases, erysipelas, fever, pneumonia, rheumatic fever, in which alcohol is given, as has been done in this case. Delirium is kept off by it. This formidable complication of acute disease ceases to trouble either the patient or the physician, if the former be duly supported from the beginning. And if delirium comes on, notwithstanding that you have been giving stimulants, you will generally find it desirable to give them more freely.

This is a fact which I have so often verified, that I am enabled to enunciate it dogmatically, that alcohol carefully administered, from an early period, in small and often-repeated doses, is the best preventive of, and antidote to, delirium in acute disease.

Indeed, many of you who watch my practice know how rarely that symptom gives any trouble. It is altogether the merest trifle, as compared with what I used to find it when I adopted the so-called antiphlogistic treatment. And thus a great source of danger to life is avoided.

This fact, as regards the influence of alcohol in the *prevention* of delirium, is one of the most important which the clinical observation of cases, treated by stimulants, brings out. It is quite inexplicable by those who refuse to study the action and the mode of digestion of alcohol, and who, adhering to old prejudices, rest content with a practice under which, to say the least, great mortality occurs, rather than be at the trouble of carefully investigating the powers of an important remedial agent.

Another interesting point in this case deserves your attention. While our patient was getting well of the double pneumonia,

pericarditis having already come on, a severe diarrhœa supervened, which depressed her very much. Did this contribute to relieve the pericarditis, as one might expect according to the ordinary antiphlogistic notions? On the contrary: immediately upon the attack of diarrhœa there ensued signs of pericardial effusion; the dyspnœa became much aggravated, and extended dulness on percussion was found to exist in a very marked manner over the cardiac region; the sounds of the heart became distant and muffled. Under the continued use of stimulants, for a short time in still larger quantity (an ounce per hour), with opium given more frequently, and free blistering of the cardiac region, these symptoms quickly subsided.

In most cases, treated as our patient Cook has been, we have found that the pulse diminishes in frequency steadily from day to day in a very remarkable way. This was not the case with Jane Cook. At first the pulse showed a disposition to fall, and it came down from 120 to 116, and remained at this point for two days; but on the occurrence of the pericarditis it rose again, but never exceeded 124. Notwithstanding the pericardial effusion, it remained at this point, and afterwards fell to 120. I believe that the fall of the pulse was opposed chiefly by the highly hysterical temperament of the patient, but partly by the cardiac inflammation. The wonder was that, with the extensive inflammation and the extreme debility, the pulse did not rise more—even to 140, or higher; this, on the other hand, was obviated by the presence of alcohol, which, when fully digested and acting favorably, tends to prevent the pulse from increasing in frequency, if it does not reduce it.

Lastly, our patient had a rapid convalescence. When once the diarrhœa was stopped, the pericardial effusion became quickly absorbed. The signs of effusion were at their highest point on the 17th of July; by the 20th they had disappeared; on the 25th the patient was fairly convalescent;—just twenty-three days from her admission. From the 25th to the 2d of August, her recovery of strength and colour was rapid, and she might have left the hospital at this time; but, as a matter of safety, she was detained until the 15th of August, when she left quite well.

Rapidity of convalescence is not the least important feature

of the cases treated by this upholding plan. When once the acute mischief is subdued, it is surprising with what rapidity the patient emerges from the invalid condition. Of this we have numerous examples in all forms of acute disease, and in none more than in pneumonia, erysipelas, and continued fever.

In conclusion, let me impress upon you, that in supporting your patients (whether in acute or in chronic disease) you should be especially careful to avoid throwing too much work on the digestive organs at any one time. Your supplies should be always administered in small quantities, more or less frequently repeated; never in a large amount at once. They should be well timed, and the exact doses defined. When alcohol is being administered largely, animal food is best given in solution, as in broths or soups. The ability of the patient to take solid animal food may be regarded as the signal for diminishing the supplies of alcohol. Experience has taught me not to give two kinds of alcoholic fluid at the same time; do not give beer and wine, or wine and brandy; any one of them will agree better, because it will be more easily digested when alone.

Patients often flush a good deal upon the first use of stimulants; this alarms the practitioner and deters him from prosecuting their further administration, or leads him to a vacillating practice, generally most injurious to the patient. It is a mere prejudice to suppose that any harm arises from this flushing of the face; generally it is an indication that the process of digestion, either of wine or spirit, or of other food,* is carried on with difficulty; and it will commonly cease by modifying the manner of its administration, such as giving less at a time, and more frequently. Sometimes, indeed, flushing will occur because an insufficient quantity is given; and an increase of the dose will get rid of it, just as an inadequate dose of opium disturbs the nervous system, whilst a larger one calms it.

In a word, I cannot too strongly impress upon you that, to do good with stimulants, you must use them early, with care and

* The popular notion that alcoholic stimulants are not food, but a mere "flash in the pan," ought not to be encouraged by medical men in the present day.

watchfulness, in very definite quantities, and not in a vacillating or timid manner. They are agents of inestimable value for saving life under all forms of acute disease, and I can say with truth, from a large experience, that the harm which they do (*in disease*) is grossly and unfairly exaggerated, and always due to the slovenly administration of them. The opponents of their use argue from their outrageous abuse in health, against their careful and scientific use in disease, forgetting how essentially different must be the effect of sixteen or twenty ounces of wine swallowed down within an hour or two, along with other food, and the same quantity carefully distributed in half-ounce and ounce doses over a period of twenty-four hours. I say it after mature reflection and a long course of observation, that there is no point of therapeutics so deserving of the study of the earnest-minded physician or surgeon, who is zealous to save life, as that of the action of these agents, both in health and disease.

LECTURE XI.

ON PYÆMIA.

Gentlemen,—The case upon which I propose to found my remarks to-day is one which has afforded us great interest in our hospital visits for a considerable time; it is a good example of that remarkable form of disease which of late years has been called pyæmia—an affection remarkable in a pathological as well as in a physiological point of view.

Case LXV (vol. xxxvi, p. 21).—The case to which I refer is that of a young man in Fisk ward, of the name of Gordon, who entered the hospital on the 27th of March, 1852. He is only twenty-three years of age, and of a strumous habit. Suppurative tendencies appear to have existed in members of his family; his father died from an abscess in the thigh, and his brother, some time ago, suffered from suppuration of the lymphatic glands on both sides of the neck; his mother died of phthisis.

The beginning of our patient's misfortunes was a gonorrhœa, which was followed, soon after, by a swelling in the perinæum; this increased and became exceedingly painful; an abscess at last formed, which was opened in a metropolitan hospital, and about four ounces of pus escaped. The wound, however, did not close, but continued to discharge for a month afterwards. The gonorrhœa, which, up to this time, had not been attended to, was now treated and soon relieved; the abscess also closed, and appeared to be quite cured; the subsequent history, however, of the case renders it exceedingly probable that the suppurative process did not entirely cease, although the external wound healed perfectly.

On the 21st of March he went down to Deptford, and indulged in beer and spirits more freely than he was wont to do.

This intemperance was followed by fresh inflammation in the site of the abscess, with severe pain and swelling, and a sense of constriction around the anus. These symptoms were accompanied by fever, prostration, sweating, and pain in the joints, so severe as to prevent the patient from moving his limbs; the region of the abscess swelled considerably, and the pain and tension of the soft parts became very great. At this time he was seen by a medical man, who considered that he was labouring under an attack of rheumatic fever. In a day or two he was removed to the hospital, and his condition was as follows:—He had a rapid pulse and was sweating profusely; the tongue was foul, and there was an aphthous condition of the throat. The patient was very weak, and fast sinking into a low typhoid state; an abscess had formed in the perinæum, and his strength was rapidly failing. Such a train of symptoms indicated the necessity of support, and we therefore at once put him upon a liberal system, and supplied him with nutritious food and stimulants.

Although the constitutional derangement resembled very much that of rheumatic fever, we were not long in determining that the patient was not suffering from that affection. The history of the access of his illness was not that of rheumatic fever, neither was there any indication of the presence of the rheumatic diathesis. The existence of the gonorrhœa and the previous inflammation, although they did not negative the idea of the rheumatic nature of the complaint, at least pointed to another more probable explanation of his symptoms.

On April the 6th, symptoms of pleuro-pneumonia manifested themselves: a friction sound was heard at the base of the left lung behind; in fact, we had here an excellent example of a dry rubbing sound, audible both upon inspiration and expiration; at the same time we found crepitation at the base of the right lung. The nature of the case became cleared up by what took place on April the 10th, when a symptom manifested itself which could leave but little doubt of the true origin of the other symptoms from which our patient suffered. At the outer border of the right ulna we found a collection of fluid in the areolar tissue,—in fact, there was an abscess of considerable size in that situation. Upon further examination, we dis-

covered two similar collections of fluid, in the subcutaneous areolar tissue, over the lower angles of both scapulæ. All these abscesses were immediately opened, and a free discharge took place from the wounds.

On the 13th, the lung symptoms increased in severity: the respirations had risen to 30, and the friction sound at the base of the left lung was very distinct; crepitation was still audible at the base of that lung,—it had become soft and loose in character, and almost amounted to gurgling; the breathing in the same situation was tubular, and the percussion dull. The other symptoms continued much as before: the hectic fever did not abate, there was still profuse sweating, and the pulse remained quick. At the same time the patient's strength appeared to be gradually improving, which favorable alteration was no doubt due to the brandy and nutritious broths, which were administered as freely as his stomach would bear them.

Towards the end of April, free expectoration commenced, consisting chiefly of a considerable quantity of pus. On May the 12th, an abscess formed over the left hip, which was opened, and still continues to discharge. On the 5th of June (I pass over the daily reports of the case, thinking it better to mention the most important circumstances alone), a swelling appeared in the left buttock—an indication, no doubt, of the formation of another abscess, either in the glutous muscle itself, or in the cellular tissue between two of the muscles. The occurrence of this swelling was accompanied by increased fever and debility. Although the abscess on the hip continued to discharge freely, the lung symptoms had much improved: the breathing had diminished in frequency, the crepitation was less, and the friction sound had almost disappeared.

The question now remains to be answered, whether further abscesses will yet form, and whether the patient's strength will be sufficient to carry him on towards convalescence. Our best chance of success will be by supporting his vital power with plenty of nourishment, in which I include alcoholic food.

Now, what is the rationale of this case—this febrile condition and this low typhoid state, accompanied by the formation of abscesses in different parts of the body? It belongs to a class

of cases which, though originating under various circumstances and differing in the severity of their general symptoms, in the extent and situation of the local phenomena which usually characterise them, and in their duration and ultimate termination, are nevertheless alike in their essential nature. This consists in a poisoning of the blood by an admixture with it of purulent matter—either pus entire, and in its purest form, taken up directly into the circulation by an open vessel, or generated in some part of the vascular system; or unhealthy pus—decomposing, acrid, septic, received in a similar manner; or some of the amorphous elements of such pus, received by re-absorption into the vessels; or some other animal matter, allied perhaps to pus, the product of disease during life, or of decomposition after death; or, lastly, a peculiar animal poison derived from the recently dead human body, or that of some other animal.

All these contaminations of the blood seem capable of giving rise to nearly the same train of general and local phenomena similar to those from which our patient Gordon has been suffering. In the most severe cases, the disease begins with rigors, which are followed by fever of a low type, with utter prostration of strength, and the very rapid production, in quick succession, of circumscribed purulent formations, or *deposits*, in connexion with various capillary systems, followed by increasing exhaustion, sweats, collapse, and death. In less severe cases such as that of our patient Gordon, the course is slower, but the symptoms are similar: the fever is of a typhoid character, while the local suppurative processes tend continually to increase the exhaustion. To all such cases the name pyæmia—literally *pus-blood*, more freely *pus-contaminated blood*—is applicable.

It does not appear necessary, however, for the production of many of these symptoms, that the matter introduced should be pus, or even very closely allied to it: it need not be organized or even organic; inorganic substances, introduced into the blood, have produced phenomena very similar to those of the lesser grades of pyæmia, as has been proved by a variety of experiments.

The severity of a case of pyæmia depends, perhaps, in part

on the amount of morbid matter taken up, but probably much more on its quality: organic matter from dead bodies originates some of the worst cases of this kind; hence, as dissectors, you have a direct interest in this disease. The severity is also partly dependent on the previous general condition, or diathesis, of the patient: otherwise it would be difficult to assign a reason why, in one person, the reception of pus into a vessel gives rise to the formation of a permanently limiting coagulum, while in another this coagulum is not formed, or rapidly breaks down into a pus-like fluid, and diffuse purulent infection results.

Among the most important experiments made on the lower animals, bearing upon the pathology of this disease, are those of Gaspard and Cruveilhier, who injected various substances into the blood of dogs, such as different kinds of animal matter in a putrescent state, pus, insoluble powders, &c.: these were introduced into a vein, and being carried into the circulation, created obstructions in various parts of the capillary system. One of these experiments of Cruveilhier is well known: it consisted in injecting ink into the femoral vein of a dog. The limbs soon became œdematous, indicating some obstruction to the venous circulation, the obstruction no doubt being caused by the coagulation of the blood in the large vein, in consequence of which the capillary system, which supplied this vein, became obstructed, and the watery part of the blood transuded through the coats of the capillaries into the cellular tissue, producing the œdematous condition. At the same time, the areolar tissue and muscles became the seats of little extravasations of blood, or, as they were described by Cruveilhier, little apoplectic clots; and these extravasations became the foci of purulent formations. A similar course of events has occurred in our patient, as in the subject of Cruveilhier's experiment.

Since the experimental introduction of pus, and other foreign materials, into the blood gives rise to a peculiar train of symptoms, nothing can be more reasonable than to assign a similar train of phenomena, in cases where there is a manifest source of pus, from which the blood might be contaminated, to purulent infection; and even in other cases, where there is no *obvious* source of pus, and no other mode of explaining the

symptoms, we may attribute them to a like contamination of the blood by morbid matter, received from some hidden local source, or generated within the vascular system.

Let us consider, rather more fully, the different sources of infection in cases of pyæmia, and the nature of the secondary pus-formations.

That form of phlebitis, generally the result of some injury to a vein, accidental or surgical, in which there is a tendency to suppurative, rather than adhesive inflammation of the vessels is the most direct and the most frequent cause of pyæmia. This was ably demonstrated more than twenty years ago by Mr Arnott, a former Professor of Surgery in King's College, in an essay on the secondary effects of inflammation of the veins which I strongly recommend you to peruse. You will find it in the fifteenth volume of the 'Medico-Chirurgical Transactions.' The recent experiments of one of the surgeons at this hospital, Mr. Henry Lee, and others, have shown that pus and other foreign matters, introduced into the blood, often induce coagulation of that fluid around them, leading to the obstruction of the vessels, and consequent limitation of the purulent infection to a very small portion of the vascular system, and its effects to the immediate locality. It is thus, in fact, that nature operates most effectually for the prevention of a diffuse contamination of the blood, and reduces the severer cases of pyæmia to comparatively few, notwithstanding the frequent co-existence of injured or open veins, and suppuration in or about them, in surgical cases. Should the state of the blood, however, be such that its coagulation by the pus is very imperfect, or should the coagulum, as sometimes happens, pass rapidly into a state of fusion, breaking down into a fluid having many of the characters of pus, there is no longer anything to prevent the free circulation of the morbid material, and its rapid admixture with the blood mass.

There are certain surgical cases which seem more particularly prone to give rise to purulent infection. These are injuries and operations on bone, and the collection of pus pent up, and, it may be, decomposing, in sinuses and abscesses, especially in localities where veins abound.

In the former cases there is danger of pyæmia apparently for

anatomical reasons. The veins in bones are patulous, their mouths being kept open by the adhesion of the walls of the vessels to their bony channels; moreover, it appears from the experiments of Cruveilhier, who injected the cancellous tissue of bone with mercury, that fluids introduced into the cancelli find a ready passage into the osseous vascular system; hence pus and other matters may gain admission to the circulation by the same route. In the latter cases, the particular diathesis of the patient will often favour purulent infection by interfering with the natural occlusion of the vessels, and consequent limitation of the morbid agent, as described by Mr. Lee. To this class the case of Gordon belongs. The abscess in the perinæum doubtless furnished the pus. In that situation there are numerous veins communicating both with the system of the portal vein and also, through branches of the internal iliac, with the inferior cava and general circulation. It is doubtless owing to this disposition of the venous system that we so often find pyæmia following the perinæal section of stricture, and that abscess in the perinæum is so often the source of purulent infection.

But perhaps the puerperal state is, above all others, that most favorable to the production of pyæmia, or an allied disease. After the womb has expelled its contents, there remain on its surface many open mouths of large veins, only plugged by coagula, aud most favorably situated for the absorption of any morbid matter which may be present, pus, decomposing discharges, or putrescent remains of the ovum. Or the mouths of the veins themselves may become the seat of inflammation and the formation of pus, which, under certain circumstances, may find a ready entrance into the circulation.

Cases of purulent infection sometimes originate from erysipelas, when the inflammation has terminated in suppuration, and collections of pus have formed in the skin and areolar tissue.

Lastly, one of the worst forms of pyæmia is apt to arise in cases of typhoid fever, from the absorption, I believe, of some of the products of the sloughing and ulceration going on in the bowels. A case of fever may be doing apparently very well,

when suddenly the typhoid symptoms become greatly aggravated, and the patient rapidly sinks.

Lastly, you must class with these cases the formidable disease which follows wounds received in dissection, which formerly used to prove so fatal, and was the cause of the loss to science of many men of high promise, who were cut off by it at an early period of life. Wounds poisoned by diseased fluids, received from the living, are likely to produce the same train of symptoms, and to lead in the same way to a fatal result. (Case LXVIII.) I need not say how much you are each and all of you personally interested in the disease as originating in such a source.

The mode of formation of secondary pus-formations, or *purulent deposits*, or *metastases*, as they have been called, has given rise to considerable differences of opinion among pathologists. These pus-formations are found in all parts of the body, and the development of them in great numbers, with sometimes extraordinary rapidity, in various parts successively forms one of the most remarkable features of the higher grades of pyæmia.

First, they are found in the parenchymatous organs, especially the lungs and liver, where they appear to commence, in the first instance, as red capillary obstructions, with solidification of the textures around, and followed immediately by sloughing or suppurative fusion of the whole patch involved; so that a collection of pus is the result, with ordinary inflammation immediately around it.

Secondly, effusions of pus may take place on the surface of serous membranes, as the peritoneum; but they are far more frequently met with in the synovial sacs, and they there lead to the corrosive disorganization of the joints. Sometimes we find a rapid development of pus on the surface of mucous membranes, the bronchial for example, giving rise, in that case, to a copious purulent expectoration.

Lastly, and very commonly, the muscles and areolar tissue are the seat of secondary abscesses.

What determines the particular situation in which these pus-formations are found, in any individual case, it is not easy to conceive; but generally it seems that the first capillary system

through which the infected blood circulates becomes the seat of the formation of abscesses. In most instances, the first system is that of the lungs, and here we shall generally find evidence of capillary obstructions. This has been found, both in the experiments intentionally performed on animals, and in those which we unfortunately have many opportunities of seeing performed by disease on the human subject. In other cases, where the portal venous system is primarily infected, the secondary abscesses form first in the liver.

Some pathologists have considered these pus-formations to originate in the mechanical obstruction of the capillaries by aggregated pus-cells, or by small masses of coagulated fibrin, called *emboli*, too large to traverse them, and the subsequent mere deposition of pus there, or its multiplication from the blood elements. But this view, so purely mechanical, does not, to my mind, fully account for the rapid breaking down of the textures around the obstruction by an active inflammatory process. It is not necessary that pus should be introduced into the blood to give rise to these pus-formations: other fluids injected into the blood, as shown by the experiments of M. Gaspard, have produced them. And, therefore, it seems to me, we must look for some process of *contamination* of the tissues surrounding the obstructed point, to account for their destruction with inflammatory phenomena, and to explain the attendant constitutional disturbance. The obstruction by *embolus*, or in any other way, explains the localization; the depravation of nutrition by some material of the nature of a poison is needed to explain the inflammatory process in the surrounding texture, unless, indeed, that process be considered as the necessary result of the death of a certain portion of tissue from the sudden or rapid stoppage of its blood supply.

After all, it seems to me quite as difficult to account for the very rapid formation of the little circumscribed slough, which constitutes a common boil, as these purulent deposits.

When we consider, that although the morbid matter introduced into the blood has failed to cause coagulation in the vessel through which it gained admission to the circulation, it may, nevertheless, succeed in obstructing the capillaries in that way, and becoming fixed there, it may act as a local irritant;

and when we consider also the terrible effect produced on the whole system by the contaminated blood, as though by some deadly poison, depressing the vital powers of the patient to such an extent as sometimes to cause death, independently of, and prior to, any secondary local effects, it need not be a matter of much surprise, I think, that the local irritation should determine a very asthenic inflammation passing rapidly into sloughing and the formation of pus.

I have one or two more remarks to make on Gordon's case. You will recollect, that on April the 13th we had dulness over a portion of the chest: in the situation of the dulness we heard coarse crepitation, and, after a time, crepitation almost amounting to gurgling. The matters expectorated contained a large quantity of pus, and I thought that purulent infiltration had taken place, and that the formation of a cavity was imminent; indeed, there can be no doubt that a rapid formation of pus did take place, as in pneumonia, and that it was as quickly eliminated by expectoration, but without destruction of lung-substance. We had other indications of the occurrence of inflammation within the cavity of the chest, in the presence of a rubbing sound on the right, and also on the left side, showing that there was a roughened or, at least, a dry state of the pleural membrane in these situations. Here, then, we had both the systemic and the pulmonary capillaries affected with inflammations, which afterwards became the seat of collections of pus. The case exhibits, in a most striking manner, phenomena precisely similar to those which are met with in the subjects of experiments, when pus is injected into the veins of living animals.

For practical purposes, we may, I think, divide these cases of purulent infection into three classes:

The first class of cases is distinguished by this common character, namely—that the course of the disease is very rapid and the result is certainly fatal. As an illustration of this class of cases, I may adduce the experiment of Gaspard, which consisted in injecting into the jugular vein of a living dog three drachms of healthy pus. In three minutes afterwards the dog was seized with copious vomiting and micturition. For a quarter of an hour the limbs were violently convulsed, and

complete emprosthotonos frequently occurred. The subject of the experiment died five hours after the pus had been injected into his vein, in a state of extreme exhaustion. Now, in such a case, the fatal result can hardly be explained by the supposition that a great number of local obstructions have taken place, for there can hardly be time for such an occurrence; and it seems more reasonable to attribute it to a poisonous influence acting on the nervous system in a manner somewhat similar to that by which we explain death from prussic acid and other violently poisonous substances. In such cases, it appears that death results from a general contamination of the whole system by the noxious matter, rather than from the occurrence of local mischief.

In practice we have such cases—cases in which death results in two or three days, or even less, from the first circulation of pus in the blood. Let me give you an instance:—

Case LXVI (vol. ii, p. 135).—A young woman, named Mary Riley, twenty-one years of age, was admitted into the hospital under my care, January 25th, 1841, in consequence of a black gangrenous appearance and ulceration of the lips, especially the lower one; there was also much swelling and redness about the left eye.

She stated that the soreness of the lip commenced about a week before, with a small pimple, which gradually increased, and was followed by great swelling, and a black-looking ulcer at the left angle of the mouth. She had been, she said, in good health previously.

The day after her admission, the pulse became exceedingly rapid and feeble; she complained of great thirst and much pain in the lip. Rapid collapse ensued; there was no time for treatment, and she died the following night.

On examining the body after death, I found the cellular tissue of the lip much infiltrated with pus; much hardness and swelling about the angle of the jaw on the left side, involving the salivary glands; incipient inflammation of the left jugular vein—the blood imperfectly coagulated and adhering to the lining membrane, the coagulum of a dirty brown colour; pus in the smaller veins. The uterus and ovaries were highly vas-

cular, the latter much enlarged; there were several very large Graafian vesicles, with red inner membranes, one containing a small vesicle (germinal?) surrounded by granular matter.

These fatal cases are unfortunately too frequently met with in the puerperal state, and often come on very insidiously. A woman goes through her confinement in the most satisfactory manner, and all appears to be going on well, when formidable symptoms of rapid prostration manifest themselves, and the patient dies in one or two days. In such a case the pus enters the circulation probably by the medium of the uterine veins, and fatal exhaustion, frequently accompanied by convulsions, is soon induced, as in the experiment of Gaspard. Lately I saw a lady whose confinement, I was told, had been perfectly natural, and a month had passed over in a satisfactory manner; in fact, she had been out for a short time, and everything seemed progressing favorably; but on the evening of the day after she went out, she was seized with a severe rigor, which was followed by fever and rapid prostration, and in eight-and-forty hours she was dead. I was present at the post-mortem: we could find no evidence of peritonitis, nor any signs of active inflammation in the cavity of the uterus, but there was evidence of phlebitis affecting the large uterine sinuses.

There are other cases of this kind, closely allied to the last, in which a rapidly fatal result occurs: these are cases of puerperal fever.* All fevers, I hold, are due to an alteration of the blood, induced by the introduction into that fluid of some morbid matter. It is not often that we have an opportunity, in this hospital, of observing puerperal cases from their commencement. The poor woman, the subject of the following case, was confined in the hospital, and the notes taken comprise the whole history.

Case LXVII (vol. xxi, p. 30).—Hannah Donovan, a married

* Dr. Robert Ferguson, formerly Professor of Midwifery in King's College, was the first, or among the first, to put forward, distinctly, what is now generally regarded as the true pathology of puerperal fever—namely, blood contamination from vitiated uterine secretions. See his work on Diseases of Females, published in 1839. I have also discussed this subject in my Croonian Lectures on Gout and Rheumatic Fever, delivered at the College of Physicians in 1843.

woman, nineteen years of age, obtained admission to the hospital in consideration of her having a tumour in the abdomen, which we soon discovered to be a gravid uterus, and shortly afterwards our diagnosis was confirmed, by her being delivered of a healthy child, July 18th, 1847.

Everything went on satisfactorily until the 26th, when she was attacked with symptoms of fever; great thirst and languor, a dry and brown tongue, and a rapid and feeble pulse. At the same time the secretion of milk diminished, and there was considerable abdominal tenderness, while her whole appearance was remarked as characteristic of the terrible malady from which she was suffering, namely—puerperal fever. Turpentine stupes were applied over the abdomen. By the 30th, though there was less abdominal tenderness, she had become much worse: her face wore an expression of great anxiety; there was great heat of skin, a parched mouth covered with sordes, and a furred tongue; she was only partially conscious, and sometimes delirious; pulse 129, and very feeble, respirations 30; a mucous rattle was heard in her throat. Some eight ounces of wine were given in the day, and carbonate of ammonia; but she continued to get worse, and died on the night of the 31st. The following is the record of the post-mortem:—

"The uterus was found as large as a man's open hand, flattened on the left side, from the pressure of an enlarged ovary. On section, pus was found to exude from the veins; the walls appeared semi-cartilaginous, and, at the upper and back part of the cavity, a large, irregular, suppurating surface appeared, about the size of a crown-piece; another similar but smaller one surrounded the opening of the left Fallopian tube, and several, yet smaller, were found on the inner surface of the enlarged ovary. The peritoneum covering all the pelvic viscera was much congested." "The peritoneal vessels were much congested throughout, presenting appearances of incipient inflammation. The kidneys rather large and congested; the uterus much dilated—half an inch in diameter—but equally through their whole length, and presenting no pouch." There was some evidence of pleurisy on both sides. A large fibrinous clot occupied the right ventricle.

I shall here introduce another example of pyæmia which proved quickly fatal. In this case, pus was found in some of the veins, but its source was by no means obvious. The lungs were in a state of patchy hepatization; these patches may have been the earlier stages of secondary pus formations.

Case LXVIII (vol. xiii, p. 160).—Elizabeth Miller, aged forty-two, had been assisting one of the nurses in the surgical wards, and was attacked, without any assignable cause, with shivering, headache, utter distaste for food, and vomiting. These symptoms were followed, a day or two after, May 15th, 1844, by great pains in the joints and bones of the face, with entire prostration of strength, a rapid pulse, 112, and quick oppressed breathing, 34. Both wrists then became very red and tender, and there was a slight blush on the left leg. On the 17th, the left leg and several joints remained acutely painful; sleeplessness, and the same general symptoms, continued. By the 19th she was much weaker, and, despite an ounce of brandy which was then ordered every hour, the prostration increased, the pulse became more rapid, 132, the surface bedewed with perspiration, while her body remained everywhere warm; and though she rallied temporarily under the stimulants, she rapidly sank again, and died the same night.

At the post-mortem examination of the case, the appearances found were:—Some comparatively recent pleural adhesions, but no marks of recent inflammation. When the lungs, which were somewhat congested, were cut into, several small circumscribed patches in a state of red hepatization were found. There were two inactive hydatid cysts in the liver. When the right renal vein was cut into, a quantity of pus, tinged with blood, made its escape.

Sometimes we have opportunities of observing cases in which the introduction of pus into the circulation is as certain and direct as when injected into the veins in experiments. In a former lecture (page 115) I described two cases of this kind, in one of which the source of purulent infection proved to be an abscess in the septum of the heart; in the other, an ulcer at the base of one of the mitral valves.

Another class of cases, in which the result is often rapidly

fatal, comprises those which arise in the course of erysipelas, particularly idiopathic erysipelas of the head and face, which more especially concerns us as physicians. A case may be going on in a manner apparently quite satisfactory; the pulse may go down, the tongue become clean, and the prospect of recovery may be looked upon as certain, when suddenly rapid prostration comes on, the patient becomes comatose, and often within twenty-four hours is dead. Now, in such a case, it is difficult to prove that the fatal result depends upon the introduction of pus into the blood; but we know that in erysipelas there exists a great tendency to the formation of pus, and when we consider the great similarity of such a case to those in which we have decided evidence of the introduction of pus into the circulation, it seems not unreasonable to refer the fatal result to the same cause.

Let me give you an instance of pyæmia following traumatic erysipelas of the head; the superficial closure of the wound probably preventing the free escape of matter. The case illustrates some other points in the pathology of pyæmia: there were numerous pus-formations in the lungs, with the circumscribing inflammatory areola well developed. Profuse expectoration of pus was the chief feature of the case, which, I have little doubt, was derived chiefly from the bronchial mucous surface.

Case LXIX (vol. xvii, p. 37).—Patrick Shannon, an Irish labourer, was admitted into the accident ward of King's College Hospital, with symptoms of concussion and a scalp wound, the consequence of a severe blow on the head. He recovered from the immediate effects of the blow, but erysipelas attacked the wound, and spread to the head and face. From this he seems to have recovered only partially: the scalp wound healed deceptively, and on the surface only, and he then began to cough and expectorate a large amount of purulent matter, for which he was removed to the medical wards, and placed under my care, May 2d, 1846. He was then much emaciated, and his breath and whole body exhaled a sour and sickly odour of pus. The pulse was 136; the respiration short and quick—about 36; he

had a constant hacking cough, with expectoration of true pus. On percussing the chest, it was found generally somewhat dull. There was one patch of much more marked dulness behind, about the angle of the right scapula; and at the same spot crepitation was heard of a mixed character, with gurgling on coughing. Elsewhere, varying crepitation and rhonchus, with breathing, sometimes puerile, sometimes tubular, were heard. Carbonate of ammonia and brandy were ordered.

On the next day, the 3d, the same symptoms continued in an aggravated form: the same copious expectoration of pure pus; precisely similar, but more developed chest sounds; the same rapid pulse and breathing, numbering 132 and 42 respectively; some delirium. On the 4th, the symptoms were nearly the same, but of a still more typhoid type; there was great heat of surface, and some reddish discoloration of the integuments about the chest had taken place—a not unfrequent occurrence in these cases. The pulse and breathing were 160 and 52 respectively. The chest signs were similar to those on the 3d, but more developed. I shall not trouble you with the long detail of them. The bowels showed some tendency to become relaxed, which was greater the following day, and several watery evacuations were passed. On the 6th, there was increased delirium; the same copious expectoration of pus continued, and his breath was very offensive; the other symptoms were much the same as before. The brandy was now increased to three drachms every hour. On the 7th, he was much worse—more exhausted and feeble; and he died that evening.

The error in the treatment here consisted in putting off the free administration of brandy and nutriment until the eleventh hour; though it is, I think, highly improbable that this patient would have recovered under any plan of treatment, for the disease was of a high grade, and the local disorganization which it gave rise to was very great, as the following results of the post-mortem examination show:—

On opening the chest, the lungs *appeared* natural, though perhaps slightly emphysematous; but on cutting into them, numberless small cavities were found in clusters, filled with pus, and surrounded by lung tissue in a state of red hepatization; the bronchial mucous membrane, towards the base of each lung,

was red, soft, and velvety; there were some pleuritic adhesions. The brain and the other viscera were healthy.

Another set of cases of this kind are those in which this train of symptoms follows severe injuries or operations, more especially operations on the bones, as I have before mentioned.

A very unfavorable termination not unfrequently occurs after the operation of lithotomy, and after division of old strictures, where the case has been progressing favorably up to a certain period (the fever becoming less and the wound looking healthy), when fatal prostration comes on; and it can hardly be doubted that the sudden change is due to the introduction of pus into the circulation—the symptoms agree so closely with those of cases of pyæmia.

The same train of symptoms is sometimes brought about by the absorption of pus from Peyer's glands in a suppurating state.*

In a second class I would include those cases in which the result is no less fatal, but in which the course of the malady is extended over a much longer period of time. Many of these cases are also puerperal, and have been described by some French authors as rheumatic in their origin. These cases commence with tenderness over the region of the uterus, followed by thirst, heat of skin, fever, and swelling of one or more of the joints. The sterno-clavicular joint is that which is, perhaps, most frequently first affected, then the shoulder, and soon afterwards a knee-joint becomes swollen and tender. When you find the sterno-clavicular, or any other joint very full of fluid in a puerperal woman, you must not at once set it down as dependent upon a rheumatic condition, but must anxiously watch the case, and hesitate to pronounce a favorable prognosis, lest it should be followed by the formidable symptoms I have described.

You may sometimes see every joint in a patient's body thus filled with purulent fluid. The pus circulating in the blood probably creates obstructions in the capillaries of the joints, which afterwards become foci of pus-formations, as we have seen in

* For the further illustration of this form of pyæmia, see the case of John Gavin, Case XVI, p. 99 in the present volume; and the remarks upon it.

other cases. The strength of the patient soon fails, and, in spite of your best-directed efforts, you are unable to prevent a fatal result. On examination you find many of the joints quite full of pus, and the synovial membrane, and frequently the articular cartilage, destroyed. Sometimes the ends of the bones are laid quite bare, and the cartilage dissolved in the purulent matter: occasionally also pus is present in the muscles. The result is uniformly fatal, although the affection may run a course of several weeks.

The annexed case is a good illustration of what I have just said. Here the joints were the seat of the secondary pus-formations, and disorganization was rapid and complete. The symptoms at first were mistaken for those of rheumatism, and this, I think, is not an uncommon error of diagnosis, which indeed had been at first committed in the case of our patient Gordon.

Case LXX (vol. ii, p. 186).—Ann Davies, a married woman, twenty-three years of age, after a severe and protracted labour, and much neglect and bad nursing, suffered from what was, at first, supposed to be an attack of rheumatism. Her medical attendant (Mr. Dunn) suspected that "the veins on the left side were also affected." She was admitted into Augusta ward, February 24th, 1841, about six weeks after her confinement, with the following symptoms:—

"Great dyspnœa; a pale and anxious countenance; considerable anasarca of the lower extremities; a small and rapid pulse. There was a tumour, about the size of an orange, over the right sterno-clavicular joint, soft and fluctuating, and evidently containing pus. On moving the arm, a grating sensation was felt in the shoulder-joint of that side."

Stimulants were administered, but with no benefit; she became very delirious during the night, the dyspnœa more urgent, and she died the following morning.

After death the abscess was found to involve the sterno-clavicular joint, the cartilages of which were entirely destroyed, and the end of the clavicle was softened. The abscess extended among the muscles. The shoulder-joint was found in a condition similar to that of the sterno-clavicular, and several ounces

of pus escaped. The left knee-joint also contained a small quantity of sero-purulent fluid, and a false membrane. Pus was also found in the left sacro-iliac joint. No purulent deposits were found in the lungs or other parenchymatous organs, but the bronchial tubes contained some sero-purulent fluid. Neither the uterus, peritoneum, nor veins generally, presented any morbid appearances. The heart contained firm coagula in all its cavities, adhering pretty closely to the cardiac walls.

The third class of cases is much less fatal, and to this belongs the case of our patient Gordon. The less frequently fatal termination of cases of this class appears to be due to the different parts of the capillary system affected. In our patient the deposits have chiefly occurred in the cellular tissue and muscles; and the result will probably depend upon the state of the lungs and the patient's strength. I believe that the lungs are fast recovering their healthy condition, and therefore he has only to contend against the external formation of pus. If we can keep up his strength long enough for the large abscesses on the hip and buttock to fill up and close, and if no fresh collections of matter occur in other parts of the body, we may confidently look forward to a favorable termination.*

I will now add an instance of the successful treament of a somewhat different example of this class:—

Case LXXI† (vol. lviii, p. 15).—Sarah Butcher, a nurse in our hospital, after having been in attendance on some very bad surgical cases, became generally ill, and suffered from pain and aching in her limbs, and slight sore throat; at the same time her right thumb became inflamed, for which she could assign no cause, as she did not remember to have had any excoriation or wound. An incision was made, but no matter escaped. A small swelling then made its appearance in the axilla, but quickly disappeared again. At first she was ordered a mixture of ammonia and chloric ether three time a day, a little morphia at night, and poultices to the thumb. On the 30th of April, her pulse was 104, and her tongue covered with a thick blankety

* This man ultimately recovered; and in August was sufficiently well to leave for a convalescent institution.

† Reported by Mr. H. F. Winslow.

fur. Half an ounce of brandy was ordered every two hours, five grains of compound soap-pill at night, and, as her bowels were confined, a purge.

After this, the whole hand became swollen, tender, and extremely painful, so as to prevent her sleeping; and on the 3d of May, slight fluctuation was felt under the palmar fascia; her pulse was 112. On the following day she was seen by Mr. Fergusson, and by his advice the house physician applied ten leeches. Her pulse and breathing that day were 116 and 36. During the three following days her hand and arm continued very tender and painful, and much swollen. On the 5th, the pulse was 104; on the 6th, 102; on the 7th, 96. A peculiar rose-coloured vesicular eruption became developed on the 7th, over all her body; she complained of pain in her joints; the brandy was increased to twelve ounces; an incision was made in the hand, and though no pus escaped at the time, yet it seemed to afford some permanent relief from pain, and by the 11th there was a free discharge of pus from the wound. She was now taking one grain of opium every four hours.

On the 13th, a very great rise in the pulse accompanied a fresh formation of matter in the arm, and declined after its evacuation by incision. The wounds continued freely discharging; but on the 17th, the pulse again became more frequent; and on the 18th, the brandy was increased to twenty-four ounces, *i. e.*, one ounce every hour. On the 22d, a fresh exit was given to the matter by an incision in the palm. There was still great pain.

On the 24th, the opium was ordered to be given in the form of tincture, with bark. On that day the pulse was 92; on the 25th, it had risen to 100; on the 26th, to 108; on the 28th, to 118; on the 31st, to 124. There was no general change for the worse corresponding with this increasing pulse; but after an incision had been made over the back of the wrist, and a quantity of matter let out, the pulse fell, in a few hours, from 124 to 84; her tongue became quickly clean, the pain greatly diminished, and she improved generally. Soon afterwards the discharge of matter ceased, and cold-water dressing was substituted for the poultices, which had hitherto been constantly applied. The brandy was reduced, on the 2d of June, to

eighteen ounces; and as the improvement continued, it was still further reduced to twelve ounces on the 4th, and to eight on the 6th, and then discontinued altogether, and some porter substituted. She suffered from wandering rheumatic pains for some days, but otherwise improved rapidly, and was discharged on the 28th of June, with, of course, considerable impairment of the hand and arm.

This patient had altogether about thirty-one pints of brandy —or about an average of a pint a day, for a month.

The following case is a third example of the same class. It has many points of interest, and the diagnosis was difficult. The symptoms at first closely resembled those of acute rheumatism; but, looking back at the complete history, the true nature of the case is sufficiently obvious. I am sorry to say, the treatment was not such as I should now recommend; it amounted, in fact, to almost nothing, excepting the local measures; and to this I attribute the very tedious character of the illness and the slow convalescence.

Case LXXII (vol. xvi, p. 17).—Caroline Allen, aged forty-two, on October 6th, 1845, was attacked, without any assignable cause, with shivering, fever, and oppression; and in the evening observed a red spot on the fore-finger of the left hand, which produced much pain, and was followed by redness and swelling of the hand and arm. The red spots on the finger was lanced by a surgeon, who supposed it to contain pus, but no matter escaped. The next day the right hand and wrist became painful and swollen, and then the left foot and ankle. All these joints were swollen and tense when she was admitted on the 18th. Her pulse was then 120, and the respirations 28; the skin moist; the tongue furred in the middle; there were cough and expectoration of serous fluid, with purulent pellets and some clots of blood, but no corresponding abnormal sounds were heard in the chest.

On the 12th, there was increased fever; the right arm was much swollen and covered with a blush of erysipelas; as also the left leg, but in less degree. Warm fomentations were

applied, and a little morphia given at night. The next day, as the erysipelatous redness continued, the left ankle and foot were scarified.

From the 14th to the 18th, the same symptoms continued with little alteration: pain, superficial redness, swelling, and œdema, varying in intensity, about the left foot and ankle, and the right wrist and hand; and a pulse of 112. On the 18th, the pain in the left foot and ankle was very severe, and the palm of the right hand became swollen, prominent, and red; the swelling extended along the palmar surface of the fingers, but no distinct fluctuation could be detected in the palm. The same expectoration of mucus and pus, with small clots of blood, continued, and a pulse at 120. A grain and a half of quina were now ordered three times a-day.

On the 20th, the limbs were placed on splints, at first without, then with bandages; cold lotions were applied, and some wine and good nutritious food were ordered about this time. Still the same parts remained very painful and swollen; the pain at times was much aggravated, and at others abated; the pulse continued weak and rapid, varying from 108 to 120, and very little progress was made for many weeks. On the 8th of November, an issue was established over the right wrist, and some time later a second. Now and then we could feel, more or less distinctly, fluctuation about the joints, especially the wrists; the cough and expectoration continued without corresponding physical signs in the chest.

About the 21st of November, our patient began decidedly to amend: the pulse improved and diminished in frequency, and the local symptoms subsided. She continued on a tonic plan of treatment, very slowly improving, till December 31st, when she was discharged cured. At first the joints, as one might have expected, were somewhat stiff and useless.

Before we part, let me say a few words upon the treatment of these cases. This may be summed up in one word—*support*. In fact, in these cases, there is a struggle between the depressing influence of the pyogenic process and the strength of the patient; hence, the more support we are able to give him, the more favorable will his position be to meet the struggle. You

have seen this plan of treatment fully carried out in the cases of Gordon and Sarah Butcher. We also get a hint as regards the management of our puerperal cases, and we are practically cautioned against the fashion (now happily becoming extinct) of giving depressing medicines to puerperal women. The woman's vital powers after labour are more or less weakened; she has gone through what may be compared to a severe surgical operation, and to this the analogy of labour becomes more striking, when we consider that the part of the uterus to which the placenta was attached resembles an extensive raw surface, or immense wound; the vessels are torn, and more or less hæmorrhage must have occurred.

I believe that now-a-days experience decides in favour of upholding the strength in a moderate way after parturition; at least this seems to be the opinion of many accoucheurs to whom I have had an opportunity of speaking on the subject. The same remarks will apply to the treatment of severe surgical operations: I should imagine that it would be now very difficult to find a surgeon who would advocate the old plan of preparing a patient for an operation by bleeding, strong purging, and other very lowering treatment, as was formerly the custom. It is the same as regards the treatment of severe injuries, including burns; we must supply the patient with moderate support, and give him as much nutritious food as his powers of digestion will bear. In cases of erysipelas, we must always bear in mind the very doubtful nature of the case, and must avoid giving, unhesitatingly, a very favorable prognosis, lest, when we least expect it, a formation of pus should occur, and some of the purulent matter entering the circulation, rapid exhaustion should follow, and our patient die under the depressing influence of the poison, just as takes place with the subjects of experiments.

LECTURE XII.

ON CERTAIN ACUTE DISEASES.

PNEUMONIA AND ITS COMPLICATIONS.

GENTLEMEN,—We have lately had some cases of pneumonia treated, with a highly favorable result, upon a plan which differs materially from that laid down by some of our highest authorities in the practice of medicine. I propose, then, in my present lecture, to make these cases the basis of some remarks on the treatment of pneumonia, with a view to solving the problem, what is the best mode of treating this disease; that is, what mode of treatment is best calculated to lead to a speedy resolution of the inflammation, with least injury to the patient's constitution, and with the shortest convalescence.

I will observe, *in limine*, that the plan of treatment which I have pursued in the cases now convalescent, as well as in many others, consists, not in the use of remedies directly antiphlogistic (so called), that is, of remedies intended directly to knock down inflammation by withdrawing blood, the supposed fuel of all inflammation, and by reducing vital power; but in the employment of means which will promote the free exercise of certain excretory functions, by which the blood may be purified, and certain matters removed from the system, which, remaining in it, tend to keep up a state very favorable to inflammatory affections. The remedies to which I refer tend to promote the free action of the skin and of the kidneys, and, in a less degree, that of the intestinal mucous membrane, whilst, at the same time, a free stimulation is maintained of that part of the skin which is near the seat of the pulmonary inflammation. An essential part of this treatment is that, while these remedies are being used, we do not aim at reducing the general powers of the system, but endeavour rather to uphold them by such fre-

quent supplies of nourishment, duly apportioned, both in quality and quantity, and easy of assimilation, as may be readily appropriated, to supply the waste which during the inflammatory process must necessarily take place in the most important tissues of the body, especially the muscular and nervous.

Some physicians have drawn a distinction between cases of pneumonia, which is useful with reference to treatment. There are, they say, two classes of cases of pneumonia,—the one sthenic, the other asthenic and typhoid; the former capable of bearing the most active antiphlogistic treatment, and for which, indeed, they say, that the treatment is absolutely necessary; the latter requiring a supporting and even a stimulating plan, and for which an antiphlogistic one would be extremely hazardous and dangerous. Now, while I fully recognise and admit the practical value of such a distinction as this, I must remark, that it seems to me it ought to be expressly differently. I would say that in all cases pneumonia has, independently of this or that mode of treatment, a decided tendency to depress the general powers of life—in some more, in some less; that, with all, a very decided direct antiphlogistic treatment is hazardous,—with some extremely so,—and in none is it absolutely necessary; but, with others, there is no safety for the patient, unless the treatment from the beginning be of a decidedly supporting and stimulating nature.

You will note the distinction which I make between remedies *directly* and *indirectly* antiphlogistic. The former is a class of remedies whose supposed efficacy is founded upon a notion (an erroneous one, as I think) that certain acute and sthenic inflammations are attended with an undue exaltation of the vital forces, both local and general, and that these must be reduced before the inflammation will yield. I say, I think this view erroneous; for it seems to me quite plain, from the clinical history of the malady, that the local inflammation draws so largely upon the rest of the system, as to depress the general powers of life; else, whence the weakness, the exhaustion, the failure of appetite, the wasting, which take place in the course of the disease, even when favorable, independently of any particular line of treatment?

The remedies indirectly antiphlogistic are those by which it

is proposed to promote and exalt some particular functions,—as sweating, or some other secretion, which tend to purify the blood, by eliminating noxious matters through their proper channels, and by such purification of the blood to reduce or remove febrile symptoms.

Case LXXIII (vol. xxxii, p. 88).—The first of the cases to which I shall refer to-day is that of Edward Mills, aged twenty-eight. He is a railway porter—a good specimen of a strong, athletic man, of active habits, and accustomed to hard work, and, at the same time, evidently one who has been well fed. In short, he is a person just adapted, by constitution and habit, to bear the so-called antiphlogistic treatment, if such were necessary.

On the 3d of January, 1851, he was seized with shivering, headache, shortness of breath, and cough, soon followed by the occurrence of a sharp pain in the left side, particularly upon taking a deep breath, and all this accompanied by loss of appetite and fever. The next day he began to expectorate a quantity of very viscid and rusty mucus, the breathing became more rapid, and the cough more frequent and troublesome; on the 5th he came into the hospital.

The character of the sputa at once attracted our notice; and those of you who came round with me will recollect, that I particularly directed your attention to their extreme viscidity, and showed you how they adhered to the vessel in which they were received; so much so, that I could turn the pot upside down without the least displacement of its contents. We likewise observed the peculiar rusty colour of the sputa. The matter expectorated in such cases looks exactly as if it had been mixed with iron rust. The peculiar colour is caused by the intimate admixture of a certain amount of the colouring matter of the blood, which may be seen in it through the microscope. The mixture is evidently very intimate: there is in fact, in these cases, a hæmorrhage, doubtless from the naked vessels of the pulmonary air-cells. But the appearance and colour of the sputa differ very decidedly from those of the expectorated matter in ordinary cases of hæmoptysis. I think the difference is to be explained thus:—In pneumonia, a copious

secretion of mucus takes place from the membrane of the extreme bronchial tubes or bronchial passages; and blood, escaping from several minute vessels, becomes intimately intermixed with the mucus, and gives it its rusty colour. The escape of blood and the secretion of mucus take place simultaneously, and in nearly constant proportions, and are dependent on the same cause, namely, that which irritates the lung. But in hæmoptysis the escape of blood is independent of any secretion of mucus, and often takes place without it, and the quantity of blood is always greatly in excess of that of mucus.

When present, this viscid and rusty state of sputa is a pathognomonic sign of pneumonia. Indeed, the great viscidity alone, even without any red colour, but with a yellowish, bilious hue, ought always to excite our apprehension, lest pneumonia may be commencing. Passive congestion of lung, such as we may meet with in low fevers or in heart disease, will, however, sometimes give rise to sputa not unlike those of pneumonia, but to be distinguished from them by their being less viscid, and containing more blood, which is much less intimately mixed with the mucus.

Although the rusty and viscid character of the sputa will often enable us, with certainty, to diagnose the presence of inflammation of the substance of the lung, we must not conclude that the absence of this characteristic expectoration is a positive proof against the existence of pneumonia. Many cases of pneumonia pass through all their stages, and resolution takes place, without the occurrence of any expectoration, or with that of a very trifling amount of colourless mucus. In the cases of typhoid pneumonia, it is not uncommon to find that the patient does not expectorate, probably owing to his weak and sluggish state, his sensibilities being much blunted; or the expectoration has peculiar characters, being much less viscid, non-adherent, and its colour much darker, exhibiting an appearance which has been likened to that of prune juice.

We found that our patient exhibited certain well-marked constitutional symptoms. He had a flushed face, a hot and dry skin, and a tongue coated with a thick white fur. He also complained much of thirst, and of a troublesome cough, and he suffered from a shooting pain in the left side, below and a little

in front of the scapula, and extending over the shoulder, which impeded his breathing, and was increased by deep inspiration. At the same time there was no very great increase in the quickness of pulse (it was 96), but the respirations were much more frequent than in health, being 36. The pulse was full and strong, and such as would have not only justified, but invited bleeding, if we had allowed ourselves to be influenced by that single symptom in adopting such a line of treatment.

A careful examination of the chest at once enabled us to determine the exact nature of the evil under which this patient was suffering. By percussion and auscultation we found a normal state of the right lung, the breathing being, however, rather more intense (puerile) than in health. On the left side, the percussion sound was quite natural in front, and good vesicular breathing was audible. Such, likewise, was the case all over the scapular region behind. But at the base of the left lung, all about the region to which he referred his pain, a decidedly dull sound was elicited by percussion.

Now, this dulness of percussion may arise, as you know, either from an effusion of fluid between the pleural membranes, or from a thickened state of pleura, or from a condensation or such other alteration of the lung as prevents its free distension by air. When fluid is interposed between the pleuræ, the vibrations excited in the lung by the voice are not propagated to the wall of the chest, unless in cases where some old bands of adhesion serve to connect the surface of the lung with the costal pleura.

In the present instance, the vibrations were sensible to the hand over the dull surface, and, therefore, we had to seek some other cause for the dulness, besides pleuritic effusion. There was no history of any former attack of pleurisy; therefore it was improbable that there could have been any thickened state of pleura. We found, however, a decided modification of the voice and breathing, which sufficiently accounted for the dull percussion. There were both bronchophony and bronchial breathing. All these conditions occur only in a solidified state of lung; and when accompanied by viscid, rusty sputa, and the train of symptoms which we found in our patient Mills, we have no hesitation in referring the solidification to the effusion

of plastic matter into the air-cells of the lung, which likewise serves to exclude the air from them.

The seat of the effusion of plastic matter in pneumonia is in the fine air-passages within the lobules of the lung, the interlobular passages being free. The air, therefore, in inspiration rushes through these tubes; and the vibrations which it excites on the walls of the tubes are readily propagated by the solid lung to the surface, and thus the phenomenon of tubular or bronchial breathing is heard—a sound similar to that which one may cause by simply blowing into a tube, or of which you may get a natural example by applying the stethoscope over the larynx and trachea in the neck.

And in bronchophony, it is as if the vocal sounds were generated in the air-passages; the voice seems to come from the lung. The fact is, that the vibrations excited at the local cords in the larynx are propagated along the walls of the bronchial tubes; and instead of being diffused through the soft, spongy lung, as in health, they are conducted in full force to the thoracic wall by its solidified portion.

You must not lose sight of the fact, that bronchial breathing and bronchophony may be present whenever a part of the lung is solid, whatever be the cause of the solidification, provided only a great portion of the bronchial tube leading to it be pervious. Thus it is that we often find these signs, when tubercular deposit has solidified a greater or less portion of lung. Sometimes pneumonia, running an insidious and chronic course, will make the lung solid, and develope these signs; or cancerous deposit may have been slowly taking place. To determine, then, the exact signification of these signs, you will have again to call to your aid the history of the symptoms of the case.

Again, you may have bronchial breathing when a slight pleuritic effusion has taken place; but such bronchial breathing will be accompanied, not with bronchophony, but with ægophony—a state of voice resonant and bronchial in its character, but rendered bleating by the interposition of a thin layer of fluid between the costal and pulmonary pleuræ. The existence of this modification of voice, and the simultaneous

absence or great diminution of the usual vocal vibration when the hand is applied closely to the wall of the chest, will distinguish the bronchial breathing which is accompanied by pleuritic effusion from that which is due to the simple condensation of pneumonia.

Thus, then, the percussion, which we first practised on making our physical examination of the chest, directed us to the seat of lesion, and from it, together with the bronchial breathing, the bronchophony, the rusty expectoration, the local pain, the embarrassed breathing, and febrile disturbance, we diagnosed with confidence and certainty that the lower lobe of the right lung was in a state of inflammation, and had passed rapidly into hepatization. It is probable, too, that the inflammation of the lung was accompanied by some degree of pleurisy. Of this, however, we had no certainty, but three circumstances rendered it probable: first, because the local pain was sharper than it usually is in pneumonia; secondly, because on one day the brochophony was decidedly ægophonic in character; and thirdly, because pneumonia (unless quite central and deep-seated) rarely occurs without some degree of pleurisy affecting the pulmonary layer of the pleura.

The portion of lung involved in the inflammation was rather more than one third of the posterior part—the inferior third—and the inflammation extended half-way, or possibly two thirds, forward, towards the anterior surface of the lung. This was also the case in the second example of this disease which I shall bring before you to-day.

It is a remarkable feature in the clinical history of pneumonia how prone it is to attack the lower part of the lung, and how much more frequently the posterior part is affected than the anterior, and how often the inflammation involves only a portion of one lung, and that portion not exceeding one third or one half, not often reaching to the anterior surface, and seldom extending through the entire lung; and, lastly, how when both lungs are involved, they are symmetrically affected, so that you rarely find pneumonia in the base of one lung and the apex of another, but in the bases of both or the apices of both. I know of no satisfactory explanation of its partiality

for the posterior and inferior part. Perhaps dependence of position, as likely to affect the circulation, may have something to do with it.

The extent of the inflammation, and, perhaps, in some degree, its position, ought always to be taken into account in forming a prognosis. If only a portion of one lobe is affected, recovery is much more frequent than the reverse, provided the treatment have not been of a too depleting nature; should the whole of one lobe be involved, or more than half the lung, the chances of recovery are much diminished; and should the inflammation engage the whole of one lung, the disease is very frequently fatal. When the pneumonia is seated in the upper lobes, the chances of recovery are much less than when it occupies the lower lobe. This is partly because when the disease attacks the upper lobe it is of a more asthenic nature, and partly also because this part is most apt to be affected in old persons. The clinical fact is highly deserving of your attention, and ought to exercise an influence upon your treatment.

Treatment.—Now, the treatment to which this patient, and the others whose cases I shall presently detail, were subjected, consisted in free counter-irritation by the application to the back and side, over the region of dulness, of flannels soaked in warm spirits of turpentine, which were kept on for half an hour. These stupes were applied at three several periods of the day, for the first three or four days. They excited considerable irritation and redness of the skin. A diaphoretic medicine was also administered, consisting chiefly of the liquor ammoniæ citratis, of which as much as six drachms were given every three or four hours; an occasional dose of a mild aperient medicine was given, and for food the patient was allowed at least a pint of beef-tea daily, with milk and bread.

Let us see, then, the progress of the case under this treatment.

The patient, you will remember, came in on the 5th of January, which, reckoning from the date of the first occurrence of rigor and pain, was the third day of the disease.

On January 6th, he was much the same as on his admission. Pulse, 96; respirations, 36.

On the 7th, a small patch of herpes was noticed at the right angle of the mouth, a phenomenon very common in pneumonia, and for some reason which I cannot explain, generally of favorable import. Pulse, 90; respirations, 36. A decided crepitation was heard at the end of each inspiration, although the breathing remained tubular. Sputa abundant, and less viscid.

On the 8th, pulse, 80; respirations, 40. The dulness on percussion was less extensive, and returning crepitation was now audible over the whole of the region of inflamed lung; fever less; tongue cleaning. The pain in the side being troublesome, ten leeches were applied.

January 9th (seventh day of the disease).—The pain was much relieved; bronchial breathing completely replaced by vesicular breathing and crepitation. Pulse, 96; respirations, 30.

On the 10th, the skin was moist and soft; sputa no longer rusty; crepitation audible on deep inspiration; and the voice slightly resonant beneath the scapula. Pulse, 60; respirations, 32.

On the 12th, the crepitation being still present, a blister was applied to the side; and on the 13th it was reported that the breathing was nearly pure, crepitation having almost disappeared. Pulse, 68; respirations, 28.

On the 17th, fourteen days after the first seizure, our patient was quite convalescent.

Case LXXIV (vol. xxxii, p. 92).—The second case is that of a lad named Minns, aged seventeen, a waiter at a coffee-house. In his vocation he is a good deal exposed to cold, but his health has always been very good. His illness commenced on February the 8th, with shivering, loss of appetite, cough, pain in the right side, and vomiting. In this patient the signs were of the same character as in the former, but were situated in the lower part of the right side. There was a similar acceleration of pulse and breathing. The percussion was dull over the lower part of the right lung behind, where also there was a total absence of vocal vibration. In other parts of the chest, the breathing was pure, and the percussion resonant.

Let me remark here, that in watching cases of acute chest

disease, it is very important to note the frequency of the pulse and respirations, as furnishing one of the most useful guides in the progress of the case. When matters do not go right, you find the frequency of pulse and breathing increase from day to day, or remain stationary; but if, on the other hand, you find a gradual fall in the frequency of the pulse and breathing, you may feel sure that your patient is making satisfactory progress.

These physiological signs are not inferior in importance to any afforded by the patient in the course of his malady; and you may even trust to them alone, when they take a favorable course, to assure you, in the first instance, that the disease is not extending itself, and afterwards, that the inflammation is being resolved.

The diagnosis of this second case differed from that of the first, in this,—that, inasmuch as, in addition to the dulness on percussion, the usual vibrations excited by the voice were not felt when the hand was applied over the dull surface of the chest, there must have been fluid interposed between the lung and the pleura, to prevent their propagation to the walls of the chest; whence we concluded, that a slight pleuritic effusion accompanied the hepatization of the lung.

The progress of this case, under the same treatment as that applied to the first, was equally satisfactory.

Thus, he came in on the 10th of February, the third day of the disease; his pulse was then 118; his respirations, 38.

On the 11th, the pulse was 128; respirations, 34.

On the 12th (the fifth day of the disease), we found the pulse still high, 120; respirations, 40. There was, however, a manifest improvement in the physical signs. The vocal vibration was now to be felt, showing that the pleuritic effusion had been absorbed. Slight crepitation began to be audible near the base.

On the 13th, the pulse had fallen to 96, but the respirations were still as high as 42.

On the 14th, the seventh day of the disease, the pulse had fallen still lower, and, what was more important, the frequency of the breathing was reduced to 32; crepitation was now distinctly audible over the whole of the diseased portion of lung.

On the 15th, the crepitation had become much larger and

moister, and a good deal of vesicular breathing was audible at the lower part of the lung; and on the 17th (the tenth day of the disease,) all traces of crepitation had disappeared, and the dulness on percussion had diminished; the pulse was 78, and respirations 29.

Four days afterwards the patient left the hospital, quite restored to health.

Here, then, are two cases which I think you may take as examples of pneumonia, or more correctly, of pleuro-pneumonia of an average degree of severity. They by no means belonged to what is commonly called the asthenic or typhoid kind. The first, indeed, was distinctly sthenic; and the patient was a strong, athletic, muscular man, just such as one would suppose might be bled without much hesitation. The other patient was not of so vigorous a frame; but still, neither his constitution nor his symptoms were such as would have justified our regarding the case as, in the ordinary sense, asthenic. Yet you will observe, that, in the first case, the inflammation was fairly undergoing resolution on the seventh day of the disease, and on the eleventh day the lung was in its natural condition; on the fourteenth day the patient was convalescent. In the second case, resolution was fairly established likewise on the seventh day of the disease, and on the fourth day of the treatment; and pure breathing was audible on the tenth day. A fortnight likewise was sufficient to restore this patient to complete convalescence. It is remarkable that, in both cases, the resolution should have taken place on the same day of the disease; but then it must be noted, that in both the treatment began on the same day, namely, the third from the seizure.

I have brought these cases before you as good illustrations of the progress of the disease under a mode of treatment which I have found most successful in a considerable number of cases both in hospital and private practice. In this treatment, no attempt is made to cut short the disease: it is founded upon the observation of the way in which the disease is often spontaneously cured, through critical evacuations of sweat or urine or of both, and consists, as I have already said, in an attempt to promote both these secretions.

But I am quite prepared to hear it objected, that such a treatment is really doing nothing but leaving the disease to take its own course. Well, and if that course be to recovery in a short time, and at no expense to the powers of the patient, can we adopt any plan better suited to him? I do not admit, however, that the frequent application to the chest of such counter-irritants as mustard or turpentine (three or four times a day), and large doses of the acetate or citrate of ammonia, and occasional purging, exercise no influence, either upon the whole system or upon the local disorder. The drugs cause, undoubtedly—and especially when the patient is kept in bed—free sweating or free diuresis, and often both; and it is quite consistent with all experience, that frequent counter-irritation exercises a beneficial influence on internal inflammations, and relieves pain.

I am not, indeed, aware of any mode of treatment which can be said, *bonâ fide*, to cut short the disease. The plan by bleeding and tartar emetic does not do so, certainly. I have heard it stated, that large doses of digitalis will sometimes cut short pneumonia. Digitalis is an uncertain drug, not always possessing an equal amount of power, and sometimes not very controllable. Moreover, there are certain idiosyncrasies which do not bear the use of digitalis; nevertheless, it is not undeserving of trial in cases chosen with judgment. But, generally, I believe we do more good in pursuing a simple plan of treatment, such as I have described, than in endeavouring to cut short the disease by remedies whose action is at most uncertain, but which may, now and then, do serious harm. By following the plan I have laid before you, if the patient dies, it will be rather from a negation of treatment than from any other cause: the treatment will prove unsuccessful, simply because the disease went on unchecked by it; and in such cases it is often a question if, by any known plan of treatment, such a result could have been averted.

Now, in the particular cases under consideration, a manifest check to the advance of morbid change took place immediately the treatment was begun, and the signs of resolution followed very speedily; and I have already alluded to the curious fact, that the resolution in both cases was established on the same

day of the disease, the treatment having likewise commenced on the same day—a fact which seems very much to indicate that the treatment had a good deal to do with the early resolution of the inflammation.

In estimating the value of this or that mode of treatment in any given disease, we should ascertain what are the natural tendencies of the malady—to recovery or to death. Is it a very fatal disease? When recovery takes place, what is the process? When death occurs, what are the immediate antecedents, and what its immediate causes?

These are points of clinical history upon which our returns are, as yet, far from being complete or exact. The numerical returns which we have respecting pneumonia are unsatisfactory because in those returns all cases of the disease, whether asthenic or otherwise, and whatever be the extent of lung involved in the inflammation, are classed together. Suppose you were making inquiry respecting the results of treatment in cases of burns, how little information could you derive from numerical returns, if the cases were not classified according to the extent of surface involved in the burn! It is just so with pneumonia; we have as yet no classified returns; but, looking to general experience and such numerical returns as we have, it may be stated that, as in burns, pneumonia is fatal in proportion to the extent of pulmonary surface involved; but that in cases where one fourth or a less portion of lung is inflamed it has, on the whole, a very decided tendency to recovery. On the other hand, when the whole of one lung is involved, or when a considerable extent of both lungs is engaged, the tendency is as decidedly to a fatal result.

The fatality of pneumonia is also much influenced by the period at which the disease may have been detected, and some kind of medical treatment adopted, to the extent even of the mere adoption of the horizontal position in a warm bed. The Grisolle's tables show that when the cases were brought under treatment within the first three days, only one thirteenth died; but if not brought in before the fourth day, one eighth died; on the seventh, one third; on the eighth, so large a proportion as one half died. Age likewise exercises an important influence, and there can be no doubt that (excluding infancy, respecting

which our facts are of the most unsatisfactory nature) the mortality increases with the age; and, at the advanced periods of life, pneumonia must be regarded as a very fatal disease.

There are those who think, that when pneumonia affects the apex of the lung, it has a more fatal tendency than when it affects the base. I myself lean very much to this opinion, because in such cases (without referring to complication with tubercles) the disease is generally of the low or typhoid character; nay, under such circumstances, the pneumonia may be erratic, and, like erysipelas, pass from one part to another of the same lung, and even to the opposite.

It may, indeed, I think, be laid down, that in all these cases the pneumonia has a fatal tendency in proportion as it tends to exhaust, whether by its extent, or through too feeble powers of resistance on the part of the patient, or by exposure to cold and want of food, or by mental or bodily exertion during the early stages of the disease, or through some powerfully depressing influence connected with the original exciting cause of the disease, such as influenza, rheumatic fever, &c.

But if we take cases of pneumonia occurring in persons in the full vigour of life, and not involving a very large portion of the lung, and coming under treatment early, we may regard it as a disease of not very fatal tendency, but rather prone to get well, when the vital powers of the patient have not been too much depressed; and such cases will get well whatever be the treatment early adopted, provided no great error has been committed in either direction, either in that of reducing too much or of supporting too much, and it must be obvious to you, that that which most easily admits of correction is the latter. What we have to do in such cases is, to adopt the treatment which favours the shortest convalescence, and in the more severe cases we have to discover a mode of treatment which will promote the reparative process and uphold the powers of life.

The plan of treatment which has been recommended by some of our highest authorities, I need not tell you, is that by bleeding and tartar emetic. You bleed early from the arm, and if necessary you bleed a second or a third time; and if under this treatment resolution does not speedily take place, you bleed locally by leeches or by cupping, and likewise give tartar-

emetic more or less freely; to all which counter-irritatio may be superadded in the more advanced stages. Mercury i also to be given freely, even to salivation, combined wit opium.

I have had ample experience of this treatment; and I mus confess it has given me so little satisfaction, that I have for som years ceased to adopt it; for, under this treatment, I have see too many die; and when recovery has taken place, in too man instances has it been with a tedious, lengthened convalescenc Indeed, of all the fatal cases which it has fallen to my lot t witness, the great majority have been treated in this way; an in most of them antiphlogistic treatment had not been carrie to an excessive or unreasonable extent.

In bleeding, the difficulty is to determine how much blood yo may safely take away. Upon this point, I think, all who vie the matter candidly must acknowledge that we have no satisfa tory rule, notwithstanding the immense experience we have h of the practice. A loss of blood which scarcely makes an ir pression on one man will seriously reduce another; or a patien who in a former illness has borne bleeding well, will suffer fro it very much on a subsequent occasion; or losses of blo which were borne with impunity in one epidemic are injurio in another. All these are difficulties with which the greate tact and judgment frequently find it impossible to cope. A although, in particular cases, relief may be afforded to certa symptoms by a timely bleeding, there is no doubt that in ma it exercises no real influence in checking the progress of t disease; for, notwithstanding early bleeding, the lung becom fully hepatized; nay, I would go so far as to say, that in so cases it favours hepatization by relaxing the blood-vesse and permitting a more ready transudation of the liqu sanguinis.

And as regards tartar emetic, I have long noticed t patients do best when the drug neither sickens nor purges. this subject I am glad to fortify my own opinion, formed in pendently, by those of two such excellent authorities as the l Dr. Thomas Davies and Dr. Watson. Dr. Watson, alluding Dr. Davies, says: "He states, and this is accordant with own experience of the remedy, that the tartar emetic alw

acts best when it produces no effect except upon the inflammation itself; *i.e.*, when it does not cause vomiting, or purging, or a general depression of the powers of the system." When, indeed, you can ensure your patient against these effects of the drug, it is a very safe and useful remedy in pneumonia; but the difficulty is to limit its action in this way. This may be partially done, though not as regards its depressing effects, by combining opium with it, and the combination often exercises a favorable influence.

I had asked myself, why does this combination of tartar emetic and opium often tell so favorably in pneumonia? and the conclusion which I came to was this:—because it tends very decidedly to promote sweating, and perhaps other excretions; and I was thus led to try drugs of a like tendency, such as liquor ammoniæ citratis or acetatis, in large and frequently repeated doses, which do not exercise such a depressing influence upon the patient.

In using these drugs, you must be careful to give full doses—four, six, or eight drachms, and to repeat them every three or four hours; and you must diligently apply, two or three times a day, or more frequently, counter-stimulants over a considerable extent of the surface of the chest, such as mustard, or flannels soaked in warm spirits of turpentine. With this you may often safely and with advantage combine the use of opium, and now and then a mild aperient will be necessary.

Under this treatment deaths from pneumonia have become extremely rare among my cases. The fatal cases are those of patients who come under treatment being already far advanced in the disease, or in whom the disease has rapidly invaded a large surface of one or both lungs; but even such cases often do well under this treatment, combined with support and stimulants, if begun early.

In all cases, I am careful to give support from the first, in the shape of animal broths in small quantities at short intervals; and in most cases I give wine or brandy early, in a similar way, the dose being apportioned to the degree of depression of the nervous system.

In the decidedly typhoid cases, I need scarcely say, that the free use of stimulants is of essential importance; and it is often

of immense advantage to give quinine freely, the specia indication for this latter drug being profuse sweating.

Let me now direct your attention to a third case of pleuro pneumonia. The attack was ushered in with symptoms c pleurisy, but afterwards pneumonia came on with well-marke signs. This is one way in which pneumonia manifests itself— the characteristic symptoms of the affection becoming develope upon an attack of pleurisy. I bring this case, also, before you not so much to illustrate the history of the disease, as becaus it affords a good example of a case of rather severe pneumoni proceeding favorably, and terminating in a very satisfactor manner, under the plan of treatment which I have recom mended to you.

Case LXXV* (vol. xxxvii, p. 174).—The patient is a boy i Sutherland ward, named W. Reddin, aged eleven, well know I hope, to many of you. On the 6th of the present mont (November, 1852), he was seized with shivering and seve headache, which symptoms he attributed to having got a seve wetting a fortnight before.

He was admitted on the 10th, that is, four days after th occurrence of the shivering. When he came in, he had a h dry skin, flushed face, and pain in the side; his pulse was 11 and the respirations 45. Besides these symptoms, there w cough, with sore throat; but the patient did not expectorate.

On the 11th, the following physical signs were noted: Dulness on the right side posteriorly reaching as high as t scapula, with diminished vocal vibration, as compared with th found in the corresponding position on the opposite side. Th led us to inquire whether fluid had not been effused into t cavity of the pleura in the situation above indicated. Up carrying the hand down quite to the base of the right side the chest, it was discovered that vocal vibration was tota absent,—tolerably certain evidence of the presence of flu Bronchial breathing was also audible behind, and a modifi condition of bronchophony, which may be called "ægopho

* Reported by Mr. E. Liveing.

bronchophony." From all these signs, then, we inferred that the lower third of the right lung was consolidated, as the result of pneumonia, and that a layer of fluid had been effused into the corresponding part of the pleural cavity, consequent on pleuritis in that position.

Upon listening higher up, crepitation and, a little higher still, pure vesicular breathing were audible. On the left side of the chest the breathing was puerile throughout the whole lung. Hence it was plain that we had to deal with a case of pleurisy accompanied with pneumonia, and consolidation of a certain portion of lung.

Such, then, was the condition of our patient on the 11th. Turpentine stupes were ordered to be applied to the affected part of the chest three times a day, as in the former case, and half an ounce of the liquor ammoniæ acetatis was given every two hours; beef-tea and milk were allowed as diet. Bleeding and all kinds of depletion were carefully avoided. The bowels were moderately acted upon. We were anxious to avoid pulling down the patient, the influence of the morbid process alone being very depressing.

The treatment, then, was commenced on the 11th, or the fifth day of the disease. On the 12th, the physical signs were of much the same character. On the 13th (seventh day), the respirations had fallen from 44 to 30. On the 12th, the pulse was 90, and on the 13th, 84; while on the day of his admission it was 116, and on the day after, 112.

On the 13th, the skin was cool and moist, and the tongue clean. Vocal vibration had returned, and the bronchial breathing was less intense; but it had spread a little higher up, while at the same time crepitation was beginning to return in the lower part. With the returning crepitation, a slight pleural friction sound was noticed.

On the 14th the increased vocal vibration had entirely disappeared, and vesicular breathing was heard in the lower part, mixed with large crepitation. Upon placing the ear to the back of the chest, the vocal resonance was found to be natural, showing that the fluid had been absorbed. The pulse had fallen to 74, and the respiration to 24, and the patient was perspiring very freely.

Thus this patient passed through a severe inflammation of a considerable portion of one lung in nine days; but he might be said to have been safe in seven days. The morbid action began on the 6th with rigor, and reached its height in five days, probably in less time; and the time which elapsed from the adoption of the treatment up to resolution did not exceed three or four days, from the 11th to the 14th or 15th. On the 11th there was evidence of hepatization; on the 14th the hepatization had resolved; and since then the lung has been rapidly recovering its healthy condition. If now, in this case, I had taken blood, it would have been said that this good effect was to be attributed to the bleeding, and that the disease had been cut short. Under the remedies, however, which we have been adopting, the patient has done perfectly well, and we have the satisfaction of knowing that his constitution is unimpaired by any treatment to which he has been subjected by us.

Let me next allude to a very interesting point which has been noticed in these cases of pneumonia. It may now be looked upon as an established fact, that in this disease there is either a great diminution in the quantity of the chlorides, especially of the chloride of sodium, in the urine, or these salts are altogether absent from that fluid. This curious piece of chemical history was first pointed out by Redtenbacher, who records the results of his observations of it in eighty cases, in a paper in Hebra's 'Zeitschrift der K. K. Gesellschaft der Aerzte.'

Dr. Beale has since confirmed the observations of Redtenbacher, and, upon following up this subject, has made out, that while there is a deficiency of common salt in the urine, there is a corresponding excess in the fluid poured out into the lung; in fact, that the chloride appears to be drawn from different parts of the system to the inflamed lung.*

The amount of chloride in the urine gradually diminishes up to the period of hepatization, at which time, in the majority of cases, not a trace is to be detected. As soon as resolution commences, the chloride gradually reappears, until it reaches the normal quantity.

* 'Medico-Chirurgical Transactions,' vol. xxxv.

The method of testing for the presence of this chloride is so simple, that you should always ascertain for yourselves the accuracy of this statement whenever an opportunity occurs; indeed, your record of the case will not be complete unless it contains details respecting the quantity of the chlorides. All you have to do is, to add a few drops of nitric acid to a portion of the urine in a test tube, and then a few drops of a solution of nitrate of silver. If chloride be present, a dense white precipitate of chloride of silver, which is insoluble in acids, will fall. If it be altogether absent, no precipitate whatever will occur.

In the patients Mills and Minns, the chlorides disappeared almost completely from the urine during the stage of hepatization, but returned to it on the reappearance of large crepitation, and on the resolution of the inflammation.

What appears somewhat contradictory of the above statement, in the case of our patient Reddin, is, that on the 11th we tested this boy's urine, and found evidence of the presence of much chloride; but on the 12th there was a diminution; and on the 14th the quantity had again increased, so that in this case there was no total absence at any period; and from this circumstance alone we might infer that the hepatization of the lung was not very extensive. The disease began as pleurisy and passed into pneumonia, and it was not until hepatization took place, that the chloride in the urine diminished in quantity. It is quite possible that the precipitate was due to the presence of chloride of ammonium.

In uncomplicated pleurisy the chlorides are not affected. This we had recently full means of testing in the case of Mary Coley, who has just left the hospital. The rubbing sound was very loud and well marked. She was treated with opium and large doses of liquor ammoniæ acetatis, and recovered rapidly. At no period of the case did we find any diminution of the quantity of chlorides in this patient's urine. In this case of pleurisy, then, the chlorides in the urine did not appear to be affected, while in pneumonia these salts undergo a marked diminution or disappear, and this is consonant with larger experience upon this point.

Pneumonia often comes on in connexion with rheumatic fever, or in a highly rheumatic or gouty diathesis. Such cases bear ill the bleeding and tartar emetic system, but are particularly well suited for the plan which I now recommend to you, and in them opium may be freely used with very great advantage.

Most of you will recollect a well-marked case of this kind, of more than ordinary severity, in King's College Ward, about six months ago, which was very successfully treated on this plan.

Case LXXVI (vol. xxxiv, p. 56).—The patient's name was Emma Keep; she was a pale, delicate-looking girl, seventeen years of age. From her history we learned that she had enjoyed excellent health until about a year and a half previously, when she suffered from an attack of rheumatic fever, and was ill for six months. All the large joints were inflamed, and there appears to have been some affection of the heart, as she suffered from pain in the cardiac region and oppression of breathing. As there was no evidence of valvular disease on her admission, it is probable that the pericardium was the part attacked, since when the lining membrane has suffered, abundant evidence of it is usually left, through the impairment of the valves and the production of a bellows sound.

She was admitted on the 1st of November, 1851, and had then been ill only one day; all the principal joints were swollen and painful, and the usual symptom of profuse sweating was present. The treatment at once adopted was that by opium, alkalies, and moderate purgation, with the application of blisters to the affected joints.

On the 3d, the frequency of the pulse was increasing,—a symptom which we always watch with anxiety.

On the 4th, the pulse was 116, and the respirations 34; the patient had an anxious expression of countenance, and complained of pain in her left side. Upon listening attentively in that situation, we were able to hear bronchial breathing, which extended over the lower part of the left lung behind, and there was decided dulness on percussion in the same region.

On the 5th, when listening to the heart, I detected, for the first time, a rubbing sound, accompanying both the systole and

diastole; in fact, the characteristic to-and-fro rubbing of pericarditis.

On the 6th, she still complained of pain in the left side of the chest; the pericardial rubbing sound, the bronchial breathing, and dull percussion continued, the latter extending over the inferior third of the left lung behind, when the patient was in a sitting posture; vocal vibration we found to be entirely absent over a space corresponding with the dulness, and at the junction of the inferior with the middle third of the chest on that side, there was that striking modification of voice called ægophony. Above the dull portion, and corresponding to the two upper thirds of the lung, the breathing was vesicular. At the base of the right lung behind there was some large crepitation.

The signs which I have just mentioned are the most important indications of pleurisy with effusion of fluid; there could, therefore, be no difficulty in the diagnosis so far; but was this all? was there not also solidification of the base of the lung from pneumonic inflammation? I have no doubt that such was the case, the general condition of the patient, as well as the physical signs, favoured that conclusion; but we are not able, in such a case as this, to derive the same certain evidence of the existence of pneumonia, which we usually do, from the physical signs, because a pleuritic effusion, compressing the lung, might alone be sufficient to produce them.

During the 7th, 8th, 9th, and 10th, the condition of the left side remained much the same; but the breathing at the base of the right lung acquired a decidedly bronchial character, crepitation being still audible. Rather later we were able to detect a pleural rubbing sound in the same situation. At this time the sputa were clear, but viscid and adherent to the sides of the containing vessel, and there was also present, in small quantity, the rusty mucus so characteristic of pneumonia.

On the 11th, the phenomena were as follows:—"Pulse 116, respiration 36. Loud bronchial breathing was heard on the left side behind, and vocal vibration could not be detected below the angle of the scapula. There was slight crepitation quite at the base of the right lung, and bronchial breathing just above this." A slight pleuritic friction sound was also heard; vocal vibrations

were present, though somewhat faint; the breathing at the upper part of the lung was clear.

There could be no doubt, then, that a certain amount of pleuro-pneumonia had been lighted up on the right side, in addition to that on the left; we had, in fact, to deal with a case of double pleuro-pneumonia and pericarditis. At the same time we were glad to find that the inflammation on the left side was not extending.

On the 12th, some amount of effusion into the right pleural sac seemed to have taken place, for the voice became ægophonic. Large crepitation and bronchial breathing were still heard. On the left side, the bronchial breathing was less marked; and on both sides respiration was clear, but feeble, to within two inches of the base. The pericardial rubbing sound, which had remained unaltered since its first occurrence, now ceased entirely.

For some days the rapidity of the pulse and breathing had been, on the whole, decreasing; and from this we argued favorably, although the case was a desperate one. On the 8th the pulse and respiration were 120 and 40 respectively; on the 9th, 104 and 36. On the 10th, there was a slight increase in the frequency of the pulse; it rose to 110, but fell again the same evening to 100. On the 11th, the pulse was 116, and the breathing 36. This increase in the pulse was no doubt due to the accession of pneumonic inflammation on the right side. On the 12th, the pulse fell to 96, and the respiration to 18.

On the 13th, there was a very decided improvement in the general condition of our patient. The pneumonia on the right side seemed resolving; the ægophony had disappeared; slight bronchial breathing was still audible at the base. On the left side, respiration was clear and vesicular over all the lung except quite at the base. Vocal vibrations were sensible to the hand to within two fingers' breadth of the lowermost part of the chest, where the voice was still ægophonic. Dulness on percussion remained; and this continuence of the dulness, after the other signs have disappeared, is a fact often observed; indeed, it never disappears with the same rapidity as the other signs, and it will often continue long after perfectly healthy respiration has been restored. I suppose that the pleura becomes thickened, or that some of the effused fibrinous matters are slow to become ab-

sorbed, or that the lung is not quickly restored to its normal density.

On the 14th, there was a return of the pleuritic friction sound on the right side, showing that the fluid had been absorbed, and that the surfaces of the pleura, rendered rough by the deposition of lymph, were opposed and in contact.

The articular affection had throughout been making slow but favorable progress, and by the 17th (the nineteenth day of the rheumatism, or the fourteenth of the chest affection), all the joints were free from pain, and the tongue clean. The heart sounds were perfectly normal, but feeble; the breathing over both lungs was clear and vesicular, except quite at the base, where, on the left side, ægophony was still heard, and on the right, a slight friction sound.

This patient was at first treated as we usually treat a case of rheumatic fever, with alkalies, opium, mild aperients, and blisters to the joints. But when the chest symptoms developed themselves, we then adopted a more free counter-irritation by the repeated application of turpentine stupes to the chest, as often as every two hours. Blisters were also applied over the region of the heart.

Opium was also more freely exhibited. I deemed the use of opium to be more urgently needed in this case, in consequence of the rheumatic nature and the great extent and complication of the chest disease. Usually it is not advisable to employ opium in large doses in the simpler forms of pneumonia, because it has some tendency to produce further congestion of the lungs and to depress the heart; but in such a case as this, where there was plenty of healthy lung, there was little to fear on that ground. Opium always acts beneficially in rheumatic fever: it relieves the pain, quiets the nervous system, and promotes the elimination of morbid matters by the cutaneous surface. We gave this girl as much as a grain and a half every three hours.

On the evening of the 11th, as matters were not progressing quite so favorably as we could wish, and there was evidence of a fresh inflammation of the right lung and pleura, I thought it advisable to order small doses of calomel. Half a grain was given every three hours, but altogether only three grains were

administered; for on the following day the symptoms had so much improved, that I discontinued it, that we might not give this drug credit for having wrought changes with which probably it had nothing to do. Whether the favorable turn the case had taken was the result of these half-grain doses of mercury, I must leave you to judge; for myself, I must confess that I cannot believe it.

From this time our patient continued daily gaining strength; in a few days she was able to sit up, and was ordered middle diet and tonics; and on the 29th she left the hospital quite well.

Here then, in nine days (4th to the 13th) from the commencement of the symptoms, we find a case of double pneumonia, pleurisy, and pericarditis had run its course, and the subject of it been conducted safely towards convalescence.

Now ask yourselves, in a candid spirit, what was it which mainly promoted this speedy resolution of an inflammation so extensive, involving three such important organs? Was the process of cure a natural process, or was it the result of some influence exerted by the remedies administered upon the inflamed organs? Did they exercise some antidotal influence, or did they alter the blood in such a manner as to cut off from the inflamed part due supplies of what are called the products of inflammation?

Time would fail me were I to enter on the discussion of all these matters, however interesting and important. I must content myself with stating my own conviction, that the process of cure is a natural one, analogous to the union of wounds or the healing of ulcers, and that a normal supply of blood, both in quantity and quality, is as necessary to the healing of the one as to the resolution of the other.

The following case will afford us another good illustration of the clinical history of rheumatic pleuro-pneumonia, in a severe form, and is the more worthy of your attention as it ended fatally. It was also rendered particularly interesting by the existence of symptoms which made the interpretation of the physical signs difficult, and led to a partially erroneous diagnosis.

Case LXXVII* (vol. xxxii, p. 78).—Patrick O'Reilly, a stout and well-nourished lad, aged twenty, was admitted under my care on the 27th of January, 1851. His illness commenced five days before his admission with shivering and loss of appetite, followed by copious perspirations, pains in the joints, scanty and high-coloured urine.

On admission, his skin was hot and sweating, and he complained much of thirst; all the larger joints were swollen and painful; the tongue dry, and white in the centre; respiration a little embarrassed; pulse 108. On auscultation, the heart sounds were normal; but at the lower part of the left side of the chest behind, the expiration was bronchial, and the percussion over the same part dull.

The joints were wrapped in cotton wool, blisters were applied to some of them, and a turpentine stupe to the back.

On the 29th (the eighth day of the disease), he was on the whole much worse, although the articular affection was not quite so bad. Pulse 92, throbbing and intermittent; breathing 57, quick and laboured; tongue dry and brown. He now suffered from thirst, and from a sense of oppression in the chest. The urine was scanty and high coloured, loaded with red lithates, of specified gravity 1030, and containing a little albumen. There was cough and a slightly rusty mucous expectoration.

On examining the chest behind, two patches of bronchial breathing were found, one on either side, below the spines of the scapulæ; the expiration was prolonged, the voice bronchophonic, and the corresponding percussion dull. The base of the left lung behind was less dull on percussion; the breathing retained a tubular character, and there was some crepitation.

There was extended dulness on percussion over the cardiac region, a slight rubbing sound at the base, and a faint bellows sound at the apex.

The turpentine stupe was repeated, and a mustard plaster, to be followed by a blister, was applied over the heart. Five grains of nitrate of potash, with a grain of opium and a grain of ipecacuanha, were ordered to be taken every four hours.

* Reported by Mr. J. H. Sylvester.

During the next two days, January 30th and 31st (the ninth and tenth days of the disease), there was no alteration in the nature of the symptoms, but the patient's countenance began to wear an anxious expression; he lay continually on his back; the respiration became catching, the præcordial pain great, and the to-and-fro rubbing louder. Dulness on percussion and pleuritic rubbing were now detected at the base of both lungs behind. The pupils were not contracted, despite of six grains of opium per diem.

Two grains of calomel were added to each opium pill. Blisters were applied to the back and front of the chest, and the turpentine stupe repeated. Mercurial and savin ointment was ordered to dress the blisters. A purgative enema was administered.

During the first week of February (the 11th and subsequent days) the nature of the symptoms and physical signs remained unchanged, but there were slight variations in their intensity, and some additional symptoms. Thus, signs of bronchial affection and pulmonary congestion, in the form of large crepitation and rhonchus, in the lower halves of both lungs, were noticed; and there was evidence of increased pleurisy in the loud, creaking, vibratory rubbing which could be heard and felt over the bases of both lungs. There appeared to be imperfect resolution of the two patches of dulness, and bronchial breathing in the infra-spinous fossæ. The averages of the pulse and respirations were about 112 and 50 respectively. On the 3d there was drowsiness, with contraction of the pupils; on the 5th (the fifteenth day of the disease) the patient was slightly salivated.

Some anomalous symptoms were also present at this time, such as tympanitic resonance, metallic tinkling, and bulging of the wall of the chest, about the lower angle of the left scapula. These signs were difficult of interpretation at the time; they led for some time to the error in diagnosis of the existence of pneumothorax; but the phenomena of tympanitic percussion, with a metallic tubular breathing at the base of the left lung, were clearly traceable afterwards to the vicinity of an enormously enlarged stomach, pushing up the lung. Nothing could be more striking than the close resemblance

between the phenomena afforded by this case, and those of an example of effusion of air and liquid into a portion of the pleural sac, and fistulous communication with the lung: the tympanitic splash on succession, the tinkling of fluid dropping from one part of the cavity into fluid beneath, and even the amphoric blowing, were present. But the tympanitic percussion sound was definitely limited by the line of a pushed-up diaphragm, whereas it should have varied its position, and spread over the whole left chest (at least posteriorly), had there been air in a non-adherent pleura. This point, if allowed its due weight, ought to have prevented the erroneous diagnosis.

On the 2d, or the twelfth day of the disease, wine was given for the first time to the extent of three ounces. On the 3d all the former medicines were discontinued, and a mixture, containing liquor ammoniæ acetatis with excess of ammonia, and camphor mixture, substituted. On the 4th, the wine was increased to four ounces; on the 5th, to six ounces, or half an ounce every two hours. Quina was now given in the day, and opium at night. On the 6th (sixteenth day) he had eight ounces of wine.

On the 8th of February (eighteenth day) some new symptoms manifested themselves. In the morning there were indications of pericardial effusion, in great pain and dyspnœa and increased extent of dulness over the cardiac region, with diminution of the to-and-fro rubbing and feebleness of the heart sounds. All medicine was omitted, and a blister applied.

In the evening expectoration of puriform matter in considerable quantity came on suddenly; the heart sounds were yet more muffled.

There was no important alteration for some days. The joints remained very painful, the pulse high, and also intermittent, the expectoration profuse.

On the 13th the patient became suddenly worse, his pulse more irregular; he now gasped for every breath, and died in about two hours, on the twenty-third day of the disease.

The post-mortem examination revealed a pericardium distended with bloody serum, except where it was firmly adherent to the surface of the left ventricle. The heart was coated with lymph and hypertrophied, and there was some aortic valvular

disease. Recent lymph was found on the pleuræ, especially over the diaphragm. The left lung was compressed and pushed forward by the heart, and by an enormously enlarged stomach, and exhibited several patches of carnification. Both lungs were much congested; the bronchial membrane congested, and the tubes filled with muco-purulent fluid.

Here was a case of intense pericarditis which seemed to run its course wholly uninfluenced by any part of the treatment. Salivation, which took place on the fourteenth day, seemed to have no control, nor had opium freely administered from the first. Had a more vigorous antiphlogistic (so-called) treatment been at first adopted, had free local or general bleeding been employed, and mercury given earlier and more freely, so as to have a rapid salivation, how would matters have stood? My experience of similar cases would lead me to say that, under such treatment in this particular instance, which *ab initio* was one of great extent and severity, the pericardial effusion would have been more rapid, the embarrassment of the heart greater, the partial resolution of the pneumonia which had taken place would not have occurred, the pulmonic solidification would have been much more extensive, and the fatal termination would have occurred on the seventeenth or eighteenth, instead of the twenty-third day. Were I to treat such a case now, I should support the vital powers liberally, and even largely, from the first with broths and wine, or alcohol in some form, and employ opium and counter-irritation much more freely. The early adoption of a supporting treatment in such a case as this would have limited, rather than favoured, the extent of the inflammation. And even had it failed in this important object, it would have given the patient a greater power of resistance to the depressing influence of disease, and promoted healing power, just as such treatment would have assisted the curative process in a large ulcer, or in an open stump.

ADDENDUM TO LECTURE XII.

The three following cases are extracted from the *Medical Times* for February 19th, 1859, and reprinted here as an important *addendum* to the previous lecture, and an illustration of

the practice there recommended in the hands of three different physicians. The third case is one of typhoid fever of which pneumonia was a prominent symptom.

Case LXXVIII.—James Jackson, aged eleven, was admitted into King's College Hospital, December 29th, 1858 (vol. lvii, p. 181). It appeared that on Christmas-eve the boy had eaten a number of hollyberries, and was afterwards seized with vomiting and violent convulsions. The exhaustion which followed was so alarming that his life was despaired of; but after taking a considerable quantity of wine, he began to revive. A day or two afterwards he was attacked with pneumonia, for which he was admitted under Dr. Todd's care.

When his chest was examined, it was found to be malformed —"pigeon-breasted," as it is commonly called. There was dulness on percussion over the whole right lung behind, and for four or five inches beneath the clavicle in front. Over the dull region bronchial breathing and bronchophony were distinctly audible. His skin was hot and dry; the tongue brown and parched. Pulse 116; respirations 38. He was ordered a mixture containing liquor ammoniæ acetatis, with excess of carbonate of ammonia and chloric æther, turpentine stupes to the chest night and morning, and half an ounce of brandy every two hours.

The report of the next day, December 30th, is as follows:— "He coughs occasionally, but swallows the expectoration. Has rather a drowsy, intoxicated appearance, and comprehends with difficulty what is said to him. Urine, specific gravity 1017; the quantity of chlorides greatly diminished. Pulse 128; respirations 48."

On the 31st the pulse was 123, the respirations 46. He was then expectorating viscid brownish sputa. The drowsiness continued. Chlorides were all but absent from the urine. On the 2d of January the pulse and respirations were 112 and 44 respectively. As his skin continued very hot and dry, the dose of liquor ammoniæ acetatis was increased.

On the 3d, the ninth day of the disease, and the sixth of treatment, resolution appeared to be commencing. Bronchial breathing and bronchophony were persistent over the right lung behind, but in front loose crepitation, mixed with bronchial

breathing, was audible. The quantity of chlorides in the urine was slightly increased. Pulse 104; respirations 40.

By the 5th there was a very great improvement; the pulse had fallen to 90, and the respirations to 24; the breathing was much quieter, and crepitation was audible behind as well as in front. His cough continued troublesome, and he slept badly. The following day the quantity of chlorides in the urine had greatly increased.

On the 10th the pulse was 70, and the respirations 24. The dulness over the right lung, both in front and behind, had diminished, and bronchial breathing and bronchophony were disappearing; loose crepitation remained.

On the 12th, the eighteenth day of the disease, and fifteenth of treatment, the resolution was nearly complete; the dulness remaining over the right lung was very slight, and the chlorides had returned in normal quantity to the urine. There was still increased vocal resonance, with rhonchus, sibilus, and loose crepitation. Pulse 72; respirations 24. Turpentine stupes discontinued.

On the 14th the frequency of the pulse and respirations had slightly increased; they were 88 and 28. The patient's face was flushed, and there was increased dulness, with crepitation, over the right lung. The turpentine stupes were resumed. This unfavorable change was very transient, and by the 17th the pulse and respirations were again 72 and 24, and the dulness was confined to the base of the right lung behind.

By the 19th a small secondary abscess had formed on the left cheek, and was opened. The pulse rose to 100, the respirations to 32. He was ordered three grains of citrate of iron three times a day.

On the 24th the abscess had healed, and he was reported convalescent. He was discharged on the 27th.

Tabular View of Pulse and Respiration.

Date.	Pulse.	Resp.	Date.	Pulse.	Resp.
Dec. 29	116	38	Jan. 6	90	24
„ 30	128	48	„ 10	70	24
„ 31	123	46	„ 12	72	24
Jan. 2	112	44	„ 14	88	28
„ 3	104	40	„ 17	72	24
„ 5	90	24	„ 19	100	32

Case LXXIX.—J. D., aged six, was admitted into King's College Hospital, under the care of Dr. Budd, on December 2d, 1858, in a state very closely resembling delirium tremens. She had been placed out to nurse for some time previously, and her mother could give no account of her illness, except that on November 29th she first complained of feeling unwell. She stated, too, that her child had been fond of spirits, but, as far as she was aware, had never had more than two or three teaspoonfuls occasionally.

On admission: face slightly flushed, with anxious expression. She is in a constant tremor, occasionally delirious, moaning and crying out, and now and then asking for beer and gin; frightened by slight causes. When an attempt is made to raise her in the bed, she cries out; sleeps ill; has frontal pain, and the forehead is very hot. Breathing rather laboured. Chest and heart sounds normal. Skin hot and feverish. Urine loaded with urates; contains no albumen. Pulse 136; respirations 36.

℞. Tr. opii, ℥j; sp. æth. chlor., ℥ij; aquæ, ℥j; statim sumend.

℞. Sp. ammon. arom., ℥x; mist. camph., ℥j; ter die sumend.

Dec. 6.—Pulse 136; respirations 52. She lies in a very drowsy state, but has lost the constant tremor which she had on admission. Has been wandering a good deal, and yesterday she was delirious. On examining the chest behind, there is dulness on the left side about the spine of the scapula, with bronchophony and bronchial breathing. Large and small crepitation are audible over the whole of the left lung behind. Tongue coated and dry. She passes her urine under her.

℞. Sp. ammon. arom. ℥xx; sp. æth. chlor. ℥v; mist. acaciæ, ℥ss; 4ta quâq. hor. sum.

Turpentine stupes to chest night and morning. Wine ℥ij every hour.

8th.—Pulse 100; respirations 40. Breathing less laboured. Still lies in a drowsy state, and is with difficulty roused. No change is perceptible in the condition of the left lung. Tongue coated. Urine turbid, pale, of neutral reaction; contains no albumen, and the chlorides are entirely absent.

9th.—Pulse 100; respiration 40. Crepitation is now heard

over the front of the chest on the left side. Bowels rather relaxed.

10th.—Pulse 100; respiration 36. Face rather flushed; looks more lively. Tongue clean and moist. Takes her food well. Bowels moved once. The physical signs are the same.

11th.—Pulse 88; respiration 28. The crepitation has disappeared from the front of the chest, but bronchial breathing and bronchophony are still audible behind. Urine of normal colour; the chlorides have returned, and are in the ordinary proportion.

13th.—Going on favorably. Omit wine. Pulse 80; respiration 26.

14th.—Respiration 24; pulse 76, weak and intermittent, corresponding with the action of the heart. She looks bright and cheerful, and feels much better. Slight tubular breathing is still heard about the inferior angle of the left scapula. Appetite very good.

17th.—Pulse 64; respiration 24. Looks rather pale and weak.

20th.—Gets up a little every day. Looks much better, and is getting stronger.

23d.—Convalescent.

31st.—Discharged cured.

Tabular View of Pulse and Respiration.

Date.	Pulse.	Resp.	Date.	Pulse.	Resp.
Dec. 2	136	36	Dec. 11	88	28
„ 6	136	52	„ 13	80	26
„ 8	100	40	„ 14	76	24
„ 9	100	40	„ 17	64	24
10	100	36			

Case LXXX.—M. K., aged thirty-six, resident in London for the last twenty-six years, was admitted into King's College Hospital, under the care of Dr. Johnson, on January 13th, 1859. Has been a nurse in the Fever Hospital for the past two years. Says she never had a day's illness in her life before. The present attack came on about three weeks ago, with pains in all the limbs, and rigors. She answers questions in a very con-

fused manner. Bowels constipated; tongue coated with a dark-brown fur; sordes on the teeth. There is considerable engorgement of the right lung posteriorly and inferiorly. The expectoration is scanty and very viscid. The abdomen is covered with a large number of rose-coloured spots, but there is no tenderness on pressure. Urine acid, contains no albumen. Pulse 120; respiration 36.

℞. Ammon. ses. carb., gr. v; sp. æth. chlor., ℥x; mist. acaciæ, ℥jss; 4to quâq. hor. sum. Turpentine stupes night and morning. Brandy, ℥ss every two hours. 9·30 p.m., pulse 132; respiration 40.

Jan. 14th.—Continues much the same. There is rhonchus and sibilus over both lungs posteriorly. Urine decidedly deficient in chlorides. Ordered—Brandy, ℥ss every hour; adde sing. dos. mist. liq. ammon. acet. ʒiij. Pulse 120; respiration 32.

15th.—Pulse 124; respiration 32. Rhonchus and sibilus over both lungs, anteriorly and posteriorly: over the right back there is slight dulness on percussion. Bowels moved once yesterday.

17th.—Pulse 104; respiration 33. Bronchial breathing over the right lung posteriorly, and the dulness has increased. Tongue clean and red at the edges, and coated with a brown fur in the centre. Expectoration viscid, and of a rusty colour.

20th.—Pulse 88; respiration 36. Dulness and bronchial breathing diminishing over right back, and loose crepitation is now audible there. Rhonchus and sibilus heard over both lungs anteriorly and posteriorly. Expectoration copious and rust-coloured.

22d.—Pulse 88; respiration 34. Loose crepitation alone is audible over the right lung behind. Cough much less troublesome; expectoration free, copious, and losing its brown colour.

27th.—Nothing but rhonchus and sibilus heard over the chest; expectoration quite clear. Patient feels well, and suffers only from weakness.

℞. Quinæ disulph., gr. j; acid. sulph. dil., ℥v; aquæ, ℥jss t. d. s.

Feb. 1.—Chest sounds normal; the patient is now convalescent.

Tabular View of Pulse and Respiration.

Date.	Pulse.	Resp.	Date.	Pulse.	Resp.
Jan. 13	120	36	Jan. 17	104	33
9·30 p.m.	132	40	„ 20	88	36
Jan. 14	123	22	„ 22	88	34
„ 15	124	32			

LECTURE XIII.

ON CERTAIN ACUTE DISEASES.

PNEUMONIA.

GENTLEMEN,—The purport of a clinical lecture is to bring more immediately before you the prominent points of one or more cases which may be actually under observation, or may have been recently so. Whilst the more immediate aim of this kind of instruction is to teach you the clinical history of disease by examples, it also tends to assist and direct you in making observations and in keeping records of your observations with such fulness and accuracy, that they may be useful hereafter, not only to yourselves, but also to others.

A diligent and accurate chronicler of the day-by-day phenomena and changes which occur in particular cases of disease is, in his way, a highly important contributor to the cultivation of medical science. He supplies the material out of which may be framed hereafter the most valuable additions to our views of pathology and treatment.

> "Sicut
> Parvula (nam exemplo est) magni formica laboris
> Ore trahit quodcunque potest, atque addit acervo
> Quem struit, haud ignara ac non incauta futuri."

I am sure that it would be impossible for me to thank too cordially, or commend too highly, the gentlemen who, acting as my clinical clerks, have from time to time kept accurate records of such cases as I have had to treat in this hospital; on the other hand, those who have neglected that important duty, and have been slovenly and careless in their records, would be the first to regret their want of attention on finding how utterly

useless and fruitless their records prove to be, for any purpose, either of learning or teaching.

These remarks have been suggested by my having had to look into the records of my cases with reference to the present and one or two succeeding lectures. You know that, now and then, I like to give a group of lectures on some particular malady or class of maladies, and to illustrate them by reference to cases recorded in former years, as well as by those recently under observation. The records of former years are, therefore, of great importance to me, and I am obliged to lay my case-books freely under contribution. During the present session, I propose to direct your attention a good deal to diseases of the lungs.

Although there is no necessary connexion between the acute and the chronic forms of disease of all the great internal organs in general, and of the lungs in particular, one naturally, in discussing the diseases of an organ or tissue, begins with that of simple inflammation; and it forms a very fitting introduction to the study of pulmonary diseases in particular, as tending to familiarise you with some of the more important signs and symptoms of those diseases, and to teach you to what an extent the anatomical characters of the lung may undergo alteration, and yet the organ may ultimately perfectly recover itself. Inflammation of the lung-tissue, too, is the most formidable, although not the most fatal, of the acute diseases of these organs, and under certain circumstances leads to the destruction and breaking down of the lung-substance in a very short time.

It so happens that, during the last week, an excellent example of *simple inflammation of the lung* has been under our treatment in the hospital, in the case of a little boy in Rose ward, and this has afforded us a good opportunity of studying the disease. I need, therefore, make no further remarks on the propriety of my taking first this important subject, but will proceed at once to discuss the clinical history of the disease.

Let me commence, then, with noticing the various circumstances under which PNEUMONIA is met with in practice; in other words, let me state what are its clinical varieties. They are these:—

1st. *Simple pneumonia* (of which you have an example in the

case now under observation, to which I shall have to direct your attention presently); that is to say, pneumonia uncomplicated with disease of any other organ, and occurring in a subject who possesses no marked peculiarity of constitution. But pneumonia is most frequently complicated with inflammation of the pleura, and, indeed, I suspect that we seldom meet with a case of this disease in which this membrane does not participate, more or less, in the inflammatory condition; for the pleura, as most of you are aware, is a delicate film of membrane covering the lung, and receiving its nutrition from the pulmonary blood-vessels; and it is, I apprehend, scarcely possible for the superficial lung structure to become inflamed, without this membrane, which is so intimately connected with it, being also involved in the morbid process. Hence we have the term "*pleuro-pneumonia,*" which is applied to this inflammatory condition of the pleura and lung.

Simple pneumonia is very rare in another sense also, that is, in its freedom from complication with or dependence on some peculiarity of constitution. To make this clearer to you, let me take an illustration. If two men, A and B, both in good health, be exposed to some noxious influence, cold for example, at the same time and for the same period; A will get a severe attack of pneumonia, and B will not. Now at first sight one can scarcely conceive why the pneumonia should attack the one and not the other, for they were both apparently equally well at the time of the exposure to cold; but, if we carefully examine into the previous history of these individuals, we shall find that A is of a gouty or strumous constitution, or has some peculiarity of diathesis which B does not possess, and it is by reason of this that A is seized with pneumonia when subjected to the noxious influence, which produces no such injurious effect on B. No doubt, there are few cases in which there is not some peculiarity of constitution which may determine more or less the access of pneumonia, and influence its duration and mode of termination; still when inflammation of the lung occurs without any concurrent disease, and without any marked peculiarity of diathesis, it is convenient to call it "*simple pneumonia.*"

2d. Pneumonia complicated with *acute gout* or with *rheumatic fever,* or associated with a decided gouty or rheumatic

diathesis. This form of pneumonia is of very common occurrence, especially in aggravated states of the gouty or rheumatic constitution. It sometimes comes on in the middle of an attack of acute gout or rheumatic fever; at other times it ushers in the attack; while, in a third set of cases, it follows the constitutional malady,—the one or other of these, as the case may be, passing away, and leaving behind it inflammation of the lung.

3d. *Strumous pneumonia; i. e.,* pneumonia connected with the development of tubercles in the lungs, or occurring in subjects of a strumous constitution, without any evidence of tubercular deposits in these organs. This latter form is frequently met with in strumous children, and is often, I suspect, mistaken for *phthisis.*

4th. *Typhoid pneumonia;* by which I mean, inflammation of the lung coming on in a low state of the system, and associated with a series of typhoid symptoms, such as great prostration, a brown tongue, and a languid and feeble condition of the circulation; or you may have the disease specially connected with either typhoid or typhus fever. (Case lxxx.)

5th. *Traumatic pneumonia; i. e.,* pneumonia succeeding and consequent on injuries to the chest, or following severe surgical operations.

These are the distinct and undoubted clinical varieties of pneumonia, which you must be prepared to deal with in practice.

Some physicians speak of *lobular pneumonia* as occurring in young children, and associated with extensive bronchial inflammation or with hooping-cough. But I believe that the condition of lung which in these cases has been attributed to inflammation is not so, but is merely a state of carnification due to the exhaustion of air from parts of the lung by the excessive expiratory efforts which occur in these cases.

Let me now explain to you in what pneumonia consists, and what are the anatomical characters of the disease.

Pneumonia may be defined to be "that condition of lung which leads to the formation of a plastic deposit in the cavities of the air-cells, which plastic material, by filling up the interior of these cells and the finest bronchial tubes, consolidates the

previously soft and crepitant pulmonary tissue." This plastic substance is probably of an albumino-fibrinous nature, formed, no doubt, from the liquor sanguinis which exudes from the pulmonary blood-vessels. These vessels project, as you are aware, uncovered, from the walls of the air-cells, and are, consequently, very favorably situated to permit transudation through their coats. This exudation, by its coagulation, very quickly consolidates the lung, and, being mixed, more or less, with the colouring-matter of the blood, gives that organ, when cut into, very much the consistence and colour of liver. I have known good anatomists mistake for liver a piece of lung in a state of *red hepatization*. This condition, which, as I just now said, is simply produced by the effusion of a plastic material entangling blood-particles, and the consequent consolidation of the air-cells and finest bronchial tubes, converting the previously soft and spongy pulmonary tissue into a solid mass, is the only *certain* indication, with which I am acquainted, of pneumonia having existed during life.

This state of red hepatization is, nevertheless, preceded by one in which the blood is delayed in that portion of lung about to become hepatized, and very probably also attracted to it in increased quantity—a state, in fact, of what has been called *active congestion;* in this consists the first stage of pneumonia.

The real nature of the organic changes which constitute this state of active congestion may be thus explained. Some matter introduced either through the bronchial tubes with the inspired air, or through the blood, irritates a certain portion of the lung; in other words, disturbs its nervous influence, and deranges its nutrition. The immediate result of this nervous derangement is an increased action of the heart, a dilated and enfeebled state of the contractile wall of the finest capillaries, which offer no resistance to the flow of blood to that part, and allow it to accumulate there in greatly increased quantity. This relaxed state of the capillary wall increases the size of its pores, and allows a freer transudation of liquor sanguinis than takes place in health, and gives rise to those subsequent changes which produce the condition of red hepatization.

It is not possible, in the present state of our knowledge, to

lay down any anatomical characters by which this first stage of pneumonia can be positively determined after death. The existence of a patch of reddened lung, with distended capillary vessels, the pulmonary tissue being slightly œdematous and increased in density, and the borders of the patch gradually shading off into healthy tissue, would lead me strongly to suspect that inflammation had existed during life, and that that portion of the lung would have speedily passed into red hepatization. But it is very rare for patients to die in this early stage of pneumonia.

Sometimes active congestion will exist in connexion with acute bronchitis. In such a case there will be great intensity of colour in the mucous membrane of the bronchial tubes, large and small, and the congestion will involve the greater part or the whole of one lung, and very frequently of both lungs. But there are no distinct anatomical characters attaching to such a congestion, which would enable the anatomist to predicate of a portion of lung placed before him, this is the congestion of bronchitis, and not of pneumonia, or *vice versâ*.

Passive congestion of the lung may be induced by anything which impedes the passage of the blood through the pulmonary veins, such as diseased heart, or the pressure of a tumour or aneurism on the root of the lung, or on one or more of the pulmonary veins. In asthma and its resultant emphysema, the lung becomes congested in consequence of the imperfect aeration of the blood, and the failure of the proper capillary force, which is so important an aid to the pulmonary circulation. So also in asphyxia, congestion is due mainly to imperfect aeration, and the consequent failure of the same important aid to the capillary circulation.

Whatever delays the flow of the circulating fluid, throws back the blood on the left auricle, and thereby impedes its passage through the pulmonary veins, will produce congestion of the lungs. In various low diseases, which involve a prolonged decubitus on the back, a considerable amount of pulmonary congestion is invariably found as the result of gravitation. In typhus, measles, pyæmia, and other diseases due to the influence of an animal poison, the pulmonary congestion, which is so apt to occur in them, is partly passive and partly active,

owing to the irritant action of the animal poison on the bronchial membrane.

You will see, therefore, that the fact of a lung being gorged with blood, which flows freely from the divided vessels when it has been cut into, is no proof of the existence of inflammation in it during life. This point can only be determined (for the first stage) when such anatomical characters as I have described follow certain symptoms and physical signs; and it is one of those cases which indicate how valueless is morbid anatomy if studied without the light of careful clinical observation.

In the commencement of the second stage of pneumonia or that of *red hepatization*, the plastic material, which is poured out in the air-cells and finest bronchial tubes, takes the shape of these cavities, and is sometimes in part spat up, giving rise to *casts* of the cells and finer air-tubes, which, when subjected to microscopic examination, are found to be analogous to those which are formed in the kidneys in consequence of irritation in the renal tubules.

It is remarkable how rapidly the first stage of pneumonia, or that of active congestion, passes into that of red hepatization; in other words, how quickly a lung becomes hepatized. Laennec asserts that a *fine crepitant râle* may be heard on listening to a lung in the first stage of pneumonia, or that of active congestion, and that this is speedily succeeded by the signs of red hepatization; but, in the whole course of my experience, I have had but very few opportunities of hearing this râle ushering in the stage of red hepatization, and which was considered by Laennec as pathognomonic of pneumonia. This is explained partly by the rapid transition to which I have referred, and partly by the fact by which Skoda has directed attention, that this condition of lung is in general immediately preceded merely by catarrhal râles in the bronchial tubes, and not, as believed by many, by a peculiar kind of fine crepitation.

The second stage of pneumonia quickly passes into the third, or that of *grey hepatization;* the red colour of the lung disappears, but the organ remains solid, and when cut into, looks not unlike a piece of grey granite. This stage, too, like the second, in a very short time passes into one in which more or less of pus is infiltrated into the substance of the lung, and the

matter scraped off the cut surface exhibits, under the microscope, great numbers of cells, having the distinctive characters of those of pus. When pneumonia has advanced even to this stage, the lung may perfectly recover itself, the patient expectorating a large quantity of pus; indeed, so large is the quantity sometimes spat up in these cases, that one is often led to fear that an abscess has formed in the lung. But, curious to relate, a true pneumonic abscess is one of the rarest things which we meet with in morbid anatomy; and I cannot now call to mind more than three instances of this kind that have fallen under my own notice. This fact of the extremely rare occurrence of abscess of the lung following pneumonia is a point upon which authors are singularly unanimous. If death occurs in pneumonia, it is most likely to take place in the stage of purulent infiltration; but even then the patient may get quite well by the free discharge of the purulent matter. Recovery may take place in any stage of pneumonia. It generally follows red hepatization, and nothing is more extraordinary than the rapidity with which a lung will pass from the state of red hepatization to that of health. The way in which this change occurs is probably this:—a fresh effusion is poured out, which, consisting chiefly of serum, dissolves the plastic material with which the cells and finer air-tubes were previously blocked up; a portion of this is re-absorbed, while the remainder is quickly expectorated, the dilated capillaries gradually resume their natural size, and the lung returns to its normal condition. In the case which I shall presently bring before you, the rapidity of this change was very striking. When recovery takes place in the stage of grey hepatization, it is much more slow; and the same obtains in the stage of purulent infiltration, as I just now observed.

Case LXXXI (vol. xl, p. 130).—Such, then, are the preliminary remarks with which I have thought it expedient to bring under your notice the case of a boy named Everitt, now in Rose ward. He is a fair-complexioned lad, of a strumous diathesis, and his antecedents indicate that he has been much in the hospital; for "when two years of age he had hooping-cough, and from this he suffered greatly the following twelve months."

He "wasted away;" and so severe and trying was this malady, that "he was given up by all the doctors who attended him." After the disease had lasted twelve months, it left him; that is to say, he ceased to hoop, but the cough remained; and from that time to the present he has been a poor, weakly child, always having a cough and spitting, the matters expectorated being usually "thick yellow and greenish phlegm." In February last (1853), he had an attack of scarlatina, for which he was three months in the hospital under my care. The fever was followed by a slight degree of general dropsy, and his urine contained at that time a considerable quantity of blood (free, and also in casts of small diameter), with a few epithelial casts, but with very little desquamated renal epithelium; and it was also slightly albuminous, but probably not more so than would be due to the amount of blood present—all these signs indicating, as you are aware, an irritated and inflamed condition of the kidneys.

The pneumonia in this boy came on in the regular way; indeed, the case affords a good example of the mode in which this disease usually begins. "His present illness," says the report, "commenced on the 19th inst. (October, 1853), when he was taken with running at the nose, shivering (distinct rigors), a *stitch* in the left side, and vomiting." All these symptoms are of nearly a constant occurrence in the development of a well-marked case of pnemonia, except the vomiting, which is rare.

"On the evening of that day the shivering left him, and he became very hot and feverish;" and I may here remark that this heat of skin is looked upon by some physicians as highly characteristic of pneumonia. "On the following day he appeared to be getting worse, his breath began to get very short, and the vomiting and stitch continued, and on the 21st he became an out-patient of the hospital;" but of the nature of the treatment upon which he was put at this time, there is no record in the notes of the case. "All this time his cough, which was usually very troublesome, seemed to get better—he coughed very little, and did not spit up anything—it (the sputum) seemed too thick to get up." From the characters of the sputa we derive, as you know, considerable aid in forming our diagnosis in cases of

pneumonia; and from the patient's own description of facts we oftentimes receive very useful hints. This very circumstance of the sputa "being too thick to get up" would at once lead a person, accustomed to think on this subject, to suspect the existence of inflammation of the lung, as pneumonic sputa are very thick and viscid, and cling to the sides of the vessel in which they are contained, so as not to fall out even when it is inverted. Indeed, so very adhesive is the pneumonic expectoration, that it sticks to the sides of the bronchial tubes, trachea, and larynx, this property being due, no doubt, to its containing a large quantity of the plastic material of which I spoke at the commencement of the lecture; and it is in this thick and viscid mucus that we sometimes find those little "casts" of the finer bronchial tubes, which are almost pathognomonically characteristic of the sputa in pneumonia. Another character peculiar to the matters expectorated in pneumonia is their colour, being tinged by an intimate admixture of more or less of the colouring-matter of the blood, which usually gives them a *rusty* hue. Sometimes, however, this rust-colour is not so distinct; and occasionally, in genuine cases of pneumonia, the sputa have, instead of it, a bilious tint.

After the 21st inst., we have no distinct history of the case until the patient's admission into the hospital on the 25th. "He appeared," say the notes, "to get worse, his breathing became more uneasy, and the fever increased;" and on his admission on the 25th, his condition is thus described:—"A weakly, delicate-looking, fair-complexioned lad, with a flushed face, and considerable shortness of breath; P. 120; R. 50; skin hot, dryish; tongue coated with a whitish fur, moist; no appetite; great thirst; complains of pain in the left side, over the region of the left lung, and of cough, but does not expectorate at all, or if he does, he swallows the sputa, but of this last there is no decided evidence; the left side of the chest, especially at the lower part, hardly expands so much on inspiration as the right." From all these symptoms—the hot skin, the shortness of the breath, the pain localized in one spot on the left side, that side of the chest not expanding on inspiration so much as the other, &c.—one would at once be led to suspect the existence of pneumonia.

By *percussion* and *auscultation,* we obtained further signs which left no doubt as to the nature of the disease. "On percussion there was dulness over the lower part of the left lung posteriorly (the region in which the pain was felt); the dulness was not so complete as that which would be produced by the presence of fluid in the pleural cavity, but had rather the characters which would be due to a nearly solid viscus including some amount of air." We may suppose that though the air-cells were filled with the plastic material, the larger bronchial tubes, passing through the substance of the lung, contained air; and I mention this circumstance particularly, because I wish you to bear in mind that the term "dull," as applied to the sound elicited by percussion, is a *generic* one, and may be divided into several special kinds, each differing from the other in a marked manner, though all of them are essentially *dull.* Thus, there is a remarkable difference between the character of the *dull* percussion sound which is produced by an inflammatory consolidation of lung, and that which is the result of pleuro-pneumonia with the presence of a *small* quantity of fluid in the pleural cavity; and the *dull* sounds, produced by these two conditions respectively differ considerably in their characters from that which is due to the existence of a *large* amount of fluid in the cavity of the chest. "The percussion-sound over the right lung was quite clear."

Another sign, which naturally follows upon this one of percussion, is the effect of the voice on the walls of the chest; if the lung be healthy, vibrations are felt, but if fluid intervene between the lung and the costal pleura, no such vibrations can be distinguished; while if the lung be solid, but the larger bronchial tubes are permeated by air and communicate freely with the trachea, vocal vibrations, sensible to the hand, will still exist, and sometimes in an exaggerated form. In our patient, vocal vibrations "were about equal over both lungs; if any difference, slightly stronger over the left." Here, then, was an additional reason for not attributing the dulness on percussion to the presence of fluid in the pleural sac; for, had that been the case, no vocal vibrations would have been detected. By comparing the vocal vibrations on both sides, we found that they were slightly stronger over the left lung; and this brings to mind a point on

which I wish to dwell for an instant. In some cases of pneumonia the vocal vibrations are most distinct over the diseased lung, in others they are best marked over the sound one, while, in a third set of cases, they are altogether absent over the inflamed organ. This difference in the degree of the vibrations of the thoracic walls, produced by the voice, seems to me to depend on the condition of the bronchial tubes; the more freely the air passes through these tubes, the more distinct will be the vocal vibrations, and, conversely, the more plugged up these tubes are, the less perfectly will the chest vibrate.

On listening to the chest, we found the phenomenon, termed "bronchial breathing," present. I strongly recommend you to take every opportunity of studying this sign, as it is a highly important one. You may get what is very like it by placing your stethoscope over the trachea, when you will hear a tubular, blowing kind of respiration, as of air passing to and fro through a hollow tube. "The *bronchial breathing* was very well marked indeed over the whole of the left lung posteriorly, best marked over the base, less intense over the next two thirds, and but slight, and mixed with vesicular breathing, at the apex. The breathing over the left lung anteriorly was good and vesicular, as it was also over the whole of the right, except at the base, where some large, moist crepitation was audible." "There was increased *vocal resonance* where the bronchial breathing existed," there being that peculiar condition of voice termed "*bronchophony*," a condition which indicates that the voice, generated at the larynx, resounds more perfectly in the ear placed against the wall of the chest than it does in health, and seems as though it were actually formed in the corresponding bronchial tubes.

All these symptoms and physical signs led me to make the following diagnosis:—"That the lower two thirds of the posterior part of the left lung were hepatized, and that there was some condensation of the apex of the same lung; that the apex, though not yet hepatized, was quickly passing from the stage of *active congestion* into that of *red hepatization*." The signs upon which I chiefly relied in forming this diagnosis were, the bronchial breathing and the bronchophony, with the history of the disease and the general symptoms. And here I cannot too

strongly warn you against the danger of paying exclusive attention to physical signs, and thereon founding your diagnosis, for by doing this you will very frequently fall into error. If I had attended to the physical signs alone in this case, I might have come to the conclusion that the disease consisted in *tubercular infiltration* of the lung, inasmuch as this condition will produce exactly the same signs as those to which the effusion of a fibrinous material into the air-cells, leading to the consolidation of the pulmonary tissue, gives rise, viz., dulness on percussion of a like character, bronchial breathing, and bronchophony. To decide whether the state of the lung was dependent on tubercular infiltration, or on the effusion of lymph into the air-cells, I took into consideration the history of the patient; and here the exposure to wet and cold, the shivering, the "stitch" in the side, and the absence of anything like the history of tubercles in himself or in his family, all led to the conclusion that the physical signs were produced by inflammation of the lung. Several cases of solidification of the lung have come before me, the result of inflammation, which had been pronounced to be phthisis, and the patients after a time got quite well. Let me put you on your guard against making such a mistake. In this case we derived no assistance from the characters of the sputa; for there was no expectoration, and this increased the difficulty of diagnosis.

Additional confirmation of our diagnosis in this case was derived from the fact that under treatment the signs of pneumonia began to disappear. On the day after his admission (26th) the pulse and respirations had greatly fallen, the former being 88, the latter 30 in a minute, the sound on percussion was much less dull, and distinct, large, moist crepitation could be heard below the spine of the scapula, though still lower down the breathing was bronchial, but mixed with some returning crepitation; all these signs indicating that the process of solution of the coagulable material in the air-cells and finer tubes had already commenced. On the 27th, evidence of the rapid resolution of the pneumonia existed; the pulse was 98, and the breathing 28, and the condition of the chest is thus recorded:—"Posteriorly, over the apex of the left lung and left supra-spinal fossa, percussion is clear, indeed quite as clear as over the same

regions on the right side; over the base of the left lung the percussion sound is less dull than yesterday, but still duller than that elicited over the corresponding point on the right side," showing that the lung was not then perfectly penetrated by air. "Below the spine of the left scapula, also, the sound on percussion is still slightly dull; in front, percussion is clear over both lungs. There is good, vesicular breathing in the upper two thirds of the left lung behind," where the bronchial breathing had existed two days before; "and as one descends towards the base, slight large and moist crepitation becomes audible; while quite at the base distinct bronchial breathing is heard, with some crepitation on coughing and deep inspiration. The breathing is vesicular and puerile all down the right lung behind; in front the breathing in both lungs is good and vesicular."

The crepitation which we find succeeding bronchial breathing, when accompanied with a diminution in the rate of the pulse and breathing, is one of the best signs of the resolution of pneumonia; but let me caution you against concluding that the crepitation *alone* is a favorable sign, inasmuch as it may be produced by the lung passing into the stage of *purulent infiltration*. You must, therefore, endeavour by every means at your command to satisfy yourselves that this symptom, when present, does not depend on the lung advancing into a further state of degeneracy, which you may generally ascertain by carefully watching the rate of the pulse and respirations.

On the 28th, the patient was convalescent, and the appetite was returning; there was still some dulness over the lower part of the left lung posteriorly, with some bronchial breathing and moist crepitation quite at the base, showing that resolution was not yet perfect.

On the 29th (the eleventh day of the disease), he was going on well, the pulse being 92, and the breathing 24, and the physical signs were good, the bronchial breathing having entirely disappeared. I have not seen him to-day, but have no doubt he is going on extremely well.

In my next lecture, gentlemen, I purpose speaking more at length of the condition of the urine in pneumonia, and of the treatment which it is advisable to adopt in this disease.

LECTURE XIV.

ON CERTAIN ACUTE DISEASES.

ON PNEUMONIA.

GENTLEMEN,—I propose to resume to-day the consideration of the case of William Everitt, the little boy who has been suffering, as most of you are aware, from pneumonia. The diagnosis which we gave in the first instance was "that there was inflammation with consolidation of the inferior two thirds of the left lung behind." That this was the condition of the lung we made out distinctly, and I explained to you in my last lecture the grounds upon which we came to this conclusion. Let me once more impress upon you the importance of bearing in mind the various points which enable one to say of a given case, not only that the existing symptoms are dependent on pneumonia, but also that they do *not* arise from any other morbid state of the lungs—in other words, that you should thoroughly understand what has been termed (although not very happily) *the differential diagnosis of pneumonia.*

The diseases, from which you should be especially careful to distinguish pneumonia are the following:—

1st. Pleurisy, which, as you know, may be of two kinds—viz., *simple,* or *dry,* with or without an exudation of plastic lymph; and *pleurisy with effusion,* which leads pretty soon to the pouring out into the pleural cavity of a *fluid,* which may be either *serous* or *purulent.*

2d. That consolidation of lung which is due to the deposition of tubercles.

3d. That solidification of lung which is dependent on pulmonary apoplexy.

4th. A similar condition resulting from the deposition of cancerous matter in the lung structure.

All these conditions are apt to give rise to signs not very dissimilar from those produced by pneumonia.

In pleurisy, as in pneumonia, we have dulness on percussion over the diseased part; but in the former disease the dulness is more decided in its character than in the latter. In pleurisy, too, as in pneumonia, we have bronchial breathing and bronchophony; or, more properly speaking, a peculiar modification of the voice which is termed *ægophony*, but which may easily be mistaken for bronchophony by an unpractised ear.

The presence of tubercles in the lungs gives rise to quickened respiration, dulness on percussion, bronchial breathing, and increased resonance of voice; and somewhat similar phenomena are likewise produced by *pulmonary apoplexy*, which term simply means an effusion of blood into the bronchial tubes and air-cells of a few or several, more or less contiguous, lobules. From some source of hæmorrhage within the lung or opening into it, blood finds its way into one or more bronchial tubes, and during the act of inspiration is drawn down into the air-cells and finest bronchial ramifications, and thus a consolidation of lung, not unlike that which results from inflammation, is produced. The difference between the consolidation of lung dependent on pneumonia and that which is due to pulmonary apoplexy is, that in the one case the increased density is caused by the exudation of the plastic matter of the blood, with only a small portion of its colouring matter, into the air-cells and finer bronchial tubes, while in the other it results from the infiltration of the whole blood into the same channels.

In like manner, also, when cancerous matter is deposited in the lung (whether in the air-cells or in the areolar tissue around the lobules I am not prepared to say), dulness on percussion and other signs of consolidation are produced; and the dulness in this instance will always be found proportionate to the extent of the morbid growth.

When endeavouring to discriminate between pneumonia and pleurisy, you must keep in view that these two morbid conditions are very frequently associated, constituting what is termed *pleuro-pneumonia;* most cases of pneumonia, indeed, are of this last description, whilst *pleurisy, i. e.*, inflammation of the pleura, whether pulmonary or costal, but especially the

latter, more frequently occurs as a separate and distinct affection.

If then a patient were admitted into the hospital with quickened breathing, pain in the side, the respiratory movements in one lung impeded, having previously had shivering, and having dulness on percussion over the lung in which the respiratory movements were embarrassed, how would you be able to satisfy yourselves that these symptoms depended on pleurisy and not on pneumonia? If the symptoms were due to *simple* pleurisy, the dulness would be but slight, and on putting the ear to the chest a *friction-sound* would be heard. The character of the pain would afford you some assistance: in pleurisy the pain is generally a sharp "stitch," while in pneumonia it is usually described as "dull." The reason of this is, that pleurisy often begins as a muscular affection, having its origin very frequently, I suspect, in rheumatic inflammation of the intercostal muscles, which spreads to the costal pleura. Upon the presence, then, of a friction-sound, upon the absence of marked dulness on percussion, and upon the absence, also, of any sign of crepitation, and upon the presence of vesicular breathing in the whole of the suspected lung, you can form a tolerably accurate diagnosis in favour of simple pleurisy and against pneumonia. But if rapidly followed, as pleurisy often is, by the effusion of a *small* quantity of a serous fluid into the pleural cavity, so that a *slight* layer of liquid intervenes between the pulmonary and costal pleuræ, the diagnosis between pleurisy and pneumonia is by no means so obvious; because, under such circumstances, a marked dulness on percussion is produced over a space corresponding to the extent of the effusion, but the dulness will be found on careful examination to be much more marked and decided than that which would be present in a case of simple pneumonia. In such a case, you would also have decided bronchial breathing; but, on testing the voice, you would find bronchophony, not pure as in simple pneumonia, but in a modified form, and exhibiting that peculiar tremulous, bleating character of the voice, whence it is called *ægophony*.

Again, how are you to determine in a given case that these signs depend, not on pneumonic solidification of the lung, but on pleurisy *with an abundant liquid* effusion? The proofs are

these :—If the symptoms depend on the presence of liquid in the cavity of the pleura, the dulness on percussion will be very complete and decided, and if the hand be placed flat against the thoracic parietes on the affected side, and the patient made to speak, the vibrations of the voice will not be propagated to it. If, on the other hand, the symptoms are due to pneumonia, vocal vibrations will be more or less freely communicated to the hand placed against the wall of the chest, the freedom of propagation being influenced mainly by the permeability of the bronchial passages to air.

When these two signs—marked dulness on percussion, and absence of vocal vibrations—exist together, the evidence which they afford is conclusive as regards the presence of a liquid effusion in the cavity of the pleura. In such a case, too, bronchophony is replaced by ægophony, unless the effusion be very large; and the bronchial breathing has not the same intense character which it would have in the case of a lung consolidated by pneumonia, nor does it convey to the same extent the idea of nearness.

There are two points which I must here impress upon you. The first is, that now and then you meet with a case in which there has been a former attack of pleurisy which has left adhesions. In such a case, you may have vocal vibrations present, yet there may be marked dulness and ægophony. The adhesions are sufficient to propagate the vocal vibrations from the lung to the wall of the chest.

The second point is, that sometimes the voice is feeble, and its vibrations cannot be propagated beyond a certain portion of the bronchial tree, and not at all to the thoracic parietes. This occurs chiefly in women; sometimes in weakly men.

In addition to all these phenomena, there is, generally speaking, something in the history of the case which affords aid in arriving at an accurate diagnosis: thus, pleurisy is more frequently connected with a rheumatic state of the constitution, or *diathesis*, than pneumonia; and in its early stages the symptoms of the former disease are much more severe as regards pain, but much less urgent as regards the affection of the breathing, than those of the latter. There is generally less heat of skin in pleurisy than in pneumonia; and the charac-

teristics of pleurisy, on the whole, approximate those of a (so-called) *sthenic* disease, while the characters of pneumonia are more nearly allied to those of an *asthenic* affection; and patients labouring under inflammation of the pleura bear bleeding better than those who are suffering from inflammation of the lung.

With regard to the differential diagnosis of pneumonia and pulmonary apoplexy, cancer, and tubercle, you must be guided in a great degree by the history of the case. With respect to tubercular deposits, it is important to note the *position* of the dulness on percussion; in tubercular disease this occurs at the apices of the lungs far more frequently than anywhere else; while in pneumonia it is usually found over the base of these organs. The nature of the *expectoration* also affords valuable information; thus, if tubercles be present, but in a crude state, they may give rise to little or no expectoration; or the fluid spat up may be colourless, and exhibit very much the appearance of saliva; or it may be a glairy mucus; or, as the tubercles soften, it may assume more or less the characters of pus; or at a former time or times blood in small or large quantity may have been spat up; but in genuine cases of pneumonia the sputum in the early stages is almost invariably rust-coloured, viscid, and tenacious. Do not, however, lose sight of the fact that in pneumonia of the apex there is very often a total absence of expectoration.

In cancerous disease of the lungs, the history of the case, as I just now observed, comes to our aid; and here, too, there is generally an absence of fever, and the dulness on percussion is usually less extensive than in an ordinary case of pneumonia, inasmuch as the cancerous matter, in consequence of its deposition for the most part in detached masses, does not usually consolidate a large portion of the lung. In such a case, also, there will be absence of the breath-sounds to a greater or less extent, and, associated with this, a material deficiency in the ordinary movements of the thoracic walls will generally be found. In addition to all these, there is the cancerous *cachexia*, as it is called, and that peculiar, enfeebled, more or less anæmic habit of system, which an experienced eye readily detects; while the family history of the patient sometimes affords

important information as to the nature of the malady. I need scarcely add that this remark applies even more strongly to tubercular disease.

In the case of pulmonary apoplexy, we receive considerable assistance from the fact of the occurrence of a pretty copious hæmorrhage by which this state of lung is invariably produced. This morbid condition is most commonly associated with disease of the heart, and is generally caused by something which either interferes with the return of the blood to the left side of the heart, or prevents its free flow through the pulmonary blood-vessels, the consequence of the obstruction being the rupture of some of them, the effusion of blood, and the consolidation of those parts of the lung into which this blood obtains entrance.

Referring once more to the diagnosis between pneumonic and tubercular consolidation of the lung, let me put you on your guard against being led astray by a peculiar form of chronic pneumonia, which I generally call strumous pneumonia. It occurs in children and young persons of strumous constitution, and almost always attacks the apex of one lung; generally there is no expectoration, and if it occur it is not coloured. The lung becomes gradually solid, and as gradually undergoes resolution. The progress of the case affords the best aid to its diagnosis, and a practitioner who is cautious in forming his opinions, and on the look-out for such an occurrence, will seldom go astray.

Having made these introductory remarks, let me now proceed to the consideration of the condition of the urine in pneumonia; and I have here to express my thanks to my friend, Dr. Evans, as far as regards this secretion, for the able manner in which he has worked out the case, thus enabling me to lay before you the constitution of the urine of this patient, on almost every day after his admission into the hospital until his restoration to health.

The composition of the urine in pneumonia is a matter of recent observation. I am anxious to draw your attention to it, because the notes of cases of this disease are incomplete and unsatisfactory in a clinical point of view, unless the condition

of the urine from day to day is carefully recorded. There are certain salts constantly existing in normal urine, which become totally, or almost totally, deficient in this secretion, at the period when hepatization of the lung is complete, and are not fully restored until resolution is established.

To make this subject more intelligible, let me call your attention to some of the leading points connected with the composition of the urine in the state of health.

The urine consists of a large proportion of water, which holds in solution certain solid matter. These solid ingredients are of two kinds—viz., *organic* and *saline;* the former comprising *urea, uric acid,* and certain mystical chemical compounds termed *extractives* or *extractive matters,* which are further distinguished as *water extractive* and *alcohol extractive,* of the exact nature of which we shall some day know something more; the latter comprising the *sulphates of potash* and *soda, alkaline* and *earthy phosphates,* and *chloride of sodium,* with occasionally a greater or less quantity of *chloride of ammonium,* or, as it is generally termed, *hydrochlorate,* or *muriate of ammonia.*

Now in the urine in pneumonia there is a deficiency in all the *saline* ingredients, but the salt whose diminution is most marked is *chloride of sodium* (common salt). This point was first made out by Redtenbacher in Germany, who, upon observations on eighty cases of this disease, clearly established the fact, that during the whole period of hepatization, and, indeed, until resolution is freely established, chloride of sodium is wholly, or almost wholly, absent from the urine.

The presence or absence of this *chloride* in the urine may be ascertained by a chemical test, so simple in its nature, and so readily used, that it is in the power of every one to search for this substance as easily as for albumen. The chemical operation to which I allude is as follows:—about a drachm of the urine should be placed in a test-tube and acidulated with a little nitric acid: to this a few drops of a solution of nitrate of silver should now be added, when, if any chloride be present, a white flocculent precipitate (*chloride of silver*) will occur; and the *quantity* of chloride in the urine may be roughly estimated by noting the bulk which this precipitate occupies, after having

been allowed to subside to the bottom of the test-tube a short time, though, of course, when accurate results are sought for, an altogether different mode of proceeding must be adopted. If, on the other hand, no precipitate takes place on the addition of the solution of nitrate of silver, you may conclude positively and certainly that no chloride is present in the urine. This process is not always perfectly certain, inasmuch as it is invalidated by the occasional presence of chloride of ammonium in the urine, which, like chloride of sodium, gives a similar white flocculent precipitate when treated in this way; but as the presence of chloride of ammonium is a rare occurrence, this test in the large majority of instances answers sufficiently well for ordinary purposes, and the evidence which it affords, when there is a total absence of any precipitate, is quite conclusive as to the entire disappearance of chloride of sodium from the urine.

I will now relate to you what happened with respect to this matter in the case we are considering.

The lad, you will bear in mind, was taken ill on October 19th —on that day he first shivered; on the 21st he was admitted into the hospital; and the diagnosis which was then made, was, as I told you in my last lecture, that the lower two thirds of the posterior part of the left lung were in a state of hepatization, and there was some condensation of the apex of the same lung. He was put under treatment on the 21st, and on that day his urine was tested with nitric acid and nitrate of silver in the way which I just now described. To our great surprise, a copious white precipitate was produced, affording a *primâ facie* indication that chloride of sodium was present, despite of the pneumonic consolidation. This was, however, clearly exceptional, from some cause which we could not ascertain, or it was due to the temporary presence of chloride of ammonium. Upon this point we could gather no precise information, as, unfortunately, no more urine could be obtained on this day to subject to a more exact method of analysis. On the following day, however, which, reckoning from the occurrence of the shivering, was the *eighth* of the disease, a sufficient quantity of urine was obtained to submit to minute analysis, so that we did not depend on the simple mode of testing to which I have already referred. On

this day, while a great portion of the left lung was in a state of simple hepatization, with, perhaps, a little resolution just commencing, scarcely a trace of chloride of sodium existed in the urine; so that it was a reasonable inference that on the preceding day the precipitate, which was produced on the addition of nitric acid and nitrate of silver, was due to the presence of chloride of ammonium; and an analysis of 100 grains of the *solid matters* of the urine on this day gave *organic matters* 97·034 and *fixed salts* 2·966. Now the relative proportion of the organic to the saline constituents of the solid matters of ordinary healthy urine is as from 70 to 75 are to from 30 to 25; or, in round numbers, the composition of 100 grains of the solid matters of healthy urine may be represented as *organic matters* = 75, *fixed salts* = 25. You see, then, what a remarkable diminution had taken place in the amount of the saline constituents of the urine of this patient on the eighth day of the pneumonia—from 25 to barely 3 parts in a hundred!

On the ninth day the physical signs indicated that a general resolution of the inflammation had taken place; and an analysis of the urine showed that this fluid contained merely the slightest trace of chloride of sodium, while 100 grains of its *solid matters* were composed of 97·938 of *organic constituents*, and 2·062 of *fixed salts*—even less than the preceding day.

On the tenth day convalescence had considerably advanced; the chloride of sodium was, as yet, *entirely* absent from the urine, but the proportion between the *saline* and *organic* constituents had somewhat altered, the former having slightly increased, the numbers being, *organic matters* = 97·702, *fixed salts* = 2·298.

The urine of the twelfth and that of the thirteenth days of the disease were unfortunately mixed together, so that an analysis of each separately was, of course, impossible. By this time the patient had completely thrown off all traces of the pneumonia; air freely permeated every part of the lung, consolidation had disappeared, and he was up and about, and hungry, but still on the same diet (and this is a very important point) upon which he was first put when brought into the hospital, viz., the hospital *milk diet* and *beef-tea*. We now found that a remarkable change in the relative proportion of the or-

ganic to the saline constituents of the urine had suddenly taken place; the organic matters, instead of being 97 per cent. of the solids, had fallen to 72·814, while the fixed salts had risen from rather more than 2 per cent. up to 27·186; showing how very rapidly this change must have occurred, the patient having still the same diet as on the five preceding days, so as completely to exclude the idea of its being due to the mode of feeding. On this same day the chloride of sodium had returned to the urine, but in rather less than its normal amount, viz., 4·321 in 1000 grains of urine.

On the fourteenth and fifteenth days analyses of the urine gave similar results, and chloride of sodium was present on the fourteenth, in rather less than its normal proportion; but on the fifteenth, the patient having been put on *middle diet* the previous day, it had increased from 4 to 7·180 in 1000 grains of urine.

All these are very curious facts if taken alone, as carefully observed in this one case; but if you refer to the thirtieth volume of 'Medico-Chirurgical Transactions,' you will find a very interesting paper by Dr. Lionel Beale, in which the same points are fully established as founded on observations which were made on several cases. The question which these facts naturally suggest, and a most important one it is for pathology, is, "What becomes of the chloride of sodium, under these circumstances, if it does not pass out in the urine?" The object of carrying off these saline substances in the urine is probably to convey away certain effete matters derived from the waste of the tissues, or else introduced with the food; but during the first five days this patient was under treatment, he was daily taking a certain amount of chloride of sodium in his food (*milk diet and beef-tea*), which did not find its way out of the body in the urine. Dr. Beale has shown that a large quantity of chloride of sodium under these circumstances accumulates in the *sputa* and in the *inflamed portion of the lung*, while the proportion of this salt in the *serum of the blood* falls below its normal standard. From this it would appear that there exists in pneumonia some attraction between the inflamed lung and chloride of sodium; for if this substance be in excess in the inflamed lung, while at the same time it is deficient in the blood and

urine, the inference is immediately suggested that, in some way or other, it becomes attracted to the inflamed pulmonary tissue. Upon the precise object of this process it is difficult to speculate; but we must, for the present at least, take it as a fact, and endeavour to ascertain whether a similar state of things occurs in acute inflammations of other organs, or in bronchitis, &c. Any of you, who have ever had a severe attack of coryza, must have noticed that the nasal secretions have a very salt taste, as if, in this affection, chloride of sodium were attracted in undue quantity to the inflamed surface.

Another question which suggests itself here is—Why doe the chloride of sodium return so quickly into the circulation and urine? For no sooner does the attracting force set up by the inflamed lung cease to operate, than the chloride finds its way out of the system through the ordinary channel.

I may here again allude to a point which I before mentioned, viz., that a diminution takes place in all the other fixed salts of the urine as well as in the chloride of sodium. The latter, it is true, disappears altogether; but the sulphates and phosphates become remarkably diminished; for, as I just now told you, analytical examinations of the urine of this patient on the eighth, ninth, and tenth days of the disease gave 2·966, 2·062, and 2·298 respectively, as the amount of fixed saline matter in 100 grains of the dried solids of the urine, instead of the normal proportion of 25 per cent.

To enable you more clearly to comprehend these curious and interesting facts with reference to the urine, which I have just mentioned, I have here a table drawn up by Dr. Conway Evans, showing the composition of the urine of this patient on the eighth, ninth, tenth, twelfth, and thirteenth, fourteenth, fifteenth, seventeenth, and twenty-first days of the disease. By carefully studying these analyses you will notice several other interesting points besides those to which I have alluded, and which I regret time does not permit me to enter into. With respect to the table, let me add, that the first column shows the composition of 1000 grains of urine; the second column shows the composition of 100 grains of the solid constituents of the urine; or, in other words, the per-centage of the various components of the solid ingredients; while the third column repre-

	Anal. I. Eighth day of Disease. Sp. Gr. 1013.			Anal. II. Ninth Day of Disease. Sp. Gr. 1013.			Anal. III. Tenth Day of Disease. Sp. Gr. 1012.			Anal. IV. Twelfth and Thirteenth Day of Disease. Sp. Gr. 1010.		
	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.
Water . . .	944·277			957·875			969·028			972·825		
Solid Matter . .	55·723			42·125			30·972			27·175		
Organic Matter . .	54·070	97·034		41·256	97·938		30·260	97·702		19·787	72·814	
Fixed Salts . .	1·653	2·966		·869	2·062		·712	2·298		7·388	27·186	
Urea, Extractive, & Ammon. Salts .	53·302	95·650		38·927	92·410		27·428	88·559		18·733	68·936	
Uric Acid . .	·768	1·384		2·329	5·528		2·832	9·143		1·054	3·878	
Chloride of Sodium .	A slight trace.	A trace.	A trace.	A slight trace.	A trace.	A trace.	Not a trace.	None.	None.	4·321	15·900	58·486
Sulphuric Acid . .	·662	1·186	40·048	·208	·493	23·935	·308	·994	43·258	1·313	4·831	17·772
Phosphoric Acid .	·512	·918	30·973	·244	·579	28·078	·110	·355	15·449	1·115	4·103	15·092

	Anal. V. Fourteenth Day of Disease Sp. Gr. 1009.			Anal. VI. Fifteenth Day of Disease. Sp. Gr. 1012.			Anal. VII. Seventeenth Day of Disease. Sp. Gr. 1020.			Anal. VIII. Twenty-first Day of Disease. Sp. Gr. 1023.		
	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.
Water . . .	978·942			964·617			950·694			943·528		
Solid Matter . .	21·058			35·383			49·306			56·472		
Organic Matter . .	13·444	63·843		24·408	68·983		38·640	78·368		43·462	76·962	
Fixed Salts . .	7·614	36·157		10 975	31·017		10·666	21·632		13·010	23·038	
Urea, Extractive, & Ammon. Salts . }	12·851	61·027		23 379	66·075		37·397	75·848		42·810	75·808	
Uric Acid . . .	·593	2·816		1·029	2·908		1·243	2·520		·652	1·154	
Chloride of Sodium .	4·877	23·159	64·053	7·180	20·292	65·330	3·429	6·954	32·148	5·995	10·615	46·079
Sulphuric Acid . .	·757	3·594	9·942	1·381	3·903	12·583	1·774	3·780	16·632	1·399	2·477	10·753
Phosphoric Acid .	·522	2·478	6·855	1·487	4·202	13·548	1·771	3·774	16·604	2·891	5·101	22·221

sents the amount of chloride of sodium, and of sulphuric and phosphoric acids, contained in 100 grains of the fixed saline constituents.

One conclusion may certainly be inferred from these curious chemical details, namely, that the disease of which we are speaking involves profound changes in the chemistry of life—in the interchanges between the blood and the tissues, and in the chemical constitution of the blood itself. And not only so, but the process of *recovery* from the disease involves analogous chemical changes in an opposite direction, showing that there is in the human system a wonderful power of restoring the injuries inflicted by disease. When we shall have obtained a clearer insight into the recondite processes by which repair is effected in the animal body, we shall be in a better position to assign to our experimental interferences their proper position either as helps or hindrances to this inherent *vis medicatrix*.

Let me now call your attention to the treatment which was pursued in this case. The lad first applied to the hospital on October 21st, and he continued as an out-patient until the 25th; and during this period he was treated with quarter-grain doses of tartar emetic, administered every four hours, which did not prevent the progress to hepatization. On the evening of his admission (October 25th) his pulse was 120, and his respirations were 48, in a minute. The treatment adopted consisted in the exhibition of three-drachm doses of the solution of acetate of ammonia every two hours, with two drachms of wine every four hours, a moderate supply of beef-tea, milk diet, and the application of turpentine stupes over the left side of the chest.

On the following day, the pulse was 100, and the respirations were 36; on the 27th, the pulse was 76, and the breathing 28 (the numbers throughout are those of the evening), and general resolution of the inflammation had taken place; air was admitted freely into the lung, and the dulness had become much less, both in extent and intensity. From this day the improvement was very rapid; the pulse very quickly returned to its normal condition; and on the fourth day of the treatment (the tenth from the occurrence of the shivering) the patient was pronounced

convalescent. On November 1st, the diet was changed from one consisting of milk, bread, and beef-tea, to animal and vegetable food in the ordinary way.

The object of the treatment pursued in this case, and which, with slight modification, I adopt in all others, is to imitate as far as possible the course which nature pursues in this disease. If you watch the changes which a patient in pneumonia undergoes in his progress towards convalescence, you will find that about the eighth, ninth, or tenth day of the disease, or from the tenth to the twelfth day, more or less sweating almost always takes place. Sometimes the sweating occurs freely, and it is then considered *critical*. Sometimes, too, a *critical* discharge of purulent matter from the lung takes place by expectoration; now and then the kidneys about this time secrete much more urine than previously; and occasionally, though certainly very rarely, a *critical* diarrhœa occurs. Always chloride of sodium begins to escape from the system through the urine, from which, during the height of the disease, it had been excluded. All this looks as if a *something*, which caused the morbid change in the lung, were eliminated from the system in one or more of the secretions; and, therefore, to promote these should be the aim of our treatment.

It may be laid down, I think, as a general rule that large evacuations by sweating may be employed more freely and with less disadvantage to patients than by any other secretion; and after a patient with pneumonia has perspired freely, there almost always occurs a marked change for the better. In some diseases profuse sweating takes place at an early period, as, for example, in acute rheumatism and gout; in such cases it is evidently set up for conservative purposes, and to check it is highly unwise, unless it seems to be running the patient down.

A large number of cases of pneumonia are of rheumatic origin, and occur, as I told you in my last lecture, in rheumatic subjects. Indeed, in rheumatic fever many cases of pneumonia begin, while some cases of inflammation of the lung pass on and end in an attack of acute rheumatism. And another reason why you should especially promote the sweating process in such instances is because in rheumatic fever and in gout the morbid

matters, whatever they be, appear, in great measure at least, to be carried off by the skin.

Bear in mind, as a reason against adopting a violent course of treatment, that pneumonia has, on the whole, a decided tendency to get well of itself. I have never had the courage, and I don't think I ever shall, to let a patient with pneumonia alone, and trust to the *expectant system*, as it is called; and I am not acquainted with any satisfactory observations which have been made on this point; but there can be little doubt, I think, that the general tendency of the disease is towards recovery.

From these reasons, then, you will readily see that there is no necessity for having recourse to violent antiphlogistic (so-called) measures in cases of pneumonia; moreover, I tell you that in the course of more than thirty years' experience I have tried all ways—calomel and opium—bleeding—tartar emetic—and the various others which have been proposed—and if I had found that under any one of these plans no death, or even only a very few deaths, had occurred, to that mode of treatment I should have steadfastly adhered; but I found that under each one of these a great number of deaths occurred, and that it frequently happened to me to be called in to patients who had been bled several times, and yet to find the lung in a decided state of hepatization, although the bleeding measures had been adopted at the earliest moment. In the very cases before us the patient had been taking tartar emetic four days, and during this time the hepatization had steadily increased; but as soon as this mode of treatment was altered for a milder plan, the depressing influence of the tartar emetic removed, and sweating promoted, resolution of the inflammation took place with great rapidity.

Among all the cases of pneumonia which I have treated with tartar emetic, I found that none did so well as those in which a toleration of the medicine was established *early*—where it got into the system, but produced no sensible effect, or merely sweating. But the cases in which this remedy caused vomiting, and, more especially, those in which it purged, by no means turned out so satisfactorily as those in which it acted as a diaphoretic only.

What has astonished me in most of the instances of this disease that I have treated or seen treated in the manner which I now recommend to your notice, is the great rapidity with which all traces of the affection are thrown off, and the shortness of the period which elapses before the patient is restored to his usual state of health.

LECTURE XV.

ON CERTAIN ACUTE DISEASES.

PNEUMONIA.

Let me, to-day, Gentlemen, conclude the observations which I made in two former lectures this session on the subject of pneumonia, by calling your attention to two cases of this disease recently in the hospital. One of these having proved fatal, I avail myself of the opportunity of bringing before you, in a more special manner than I have hitherto been able to do, the circumstances which tend to promote a fatal or a favorable termination in any given case of this disease.

You know that I have often insisted upon the importance of paying special attention to fatal cases. Why does any man die of a curable disease? As long as this question can be asked, the *Ars Medica* is in a doubtful position; and it is far better to look candidly and honestly into all the particulars which belong to a fatal case, than to flatter ourselves with the more agreeable contemplation of those which have recovered.

In all fatal cases you should review your practice, with the object of endeavouring to ascertain whether the treatment which has been adopted has had any share in contributing to the fatal result, whether there has been any neglect on the part of the attendants, or whether, from some peculiarity of constitution, or from the age of the patient, or some other circumstance, certain conditions have existed which have tended more to an unfavorable than to a favorable issue. All these investigations are of great practical utility, inasmuch as they enable the

medical man to form a sober judgment of the plan of treatment which he has pursued, and at the same time, if properly conducted, they afford him a greater insight into the nature of the disease, and enable him to speak more decidedly in any subsequent case as to the final issue. And when you get into practice for yourselves, you will find that there is nothing more important than that you should be able to determine satisfactorily whether your *prognosis* shall be favorable or unfavorable, as you will always find your patient surrounded by anxious friends, who look to the doctor almost as if in his hands "lay the issues of life and death."

With these prefatory remarks, then, let me proceed at once to the consideration of the fatal case of pneumonia which has occurred so lately under my care in the hospital.

Case LXXXII (vol. xli, p. 165).—The patient was a man named Charles Johnson, aged 48, who was admitted into Fisk ward on January 16th, 1854; and the date of the disease was from January 12th, *i. e.*, four days before he entered the hospital. I do not propose going into any lengthened detail of the symptoms in this case, feeling confident that, from all I have told you in my former lectures on this subject, you must all be quite *au fait* with respect to the signs of pneumonia. But let me remark, that in keeping notes of cases of this affection, you should endeavour to ascertain as accurately as possible the date of the commencement of the complaint, for this point is of great importance with reference to the clinical history of pneumonia. What symptom, you will ask me, indicates the commencement of the disease? This question may be answered in a word—*rigors*. They do not always occur distinctly, and sometimes their occurrence is not noticed by the patient; but when they can be fixed to a date, they form the starting-point for the disease. This man had distinct rigors on January 12th, and we consequently take this day as the starting-point of the pneumonia. He belonged to a profession which is not perhaps the most conducive to health, for "he gained his livelihood by tumbling about the streets." Though not distinctly intemperate, according to his own account, his habits were, no doubt, not of the most regular kind; and "in consequence of the peculiar nature of his calling, he was continually exposed to wet

and cold," conditions exceedingly liable to induce affections of the chest. "His illness commenced," as I just now said, "on January 12th, when, after having had his feet wet for some hours, he was seized with rigors and a feeling of uneasiness in the chest. This was so severe as to prevent him from following his usual occupation, and on the 16th he began to suffer from pain in the left side of the chest, and shortness of breath. These symptoms increasing in severity, on the evening of that day he was brought into the hospital, when his pulse was 126, and his breathing 32 in a minute, his skin hot, his tongue furred, and he complained of pain in the left side of the chest." In short, he had all the symptoms of pleuro-pneumonia of the left side, there being bronchial breathing and bronchophony, marked dulness on percussion, and absence of vocal vibrations, showing the existence of a slight pleuritic effusion.

He was immediately put under the plan of treatment which you know I have of late been accustomed to employ in all cases of pneumonia,—viz., that which has a tendency to promote sweating, and which simply aims at imitating Nature in the course which she adopts in getting rid of the disease; for it appears to be by an augmentation in the secretions of the skin and of other organs, that the morbid materials, which, by their accumulation in the system, seem to irritate the lung and to produce, or at all events to keep up, the disease, are eliminated in the most beneficial way.

An important feature in this plan of treatment is to counteract as far as possible every influence of a depressing nature; and *though the exhibition of stimulants does not form a necessary part of it*, still the aim should be to uphold the patient's strength. This is best done by providing for him frequent supplies of good beef-tea or other animal broth. Solid food you cannot give in these cases, for not only is it digested with great difficulty, but also the want of appetite prevents its being taken; but, by small quantities of beef-tea frequently supplied, a large amount of easily appropriated nutriment may be administered in the course of twenty-four hours. When the vital powers are clearly depressed, with a pulse *increasing in quickness*, or when the patient has been previously accustomed to live well, or has been in the habit of indulging in the use of alcoholic drinks,—then wine or brandy, or, what is really the proper

way of expressing it, *alcoholic food*, must be exhibited. Our patient was in a depressed state on his admission, and there were also strong suspicions of his having been to a certain extent intemperate in his habits, and on these accounts he was put, at once, upon half an ounce of wine every four hours. In addition to this, he was ordered six drachms of the *liquor ammoniæ acetatis* every three hours, turpentine stupes to the chest, with a free supply of beef-tea and milk. The diagnosis which was formed was this :—"There is a great patch of pneumonia at the lower part of the side and front of the left lung, having a tendency to spread, with, probably, some degree of pleurisy."

On the sixth and seventh days of the disease — and I may here remark that, in keeping notes of cases of pneumonia, it is exceedingly convenient for clinical purposes to note not only the day of the month, but also *the day of the disease*, a point which both my clinical clerks, I am very glad to find, have most properly attended to—on the sixth and seventh days of the pneumonia there was a marked improvement both in the pulse and respiration, on the sixth day the pulse being 104 and the respirations 32, and on the seventh the numbers being 100 and 40, while on the fourth day they were 126 and 32, and on the fifth 112 and 40 respectively. All this looked well, and I was beginning to entertain hopes that the patient would recover, although the case was undoubtedly a very severe one. On the eighth day, however, matters seemed to take a very unfavorable course, and I wish to call your attention to the circumstances under which this change took place. The report says, "He slept better last night, but he does not look nearly so well to-day; his face is dusky and anxious, and he lies with his mouth and eyes half open; he has great difficulty in expelling the viscid sputa from his mouth; pulse 116, very feeble; respirations 30." There were now, also, signs that the disease, which at first was situated in the lower third of the left lung, was rather rapidly spreading in the upward direction; and, in consequence of the feeble character of the pulse, he was ordered half an ounce of brandy every hour, with a draught every third hour containing five grains of sesquicarbonate of ammonia with half a drachm of chloric ether. Now the point to which I wish particularly to direct your attention here is, that the unfavorable

change immediately succeeded a good night's sleep; and I have no doubt that it arose out of that lengthened sleep. I have frequently seen cases in which a long-continued sleep, occurring when the system was much depressed from any cause, has left the patient much worse; and for this reason, that the continued sleep deprives the patient of the due amount of nourishment and support which should be supplied him. This often happens in typhus fever; the patient sleeps five or six hours continuously, and his friends or the nurse imagine that the best thing that can be done is to allow him to sleep on: at the end of this time he wakes up very much exhausted, and with his pulse weaker and more rapid than before he went to sleep. As a general rule, you should not, in my judgment, allow patients in depressed states of the system to sleep longer than two or three hours at a time, often not longer than an hour, but they should be awakened gently at specified times and proper nourishment supplied them. Nor will this proceeding prevent them from going to sleep again; on the contrary, patients so treated, generally speaking, fall off to sleep immediately after they have taken their nourishment, and the sleep so obtained is sound and refreshing.

I remember (though no account of the circumstance is recorded in the notes of the case) that there was good reason for believing that this patient was neglected one night by the night-nurse, and, if my memory does not deceive me, this occurrence took place on the night of the eighth day of the disease. I should also tell you that on the sixth and seventh days, although a general improvement took place, the patient suffered much from diarrhœa, which we were obliged to check by astringents; but this had entirely ceased on the eighth day. From the ninth day the patient declined; on this day the pulse was 120, and respirations were 34 a minute, and the physical signs indicated a decided increase of the pneumonia, the whole of the left lung appearing to be involved in the disease. On the tenth day the patient was reported to be sinking, and on the morning of the eleventh he died.

In this case observations were made on the composition of the urine and sputa, and it was found that the former contained a very small, but decided. quantity of albumen, which

with certain granular *casts* of the uriniferous tubes indicated incipient chronic disease of the kidneys; and this circumstance, of course, tended to render the prognosis still more unfavorable. On the fifth day of the disease the urine contained only a slight trace of fixed chloride, and on the sixth day the report was the same; but on the seventh day this excretion contained neither chlorides nor sulphates, but in 1000 grains of urine there were nearly four grains (3·962) of phosphoric acid; while in 100 grains of the solid matters of the urine there were 7·72 of this acid, and in 100 grains of the fixed salts 60·757. This excessive amount of phosphates in the urine of this day is a very curious feature in the case, and probably increased the nervous depression.

The observations of Redtenbacher with respect to chloride of sodium in this disease were confined to the urine; but the investigations of Dr. Beale show that, while this salt vanishes from this fluid, it makes its appearance in considerable quantity in the sputa. In this case, on the sixth day of the disease, 100 parts of the solid matters of the sputa contained 8·44 of chloride of sodium, while 100 grains of the fixed salts contained 48·243 of this substance. On the seventh day, 100 grains of the solids of the sputa contained 14·544 of chloride of sodium, while 100 grains of the fixed salts contained 58·824; and on the eighth day, in 100 parts of the solid matters of the sputa there were 10·929 of this salt, while in 100 parts of the fixed salts there were 67·35.

By carefully reviewing these tabulated results of the analyses of the sputa and urine, which were made by my friend Dr. Evans, you will readily understand the interesting points in the composition of these fluids which I have just mentioned. The analyses of the *sputa* are those of the sixth, seventh, and eighth days of the disease; those of the *urine* are of the seventh and eighth days. The first column of each table represents the composition of 1000 grains of *sputum* or *urine* respectively; the second column exhibits the composition of 100 grains of the *solid matters* of either; while the third column shows the proportion of chloride of sodium, sulphuric acid, and phosphoric acid entering into the composition of 100 grains of the *fixed salts* in each.

	Anal. I. Sputum. Sixth Day of Disease.			Anal. II. Sputum. Seventh Day of Disease.			Anal. III. Sputum. Eighth Day of Disease.		
	In 1000 Grains of Sputum.	In 100 Grains of Solid Matters.	In 100 Grains of Fixed Salts.	In 1000 Grains of Sputum.	In 100 Grains of Solid Matters.	In 100 Grains of Fixed Salts.	In 1000 Grains of Sputum.	In 100 Grains of Solid Matters.	In 100 Grains of Fixed Salts.
Water	961·923			952·676			950·739		
Solid Matter . . .	38·077			47·324			49·261		
Organic Matter . .	31·415	82·504		35·623	75·274		41·267	83.772	
Fixed Salts	6·662	17·496		11·701	24·726		7·994	16·228	
Chloride of Sodium .	3·214	8·440	48·243	6·883	14·544	58·824	5·384	10·929	67·350
Sulphuric Acid . . .	1·739	4·567	26·103	1·907	4·029	16·297	·769	1·561	9·620
Phosphoric Acid . .	·972	2·552	14·590	·956	2·020	8·170	·509	1·033	6·367

	Anal. IV. Urine. Seventh Day of Disease. Sp. Gr. 1017·2.		
	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.
Water	948·683		
Solid Matter	51·317		
Organic Matter	44·796	87·293	
Fixed Salts	6·521	12·707	
Uric Acid	1·130	2·201	
Urea, Albumen, Extractive, and Ammoniacal Salts	43·666	85·092	
Chloride of Sodium	None.	None.	None.
Sulphuric Acid	None.	None.	None.
Phosphoric Acid	3·962	7·720	60·757

	Anal. V. Urine. Eighth Day of Disease. Sp. Gr. 1012·6.		
	In 1000 Grains of Urine.	In 100 Grains of Solid Matter.	In 100 Grains of Fixed Salts.
Water	964·166		
Solid Matter	35·834		
Organic Matter	30·267	84·465	
Fixed Salts	5·567	15·535	
Uric Acid	1·000	2·790	
Urea	11·001	30·699	
Extractive, Albumen, and Ammoniacal Salts	18·266	50·976	
Chloride of Sodium	A trace.	A trace.	A trace.
Sulphuric Acid	2·296	6·407	41·242
Phosphoric Acid	1·142	3·168	20·513

I very much wish that some of you would take up this subject of the exact chemistry of the *excreta* in the various stages of pneumonia, both towards a destructive as well as a reparative result. It will be only by such researches that we shall arrive at more certain views of the pathology of this disease, and through it of other acute parenchymatous inflammations.

In one or two cases of pneumonia the sputa have been found to contain grape-sugar in greater or less quantity; and this fact is highly interesting, both as bearing on the pathology of the disease, and also, in a general way, on the office of the respiratory organs in consuming the saccharine matter which is formed in the system. But in this case, though the sputa of the sixth, seventh, and eighth days of the pneumonia were each carefully examined with the tartrate of copper and potash, the sulphate of copper and potash, and the fermentation tests, no unequivocal indications of the presence of sugar could be detected.

The *post-mortem* examination of this patient showed that the left lung was solidified throughout its entire extent, a condition quite sufficient to account for the fatal character of the disease; for one of the most important circumstances, I believe, which influences the result in pneumonia is the amount of lung involved. This is one of the great points to be attended to in tabulating cases of this disease for statistical purposes; and it is one of those the neglect of which has rendered almost all the statistical observations on this malady of no worth, because the authors have contented themselves with simply noting cases as pneumonia, without specifying accurately the extent of lung inflamed. You may remember that in a former lecture I compared cases of pneumonia with those of severe burns. There is a difference among practical men, as most of you are, no doubt, aware, as to the mode of treating severe burns, some advocating a plan wholly antiphlogistic, and others recommending the very opposite of this, while a third party adopt the mean between these two extremes. Now it would obviously be not only useless, but even absurd, to compare the results of these different methods of treatment, without at the same time taking into consideration the extent and severity of the burn in each particular instance; for every one knows, that a burn, involving one fourth of the entire surface of the body, is far more likely to

prove fatal than one affecting only one eighth of that surface. So in cases of pneumonia, the greater the extent of lung inflamed, the more difficult will it be to overcome the disease, whatever mode of treatment be adopted. Each plan will be the more successful, the smaller the amount of lung involved. Fortunately, in the generality of cases of pneumonia, not more than a third of one lung is in a state of inflammation. But in the case under consideration, not only was the whole of the left lung solid, but a considerable portion of it had advanced into the stage of purulent infiltration, a purulent fluid filling up the air-cells and finer bronchial tubes; and if this patient had lived a little longer, portions of this lung would, no doubt, have broken down.

The kidneys were in an early stage of chronic disease, the epithelium generally being opaque and crumbling, and many of the uriniferous tubes stripped of their epithelial lining, while in the cortical substance there were several cysts visible to the naked eye. The liver was in an early stage of cirrhosis; and there was some degree of morbid opacity of the mitral valve. This morbid condition of the liver and heart exercised an unfavorable influence in the case; but what tended principally to the fatal result was the disease of the kidneys, which there is every reason for supposing did not eliminate effete matters from the system, as they usually do in the state of health.

Case LXXXIII (vol. xlii, p. 189). — Let me now speak of the case of pneumonia which recovered. It was that of William Davenny, aged twenty, a young man who was in the hospital about the same time as the last patient. This man, though young, was of very intemperate habits, the account of him being this:—"He was born in London, and has lived in town nearly all his life, and is a shoemaker by trade; his habits are very dissipated, and he has made it a constant practice to get drunk whenever he has had an opportunity, ever since he was very young. He has never had any illness before the present. On the evening of January 17th, 1854, he went to a public-house to spend the evening, and drank beer till he was nearly drunk; he then played at skittles till very late, and got very hot; having finished, he walked home in the rain, his clothes

became quite wet through, and he went to bed without changing his wet shirt. About three o'clock in the morning he was awoke by a severe pain in the left side of the chest and great difficulty of breathing. He began to cough, but there was no expectoration. He felt very cold, but did not shiver; and an hour or two subsequently he became very hot, and sweated a good deal. On the following day, January 18th, he kept his bed, and had alternate shiverings and sweats, and entirely lost his appetite; and from this time the pain in the side and the shortness of breath continued to get worse daily, until his admission into Sutherland ward on the 21st January, 1854." Here, then, we may fairly take January 18th as the date of the commencement of the pneumonia; and, therefore, the day of his admission would be the fourth of the disease. "He was able to walk to the hospital," continues the report; "his skin is very hot and dry, his tongue is covered with a white fur; he coughs much, but the sputa are not very tenacious: and when he attempts to take a deep inspiration, a pain catches him in the left side of his chest." We then ascertained that there were all the signs of pneumonia of the lower lobe of the left lung, and that the inflammation had spread through the substance of this lobe, for there were all the indications of solidification in front as well as behind. Free counter-irritation was at once applied to the chest by means of turpentine stupes, and he was ordered six-drachm doses of the *liquor ammoniæ acetatis* every three hours, with milk diet and beef-tea, but no wine was allowed.

The next day the pulse was 120, and the respirations were 48 a minute, and he was now ordered half an ounce of wine every third hour. On the sixth day of the disease he began to sweat, and it is noted in the report of the case, that "he has been in a constant sweat all day; pulse 104, respirations 32." On the seventh day he still sweated copiously; his pulse was 76, and his breathing 24. On the sixth and seventh days there was well-marked large crepitation, taking the place of the bronchial breathing and bronchophony, which had previously existed, and from this time the patient steadily progressed towards recovery. On the eighth day of the disease the pulse and breathing were 76 and 28 respectively; on the ninth day the numbers were 68 and 28; on the tenth day they were 84 and 28; on the

eleventh, 68 and 24; and on the twelfth, 72 and 20. With this gradual diminution in the frequency of the pulse and respirations there was a marked improvement in the physical signs—the large crepitation gave way to pure breathing, and the dulness on percussion disappeared. At one time there was a slight amount of pleuritic effusion, but this speedily became absorbed under the influence of iodine paint. On the sixteenth day of the disease the report is as follows:—"Feels quite well; there is still a little dulness on percussion over the base of the left lung behind, but the breathing is quite clear and vesicular." On the seventeenth day from the commencement of the attack this patient was discharged.

It is very possible in these cases that the lung remains in a swollen or œdematous condition for some time after the signs of pneumonia have disappeared, for it oftens happens that several days will elapse after the breathing has become perfectly pure before the dulness on percussion and increased vocal vibration will have entirely vanished.

In this case a marked improvement took place on the seventh day of the disease; indeed, there was something very like a *critical* sweat. Observations, too, were made on the urine, though, I am sorry to say, they were not quite so regular and accurate as in the other case. On the fifth day the urine contained no chloride of sodium, but on the tenth day that salt had returned in great abundance. Nothing can be more curious than this fact, which seems to be a constant occurrence in pneumonia; so much so, indeed, that the old doctors (and now the modern ones), who were in the habit of examining the *evening and morning state,* could, had they been aware of this circumstance, and known, of course, that the case was one of inflammation of the lung, have predicated almost with certainty the condition of the patient—whether the disease was progressing favorably or otherwise.

Let us now briefly consider why one of these patients recovered while the other died, or, in other words, the causes which tended to bring about the fatal termination in the one case, and the favorable in the other.

The circumstances which materially influenced the fatal result in the first case were, 1st, the extent of the disease;

2d, the age of the patient; and 3d, the state of the general constitution. In addition to these, the patient was one of those individuals who have undergone a great amount of wear and tear; and, moreover, his kidneys, heart, and liver were all damaged.

In the second case, although the lung was more extensively affected than ordinarily, the extent of pneumonia was less than in the first, and there was no disease of any other important organ. In both the treatment was essentially the same; but the younger patient was, perhaps, better attended to than the older one, aad was not quite in so depressed a condition.

With respect to the modes of termination of pneumonia generally, let me say a few words. When recovery takes place in this disease there is always, I believe, some kind of *critical* evacuation, either by sweats, or by urine, or by the free discharge of a purulent fluid from the bronchial tubes, or by pulmonary abscess. Some physicians look upon the herpetic eruption, which sometimes appears upon the lips in this disease, as of a *critical* nature; but I am disposed to think that it is rather a symptom of the disease, because it almost always occurs very early. Still, when you meet with it, you may regard it, I think, as a favorable sign; for most of the cases in which I have seen it have done well.

One can say very little with respect to the intimate nature of these phenomena as spontaneous efforts of the constitution to throw off the disease; but we find this so often the case in other maladies — acute rheumatism and gout, for example—that I think in our treatment we should adopt that method which would give the best aid to this object. I am not prepared to hold the seventh, fourteenth, and twenty-first days as *critical*, as was the custom of the old physicians, but am contented with believing that there is a tendency in the disease to terminate on or near these days. Looking to the recorded cases of this disease, a *critical* evacuation by the urine is by far the most common; from the sixth to the tenth day this excretion being generally considerably increased, and having a tendency to deposit lateritious sediments. The *critical* evacuation next in order of frequency is that by sweating; and if treatment were adopted to favour the secretion of the skin, this would be far

more common, I believe, than has hitherto been observed. A *critical* evacuation by the free discharge of purulent fluid from the bronchial tubes is not common, but I have seen it so profuse as to give rise to the supposition that an abscess had formed in the lung, and had emptied itself through a bronchial tube, when such was really not the case.

When death occurs in pneumonia, it is either by the exhaustion consequent upon a very severe febrile disease; by breathlessness—apnœa—from the large amount of lung inflamed; by gangrene; or by abscess; but by far the most common mode of death is that by exhaustion, as occurred in the first of the cases we have just been considering.

There are certain circumstances which influence the prognosis in this disease. These you must carefully consider before you pronounce an opinion as to what will be the probable issue of any given case. The patient's life may be a highly important one, and his friends will expect you, as his medical attendant, to state decidedly (but you will do so cautiously) what you believe will be the most probable result of the illness. Now the circumstances which chiefly influence the prognosis are—first, the age of the patient; second, the extent of the disease; and from these two alone you may form a pretty accurate conclusion. But you should also pay due attention to the third point, which is the patient's general constitutional condition.

It has been fairly made out that there are two periods of life in which pneumonia is very fatal; these are from early infancy to the age of five years, and the advanced periods—from seventy years and upwards. The great fatality of pneumonia in very young children and infants is explained partly by the more feeble powers of vital resistance at that early period of life, and partly by the fact that at this age it is seldom uncomplicated, and is almost always combined with bronchitis; and there can be no doubt that bronchitis in itself is at all ages a much more fatal disease than pneumonia.

According to French statistics, a death from pneumonia was rare between the ages of five and fifteen. From fifteen to thirty, in 116 cases, there were eight deaths; from thirty to forty, the number of fatal cases amounted to one seventh of the whole; from forty to fifty they were one sixth; from fifty to sixty, one

fifth; from sixty to seventy, one sixth; and above the age of seventy, as many as eight tenths of those attacked died. If, then, your patient be between fifteen and thirty years of age, and if the amount of lung involved be not considerable, the chances of his recovery are good; but if the whole of one lung be inflamed, or the half of both lungs, then you must look very gloomily at the case; while if the disease affect only the half of one lung, or only a third, or still less, you must take the more cheerful view, and, in accordance with that view, give a more favorable prognosis. Much will depend on the constitutional powers of the patient, his power of vital resistance; and the existence of any previous damage to any vital organ will, of course, militate considerably against a favorable termination. Something, too, may be due to what is termed *epidemic constitution;* for it cannot be doubted that typhus fever, erysipelas, and some other diseases, are much more fatal in some seasons than in others, and this may, to a certain extent, obtain in the case of pneumonia.

Another circumstance which you must take into consideration as influencing the prognosis is the plan of treatment adopted. I believe that in this disease you can do much more by treatment to hasten a fatal result, than to bring about a favorable termination, and that the interference of the physician should be with the view of assisting nature in getting rid of those materials, the retention of which in the system appears to produce the disease. This may be done by promoting the secretions of the skin and kidneys; at the same time paying due attention to the condition of the digestive organs and bowels. Your aim should therefore be to induce free sweating, to apply counter-irritation to the chest, and to exhibit mild aperients. Finally, the patients should always be supported by the liberal, but well-regulated, administration of good nutritious food.

If blood be taken, it should only be with the view of relieving pain, and for this purpose the application of a few leeches over the painful spot is the most successful plan; but when the dyspnœa is extreme, it is sometimes expedient to take a little blood from the arm. Those of you who are in the habit of attending my hospital practice will bear witness how very rarely

I have recourse to this proceeding. To bleed, or not to bleed, is often a question in pneumonia, and one which I earnestly recommend you, as a general rule, to decide in the negative rather than in the affirmative.

Let me conclude what I have to say on this subject, by calling your attention to a report which I have before me, and which Mr. C. Macnamara has been kind enough to draw up for me, of all the cases of pneumonia which have occurred under my care in the hospital from 1840 to 1853, and now corrected up to 1859. The cases are in all seventy-eight, and they have been taken just as they have been noted down in the case-books. They may be arranged into two periods; the one from 1840 to 1847, the other from 1847 to 1859. In the former of these, which may be termed *the period of reducing treatment*, the remedies employed were bleeding, the application of blisters, the exhibition of tartar-emetic, and the more or less free use of mercury; while in the latter, which may be styled *the period of supporting treatment*, the patients were treated very much in the manner which I have detailed to you in this and other lectures on this subject, though in a few cases calomel and opium were given. Now, the total number of cases, as I before mentioned, was seventy-eight, and of these ten were fatal.

Remember that I am now merely stating the leading points in the report, for time will not permit me to enter into any lengthened detail. Of the seventy-eight cases, twenty-five occurred in the first period, and four of these proved fatal, or about one in every six. In the second, the number of cases was fifty-three, and in these there were only six deaths, or about one in nine; fairly leading, I think, so far as we may judge from the relative mortality in these two periods, to the inference that the supporting plan of treatment is more favorable (certainly not less favorable) in its results than the severer measures which are frequently had recourse to in this disease.

LECTURE XVI.

ON CERTAIN ACUTE DISEASES.

ON THE THERAPEUTICAL ACTION OF ALCOHOL.

Gentlemen,—I have selected for to-day's lecture the case of a little girl poisoned by alcohol. I fear few of you have seen this patient, chiefly those who have been about the hospital during the summer months, as she was admitted into the wards, and indeed died, a few days before the commencement of the winter session. The subject of poisoning by alcohol is a most important one, and cases of it are rare; on this account alone it deserves special consideration, but it also enables me to make some observations explanatory of the grounds upon which a good deal of the practice in acute disease, which you will see carried out by me in the wards of this hospital, is founded.

Some of you, coming into the wards for the first time, will doubtless be surprised at seeing that a good deal of wine and brandy is administered to many of the patients. And you must feel inclined to exclaim, "This seems very heterodox practice, and altogether different from that which I had been led to expect, or am accustomed to witness." When you further examine the patients who are under this treatment, and find that many of them have a hot skin and a rapid pulse, you may be even disposed to think that such practice looks like an adoption of the principle, *similia similibus curantur;* but when you look more closely into the matter, you will find that no such absurdity holds its place here. I have been on the look-out for a case upon which I could base a few remarks upon the administration of alcoholic fluids, especially in disease; and happening to be in the hospital when the patient to whom I have alluded was brought in, the house-physician came to tell me that he had just admitted a case of marked interest,

tauntingly replying to my inquiries as to its nature, that it was one of poisoning by a favorite remedy of mine, alcohol. These circumstances, together with many instructive points exhibited by the patient, have induced me to embrace this opportunity of directing your attention to the subject of the administration of alcohol, both as a poison and as a therapeutical agent.

Alcohol, in some form or other, is a remedy whose value can scarcely, I think, be over-estimated, and one upon which, when carefully administered, I rely with the utmost confidence in a great number of cases of disease which are at all amenable to treatment. You must bear in mind that there are very many instances of morbid conditions, which will come under your notice hereafter when you come to practise for yourselves, which are not in the least degree under the influence of any remedies with which we are at present acquainted, and in which the subjects of them will die, do what you will; while, on the other hand, there are many cases in which recovery will take place, no matter what treatment you adopt, even if you give the patient nothing but cold water. Hence, in studying the effects of a remedy in any disease, you must always carefully separate, from the data upon which you are to found your conclusions, these two classes of cases, viz., those which will recover without any treatment at all, and those which will most assuredly die in spite of every effort to save them.

Case LXXXIV (vol. xlvi, p. 114).—The case, then, to which I wish to direct your attention to-day is of a child named Mary Kellar, aged three, who was admitted into the hospital about half-past three o'clock on the afternoon of September 19th, 1855. We were informed that a quantity of *gin*, stated not to have exceeded "half a quartern" (2½ oz.), had been administered by her mother, about nine o'clock on the morning of the same day. The motive of the mother in thus dosing her offspring with gin—whether it was merely a drunken frolic, or whether it was induced by some bad intent—could not be ascertained. Very soon after the dose, the child fell off into a deep sleep; she was then put into a close bed, and some time afterwards the parents, finding that they had great diffi-

culty in arousing her, became alarmed, and at length brought her to the hospital, about *six hours* after the administration of the gin.

Now, the circumstances which here strike one most are the great rapidity with which the symptoms manifested themselves, and their steady increase from the time of the administration of the poison, until the patient came under observation.

Let me read to you the description which the house-physician, Mr. Workman, gave of the little patient:—"The child was well-grown and of healthy appearance; she lay insensible in her mother's arms, as if asleep; the pulse was good, the body and extremities warm, and the eyes presented a natural appearance." As the history of the case showed that these symptoms followed almost immediately upon the child's swallowing a certain quantity of gin, the first indication for treatment plainly was to endeavour to extract from the stomach any alcoholic fluid which might yet remain there. An emetic was therefore at once administered, but was swallowed with considerable difficulty; and as vomiting did not follow soon, tickling the fauces was tried. But even then the little patient merely coughed and struggled, and did not effectually vomit. "Cold water was now poured in a stream upon her head, and as the insensibility of the patient was extreme, mustard poultices were applied to the body, and the feet were well slapped, but all without effect: after this, a mustard bath was tried. The most effectual excitant," continues the report, "was the natural one of slapping the posteriors, to which the child manifested her objections by crying loudly, and by kicking, *but with the right leg only.*"

Here was a new system demanding our special attention in the investigation of the nature of the case. The patient could move the right side only, the left arm and leg being paralysed, and the arm more so than the leg. Some warm water was now injected into the stomach, but when drawn off it did not emit any odour of gin, neither did the breath at any time smell of this fluid; but inasmuch as six hours at least had elapsed between the administration of the alcohol and the time of the child's coming under observation, there was good reason for believing that the spirit must have been entirely absorbed. At half-past

six o'clock the same evening, another new symptom manifested itself, which consisted in an epileptic fit. "The child first opened her eyes, which had been previously closed, as if about to recover her consciousness, and coughed and vomited a little; the muscles of the left side of the body and face then began to twitch convulsively, and the right side also shared in the convulsions, but only in a slight degree." This looked very much as if the peculiar influence which produced the convulsions, whatever its precise nature, was seated in the same side of the brain, and was most probably the same as that which gave rise to the paralysis. "The convulsions lasted about three minutes, and the child then fell again into her previous quiescent state; but this was every now and then interrupted by a similar convulsive paroxysm, which recurred throughout the night to the number of twenty to thirty. The bladder became distended, and in the course of the evening was emptied by means of a catheter. The urine which was drawn off was clear and transparent, and healthy. Five grains of calomel were now given, and this was followed up by a dose of castor oil."

On the 20th the report is as follows:—"The child lies quiet, taking no notice of anything except when roughly pulled about; she has passed plenty of urine, but the bowels have not been opened: the left arm is still paralysed.

"2 P.M.—The condition of the patient is much the same: there has been no return of the fits since nine o'clock this morning: the left leg is but very little paralysed, and the arm also is less so than when last reported: pulse good. The child has been fed with milk and arrow-root.

10 P.M.—About half an hour after the last report, another fit, but a very slight one, occurred; with this exception, there has been no return of the fits. She has vomited everything she has taken during the day: the pulse is weaker and more rapid, and the patient is evidently becoming exhausted. Ordered to take a tea-spoonful of wine every second hour with a little beef-tea, and to have a castor-oil enema administered at once."

On the 21st, the following report was made:—"The aspect of the patient has altered much, and for the worse; she still

lies as if asleep, with her eyes half-closed but deeply sunken in their sockets, and the whole face seems swollen; the sickness is much less than it was yesterday; the breathing is quiet; pulse 156. The pupils are equal, but oscillate a little. The patient exhibits some degree of consciousness, and opens her mouth when food is presented, and once she appeared even to recognise her parents; still she usually lies in a torpid state, occasionally turning in bed with a fretful moan, and tossing her right arm across her face; the left arm she never moves."

On the 22d this note was made: "The symptoms continue much the same as yesterday; the vomiting has ceased, and there has been no return of the fits, but the bowels have not been opened. Pulse 140; respirations 30.

"10 P.M.—The patient is gradually sinking; the pulse and breathing are exceedingly rapid and irregular, but the body continues warm. She swallows readily." The patient continued sinking throughout the night, and died at half-past nine on the morning of the 23d, about four days after the administration of the gin.

The body of this child was most carefully examined after death, but we were unable to detect any abnormal anatomical condition of either the white or grey substance of the brain, excepting an extreme degree of pallor. This state of anæmia is very opposite to that which the notions commonly entertained on this subject would lead us to expect; for, as most of you are doubtless aware, coma and convulsions are generally regarded by authors—most incorrectly, in my opinion—as dependent upon a congested state of the brain—an over-distended state of the cerebral capillaries. In this case the brain generally was so pale that all those who were present at the examination were much struck by it; but there was no alteration in its consistence, though, being that of a child, it was of course softer and more watery than the brain of an adult. There was no effusion into the cavities of the brain, nor any condition by which the coma could be accounted for by way of pressure.*

The thoracic viscera appeared quite healthy; and I ought to have told you that we had several times carefully examined this

* I very much regret that the specific gravity of different portions of the brain was not taken.

child during life, with the view of ascertaining the condition of the pulmonary vascular system (for there are many who imagine that alcohol retards this circulation, and thus gives rise to congestion). On these occasions we always found the breathing pure and vesicular throughout the lungs, except in the lowest part of the inferior lobes posteriorly, which we concluded were much congested, with more or less of an œdematous condition. This state was no doubt due to the dependent position of these portions of the lungs. You are always likely to meet with it in cases in which the vital and nervous powers are materially depressed, as for instance, in fever, in which disease the patient almost always lies upon his back. The effect of posture may be well illustrated after death; for if the body have been lying some time on the back, you will find the posterior parts of the lungs more or less congested; but if you now turn it, and allow it to remain for some time with the belly downwards, the blood will gravitate to the most dependent parts, and the congestion will be transferred to the anterior parts.

This tendency to congestion, exhibited in these cases during life, evinces a feebleness of the nervous influence in the capillary vessels, so that the blood obeys the laws of gravitation more readily than it is permitted to do in health. Thus, in weakly women and in dropsical patients, you will frequently find that the lower limbs *pit* upon pressure, and exhibit the usual characters of œdema; and this condition, you will generally learn, is aggravated towards the evening. These phenomena result from the insufficiency of the forces by which the circulation is carried on in the capillary system, and which, in such low states of vital power, are incompetent to prevent the blood from obeying the ordinary laws of gravitation.

But to proceed with the results of the post-mortem examination. Nothing could be found amiss with the stomach or intestines; the liver looked healthy, and the kidneys tolerably so, but about these last there was some difference of opinion. For my own part, I thought they closely resembled the kidneys of persons who die of that form of dropsy which so frequently follows scarlet fever, the medullary portions being exceedingly red, while the cortical substance was unusually white; this last-named condition resulting apparently from an undue accumu-

lation of the renal epithelium in the uriniferous tubules, and chiefly in their convoluted portions. But you ought never to place reliance upon the condition of the kidneys as determined by naked-eye observation; and whenever there is the least possible doubt, you should always carefully investigate the real state of these organs with the aid of the microscope.

In the present instance, the results of a microscopical examination showed that the renal epithelial cells generally, although not existing in any undue quantity, and still adhering intimately to the basement membrane of the tubular walls, contained in their interior much fatty matter, and that the kidneys were good specimens of that which has been designated by Dr. George Johnson "*the mottled fatty kidney.*"

To what this condition of the kidneys was due, it is exceedingly difficult to say. Two questions suggest themselves. Was it a condition of prior existence, and did it contribute to increase the symptoms excited by the alcohol, or to oppose the recovery of the patient? or was it an effect of the administration of the alcohol? Those of you who are acquainted with the peculiar affinities of fat and alcohol, will find here a fair field for speculation; for my own part, I must be content with simply calling your attention to the clinical facts. Were I otherwise disposed, time would not permit me to enter into so wide a discussion. Let me remark, however, that it seems highly improbable that such a state of kidneys as I have described could be produced in so short a time as that which had elapsed since the gin was taken. On the other hand, I think it not at all unlikely, as the child was pale, and had, no doubt, been always badly fed, although she was not decidedly unhealthy looking, that slow disease of this kind may have been gradually developing itself. You will perhaps remember that a state of kidneys somewhat analogous to this is frequently found in London cats, and it does not appear improbable that those London children, of the lower orders, who are reared in the same sort of darkness as the cats, while they are also irregularly fed upon unwholesome food, and are continually breathing an impure atmosphere, may exhibit the same proneness to this peculiar morbid condition of the renal organs as their feline companions are so apt to do. Assuming that this is the correct

explanation of the condition of the kidneys, we at once obtain a clue to unravel some of the phenomena which this patient exhibited, more especially the continuance of the prostration and coma after the other signs of poisoning by alcohol had entirely disappeared.

Let me now direct your attention to some of the practical information which we may extract from this highly interesting case; and it will be convenient if I at once arrange, under three heads, the points most worthy of your notice. These are:—

1. The manner in which alcohol finds its way into the system, and the organs or textures on which its influence is most directly exerted, and for which it has the greatest affinity.

2. The mechanism, so to speak, of its action on the body, and how it may operate as a poison.

3. How far it may be used as a therapeutic agent, and the manner in which it may be employed as such, whether in acute or chronic disease.

I. Perhaps the most remarkable phenomenon exhibited in this case consisted in the extreme rapidity with which the symptoms were developed after the ingestion of the gin. How is this to be explained?

If you will take the trouble to look to the histories of various cases of poisoning by alcohol, reported in books on toxicology (and I know of none better than that of Dr. Christison), you will find that there is nothing peculiar in this speedy occurrence of the symptoms, where persons—even adults—have swallowed a considerable quantity of spirit; indeed, that it may be considered as the rule, that after the ingestion of alcohol its peculiar effects show themselves with remarkable rapidity.

Let me briefly refer to two or three of the cases illustrative of this subject, which you will find recorded in Dr. Christison's work. One is that of a man who stole a bottle of whisky, and finding that he was in danger of being detected, but being unwilling to part with the stolen spirit, at once drank off the entire contents of the bottle; but he would scarcely have done so had he been aware of the remarkably rapid manner in which alcohol produces its effects; almost immediately he fell into a state of coma,

which continued until death took place, about *four hours* after he had swallowed the whisky. Another case is that of a boy who drank a large quantity of raw whisky, and immediately after taking it, fell into a state of coma, upon which, in the course of two hours, tetanic convulsions supervened. The stomach-pump was now employed, and the greater part of the whisky withdrawn, when the lad recovered his consciousness for about a quarter of an hour, but he afterwards relapsed into a state of stupor for the whole of the day. A third case is that of a lad, fifteen years of age, who laid a wager that he would drink sixteen ounces of whisky, and then, to show how little effect it would take upon him, would walk up and down the room a certain number of times. This feat he performed, and for some time after appeared none the worse for it; but, about half an hour subsequently, he walked out into the open air, and, whilst in the act of putting his hand into his pocket, he became perfectly insensible, and was, of course, unable to take his hand out again. From this moment he gradually became more and more comatose, and at length died, about *sixteen hours* after having drunk the whisky.

These cases recorded by Dr. Christison correspond very much with that which I have brought before you to-day, and they show how rapidly the symptoms peculiar to poisoning by alcohol supervene upon the swallowing of a large dose of that fluid. The principal reason of this seems to be, that alcoholic liquids, especially spirits such as whisky, brandy, &c., are, within certain limits, extremely easy of digestion. In other words, the mode in which the digestion of alcohol takes place is by no means complicated, but consists rather in the simple process of absorption. A man drinks a glass of brandy and water; what becomes of it? It passes down into the stomach, and is at once absorbed directly by the blood-vessels, which, as you know, ramify so freely in the walls of this viscus. If, however, the quantity of spirit and water taken be very great—say a couple of pints or more—and if a short time be occupied in drinking it, then a part passes down into the intestines, but the greater part still remains in the stomach, and finds its way, by a process of *endosmosis*, directly into the blood-vessels of the mucous membrane, with which it is in contact.

The digestion of lean meat is the next in point of simplicity

to that of alcohol. After undergoing proper mastication in the mouth, and a certain amount of admixture with the salivary secretion, meat enters the stomach, where it is subjected to the solvent action of the gastric fluid; and, having been thus dissolved, whilst at the same time it undergoes a certain amount of chemical change, it for the most part passes immediately into the blood-vessels of the stomach. But the process, *preparatory* to digestion, which occurs in the stomach in the case of meat, has nothing analogous to it in that of alcohol; the latter being, when swallowed, already in a form which admits of its complete and ready absorption by the gastric blood-vessels. The fact that there is no need for any previous process prior to absorption explains the rapidity with which the alcohol enters the circulation, and its immediate influence in producing the symptoms, as observed in our patient and in those whose cases are related by Christison.

It was long believed that alcohol exercised some special influence on the sentient extremities of the nerves of the stomach and intestines; and that it was not absorbed into the blood at all. But of its actual absorption, when swallowed, there is abundant evidence, as well as of the almost incredible rapidity with which it is diffused through the circulation. An important proof of this absorption is furnished by the fact that very soon after swallowing the spirit, especially when taken on an empty stomach, fumes of alcohol are perceptible in the breath expired. And it is a highly interesting fact, which we have observed very often in the hospital, that in cases of great exhaustion of the system, when it stands much in need of alcohol, this does not occur; the spirit undergoing certain chemical changes in the body, and not passing out through the respiratory organs in the form of alcohol.

II. The effects produced by the free ingestion of alcohol are all referable to the *nervous system*. The prominent symptom is *coma*. If you institute a careful search into all the cases of poisoning by alcohol upon which you can lay your hands, you will find that in every one of them a greater or less tendency to coma was present. To coma are sometimes added *convulsions*, generally of a clonic, but occasionally of a tetanic character: in

some cases the coma is preceded by *delirium*, in others it is followed by *paralysis*. When the fluid swallowed contains a very large proportion of alcohol, *vomiting* is apt to occur; the alcohol in these instances probably acting as a direct irritant to the mucous membrane of the stomach, which becomes red, vascular, and congested, as if it had been subjected to the action of an irritant poison. Generally speaking, however, symptoms referable to the *digestive* organs do not take a prominent place among those induced by the ingestion of alcohol.

And here I should remind you that ale, porter, wine, brandy, gin, &c., contain alcohol in very different proportions. With these proportions you ought to be familiar, for this is a point upon which, as a matter of dietetics, you will be hereafter almost daily consulted.

But to return to the symptoms due to the ingestion of alcohol. The principal effects, as the symptoms in the cases which I have detailed show, depend on the influence exercised by alcohol on the nervous system. Alcohol is, as you are aware, a hydrocarbon; and almost all hydrocarbons have a marked affinity for the nervous system, as compared with the other structures of the body; and it is upon the nervous centres that alcohol exerts its primary influence. At first its action is simply to augment the generation of the nervous power. When this increased action of the nervous system is kept within certain limits, the effect of the alcohol is beneficial; but when carried beyond a certain point, its action is injurious and deteriorating, the spirit at length impairing, and ultimately destroying, the nutrition of the nervous matter.

Evidence of the tendency of alcohol, when taken in excess, to damage the nutrition of the nervous matter is to be found in the peculiar tremblings and impaired mental power, which are commonly present in cases of delirium tremens. These tremors are solely indicative of too great waste of nerve-matter, and they often occur under circumstances in which there has been an undue expenditure of nervous power, such as great mental anxiety or fatigue, without any recourse having been had to the use of alcoholic liquids.

In persons who have died after a long course of intemperate indulgence in fermented liquors, we find a further proof of the

damage done to the nervous system, in the shrunken brain, the wasted convolutions, the increased amount of subarachnoid fluid. The use of alcohol in too great quantity—in other words, its abuse—causes a wasting and degeneracy of the brain, both of gray matter and fibrous matter. There is no congestion unless the death of the patient has been immediately preceded by a prolonged struggle, or by convulsions; and the subarachnoid fluid merely follows the brain-shrinking, and serves to fill up the space which that shrinking had left vacant.

Of the various unfavorable effects which alcohol is capable of producing, it behoves the practitioner to observe that inflammation of internal organs is not one. It is true that a strong alcohol, applied directly to the mucous membrane of the stomach or the conjunctiva of the eye, may cause inflammation of those membranes. But alcohol absorbed into the blood will not excite inflammation of the lungs, or the heart, or of the liver,* or kidneys, or the bowels, nor even of the brain. I have never seen, nor heard, nor read of any authentic case in which inflammation of any of these organs was attributable to the ingestion of alcohol. Look at the case of our little patient; there was not a particle of inflammation in her body, not even in the mucous membrane of the stomach. The brain was anæmic, but altered in its consistence, showing that the poison acted on the nervous matter, by attraction from the blood, and illustrating how coma, convulsions, and paralysis may all occur independently of hyperæmia or of inflammation.

I apprehend, that in all cases of brain affection from alcohol, the symptoms are due, not to congestion nor to inflammation, but to a simple poisoning of the nerve-fibre and nerve-cell, by the spirits. It is the same with chloroform and sulphuric ether, when inhaled. They act directly on the nerve-fibre and nerve-cell, and not by producing congestion. If you soak a living nerve in chloroform for a certain time, it will refuse to propagate the nervous force under chemical, mechanical, or electrical stimuli.

I think, then, we may discard the popular prejudice, that alcohol causes inflammation, and that therefore it ought not to be given in inflammation.

* Cirrhosis of the liver is an atrophy, not an inflammatory disease of that organ.

There is another notion which certain objectors to the use of alcohol are fond of adducing, namely, that if alcohol causes exaltation of nerve-force, that is followed by a corresponding amount of depression. This, I am sure, is a fallacy due to the comparison of a patient's feelings when under the immediate influence of the spirit, or other alcoholic fluid, with the sensations which accompany the absence and the want of its exciting influence. There is no evidence to show that any physical depression is induced as a sequel to the alcoholic excitement, that any increased waste goes on, or that the nervous power is at a lower ebb than *before* the administration of the alcohol, if it have been given in proper quantity. When alcohol has been taken in *inordinate quantities*, then the feeling of depression is the greater by reason of disturbed digestion; but even under these circumstances evidence is wanting to demonstrate the existence of a distinct physiological depression of vital power, unless by its repeated over-use the nutrition of the nervous system be impaired.

In its further effects upon the body, alcohol, taken carefully, increases the animal temperature; it also strengthens the action of the heart; and, when administered under proper circumstances, it reduces the frequency of the pulse. So far as it influences the nervous system, the action of alcohol is that of what is commonly called a *stimulant*, an unfortunate term, indicating a distinction without a difference. Other forms of food are likewise stimulant, but as they do not act directly and quickly on the nervous system, their exciting properties are not so apparent. In like manner, alcohol possesses its stimulating property because it is a form of aliment appropriate to the direct nourishment of the nervous system, and to its preservation; and its special adaptation to this system gives it an immediate exciting power superior to any other kind of food. Alcohol is also fitted to uphold the calorifacient process, and therefore to protect the nervous and other tissues, which, without it, would be largely called upon to furnish fuel for the oxygen which supports that process. And for this purpose, alcohol possesses considerable advantages over most other hydrocarbonaceous substances, in consequence of the ready manner in which, when swallowed, it passes into the blood. Oil is a hydrocarbon equally with alcohol,

but the digestion of the latter is a very different affair from that of the former. Oil, when swallowed, remains for some time in the stomach, where, however, it undergoes no change; it subsequently passes down into the small intestine, where it is subjected to the action of the pancreatic and other fluids, and having become converted into chyle, is absorbed by the intestinal villi. Thus the digestion of oil is a far more complicated process than that of alcohol. Hence, then, as a calorifacient form of food, as a promoter of the nutrition of the nervous system, and as admitting of easy and quick absorption into the blood, alcohol possesses a combination of qualities which render it of the utmost value in the treatment of disease.

Observe, I beg of you, that I use the term alcohol merely generically, to comprehend the various fluids which contain alcohol, in greater or less quantity. Alcohol is always, I presume, to be presented to the human stomach in a more or less diluted form. Pure alcohol, likewise called *absolute*, or *anhydrous*, can only be taken with extreme caution, and in very small doses. Its influence on the mucous membrane would be that of an irritant; that is, it would so disturb the physical condition of the elements of that membrane as to create hyperæmia, to interfere with its secretions, and consequently greatly to impair primary digestion. In the same way a large quantity of any alcoholic fluid, quickly swallowed, will produce a similar effect, and will cause considerable disturbance. And we learn from this, that the true method of administering these fluids is, to use a common phrase, by giving "a little and often." Fix upon a dose which you may think your patient's stomach will bear—from two to twelve, or even sixteen drachms—and give that more or less diluted with water, or some other bland liquid, at such intervals as his state of vital power may seem to demand.

And if we look at alcohol in this point of view, and especially as a food peculiarly adapted for the calorifacient process, and admirably suited to shield the tissues against the influence of oxygen, under circumstances when substantial food adapted to the nourishment of those tissues cannot be taken, looking, I say, at alcohol in this point of view, it is plain that the quantity to be given is that which should especially engage the atten-

tion of the practitioner. You must give enough to keep up animal heat, and to protect the tissues, without embarrassing the stomach, and without allowing the fumes of alcohol to be perceived in the breath. All that is given over and above that quantity is wasted, so far as the support of the vital powers is concerned. Now, we cannot as yet exactly measure the wants of the system, and it is only by trial and experience that we are able to adjust approximatively the quantity to be given to the wants of the system. For this reason I have adopted the plan of giving a precise dose at brief intervals, from two to twelve or sixteen drachms, diluted, just as you give so much calomel, or so much opium, at specified times.

III. Having now examined the poisonous effects of alcohol, and also explained to you its mode of action, let me call your attention very briefly to the particular forms of disease in which you are most likely to find alcohol useful for promoting the healing process by upholding vital power.

But here I think it necessary to guard myself against the imputation of encouraging the excessive and improper use of alcohol. No one who thoroughly appreciates the great value of this agent in the treatment of disease will regard with indifference the abuse of it in health. And I will yield to no one in the desire to see this beneficent gift of God to man (so easily converted into a destructive poison, capable of weakening nervous power and of annihilating all the best qualities of his mind) employed with the care and moderation through which alone it contributes to the health of mind and body. It is curious, indeed, to observe that, with the exception of the very lowest races of men, there is scarcely one of the various tribes with which this earth is peopled, which has not discovered for itself some method of preparing an alcoholic liquid; showing, I think, the existence of an instinct in human nature for such a food as alcohol.

Alcohol may be employed in all those diseases in which a *tendency* to depression of the vital powers exists; and there are no acute diseases in which this lowering tendency is not present. Many such maladies will, doubtless, get well without the interference of art; cases of pneumonia and of fever, for

example, will frequently recover without the employment of any medicines. But take any case of pneumonia thus left to follow its own course, and after eight or ten days you will always find your patient more or less emaciated, and in a much more prostrate condition than on the accession of the malady. If, however, in such a case tartar emetic had been exhibited, or blood-letting practised, how much more depressed will be his condition! Hence the great importance of upholding your patient's powers while the natural processes of the disease are taking place. In pneumonia a peculiar effusion occurs into the air-cells, which after a while become filled, the lung itself becoming dense and solid. For the recovery of the patient all this effusion has to be absorbed, and the air-cells restored to their original condition. The processes which nature has to perform in the cure of this disease are highly complicated, and no one, I apprehend, will assert that we possess any drug which by its direct agency on the system can effect this object. In the accomplishment of these changes there is a considerable expenditure, as it were, of nervous force and of blood; and therefore, we should supply to the system a kind of food which, while it is easily assimilable, is at the same time capable of upholding the nervous power, and of maintaining the animal heat. And such a food is alcohol, which, as I have already pointed out, is assimilated in the easiest manner by a simple process of endosmosis, exercises a peculiar influence on the nutrition of the nervous system, and, by its combination with oxygen in the body, supplies fuel for the maintenance of the animal temperature. When given in too large a quantity, alcohol passes out of the body unchanged in the air expired from the lungs; but when its amount is limited and proportionate to the real wants of the system, it is evolved in the form of carbonic acid and water, and promotes the secretions of the lung, the skin, and the kidneys.

The successful use of alcohol in the treatment of disease depends very much on its *mode of administration*. Alcohol should be given in such doses as experience shows are assimilated with the greatest ease. Let me illustrate this by an example. If two individuals drink a bottle of brandy each, the one in the course of one or two hours, the other in doses of

half an ounce every hour until the whole is consumed, the effect of the alcohol in the two instances will be very different. The former person will have become more or less intoxicated, or, in common language, "*drunk*," unless he have been long addicted to large potations of brandy; but the latter will suffer but little general disturbance, except that the heart's action will be somewhat excited, and certain of the other effects of the ingestion of alcohol produced, but there will be no delirium, drunkenness, or coma. This difference in the effect of the alcohol in each case arises from the *mode of administration*. A difference of a similar character is also observable in the influence of some other medicines; for example, opium.

In prescribing alcohol in disease, you should not order so much brandy, or other spirit, to be given *daily;* but you should direct a certain quantity—say a tea-spoonful or a table-spoonful —diluted with water, to be administered every *half hour*, every *hour*, or every *two or three hours*, according to the nature of the disease and the actual condition of the patient.

There are at the present moment several cases in the hospital, in which alcohol is being administered in the manner I have just pointed out, to some of which I may briefly refer.

Case LXXXV.—James Booth, aged twenty-one, a baker, now in No. 4 ward; brought into the hospital labouring under typhus fever, which came on about a fortnight before his admission. He was immediately ordered to take *half an ounce of brandy every two hours*, while at the same time another form of stimulant, namely, ammonia with chloric ether, was also administered.

The case was not a bad one; but its progress shows the steady decline of the pulse under this plan of treatment. On admission the pulse was 90, the respirations being 30; the following day, the pulse was 86, the respirations being also 30; on the third day, the pulse was still 86; on the fourth day, it was 70; on the fifth, 72; on the sixth, 65; on the seventh, 65; and so it has continued at about the standard of health.

Case LXXXVI.—A lad, aged seventeen, suffering from typhoid fever; the pulse on admission being 130, and the res-

pirations 24. *Half an ounce of brandy* was ordered to be given *every three hours*, and likewise ammonia and chloric ether, as in the preceding case. The following day, the pulse was 120, and the respirations 24. On the third day, the pulse had risen to 125, at which rate it also remained on the fourth day. Many practitioners would have now said that the brandy was disagreeing with the patient, and was increasing the frequency of the pulse; but experience had taught me otherwise, and I came to the conclusion that the amount of alcohol supplied was insufficient; the dose was, therefore, increased to *half an ounce every hour*. And my expectations were realized; for, on the fifth day, the pulse had fallen to 120; on the sixth day, it was 110; on the seventh day, also 110; on the eighth day, it was 105; and on the tenth day, 96; and it subsequently continued gradually and steadily declining under the use of twelve ounces of brandy *per diem*.

In the treatment of pneumonia, I do not consider the administration of alcohol essential, but rather regard it in the light of an important accessory.

Case LXXXVII.—A case of this disease is now under my care in No. 4 ward, in which nearly the whole of the right lung was hepatized. In this case counter-irritation has been freely applied to the chest, and the patient on admission was put upon large doses of the *liquor ammoniæ acetatis*, while wine was also administered in doses of half an ounce every second hour. Under this plan the case has progressed favorably; and the pulse, which on admission was 90, has gradually decreased in frequency, so that on the ninth day after admission it had fallen to 76.

In bronchitis alcohol is not less useful and more necessary than in pneumonia. I give you from my case-book the following case in illustration of its administration in the former disease.

Case LXXXVIII (vol. xlix, p. 234).—Phœbe Skinner, a thin, spare woman, forty-five years of age, married, with a large family, and poor general health, was admitted into King's

College Hospital, March 13th, 1855, suffering from an attack of bronchitis.

The patient stated that she had been long subject to severe catarrhal attacks in winter time, and had been twice before in the hospital with bronchitis; that for the last three months she had suffered much from indigestion, so that she had been unable to eat, even sparingly, without suffering much pain in the stomach and chest afterwards.

Her illness had commenced a week previously, with great pain in the left side, accompanied by a frequent and hard cough, and frothy expectoration; at the same time her breathing became quick and difficult.

When admitted, her face was flushed, but of a dusky hue; she coughed frequently, expectorating a greenish frothy mucus, and complained of thirst, alternate flushings and chills, and aching in her limbs. Pulse 120, feeble, and somewhat irregular; respirations 36. Tongue coated. Crepitation was heard throughout the right lung, but most developed behind, and rhonchus and sibilus in the left.

Five grains of carbonate of ammonia were ordered to be given in decoction of senega every four hours, a blister to be applied to the left side, and turpentine stupes to the front and back of the chest.

The following day, March 14th (eighth of the disease), our patient continued extremely ill; towards evening the pulse became very rapid and feeble, and the respiration extremely embarrassed; expectoration was very imperfectly performed, and with the greatest difficulty, and there seemed to be some danger of suffocation. Half an ounce of wine had been ordered her, on admission, every third hour; this was now changed for half an ounce of brandy every second hour.

By the morning of the 15th (ninth day), she appeared much relieved; the respirations were reduced to 27, the pulse to 108. The cough continued, and there was now a copious frothy expectoration, faintly tinged with blood.

On the 18th (twelfth day), the cough continued very troublesome, and expectoration, although freer, was still difficult. Wheezing and large crepitation were heard throughout the

right lung, and the posterior part of the left. The patient seemed very feeble. Pulse, 102; respirations, 27.

On the 20th, although the pulse had fallen to 88, the respirations had increased in frequency to 36. In other respects she was much the same. Two grains of quina were now ordered three times a day.

From this time there was very little further improvement for nearly three weeks: the patient continued very weak, and the cough troublesome, with much frothy expectoration. She slept badly at night, and suffered from headaches, want of appetite, a furred tongue, pain in the side or epigastrium after eating, and attacks of diarrhœa.

The brandy was discontinued, and a little wine given. On the 1st of April, the quina was changed for iron.

By the 10th of April the cough began to subside, and the expectoration greatly diminished.

On the 11th, the patient was attacked with violent pain in the stomach, and headache, and had three epileptic seizures in quick succession. The same thing had occurred to her once before, when recovering from bronchitis.

From this time she improved steadily, and was discharged well on the 26th.

You have here an example of marked improvement in the state of the pulse, following immediately upon a greatly increased allowance of alcohol. Under it the pulse fell from upwards of 120 on the eighth day, to 108 on the ninth.

There was some peculiar state of nervous system in this patient which seemed to explain her prolonged convalescence, and which was relieved by a discharge of nervous force, in the shape of three epileptic seizures. It is not likely, although not impossible, that these seizure were connected with any deranged renal action.

There are also at the present moment three cases of *erysipelas* under my care, in which this plan of treatment has been pursued with perfect success. And here let me remark, that there is no disease in which one may dogmatise so decidedly as regards the beneficial action of alcohol as in *erysipelas*. Often indeed it appears to me to act as a direct antidote to the erysipelatous poison; for you will find that when administered *early*

and steadily, not only does the malady yield readily, but those formations of pus (secondary abscesses), which under other plans of treatment are of so frequent occurrence in this disease, hardly ever make their appearance.

The early administration of alcohol, in the manner I have recommended, exercises a most remarkable and unequivocal influence in preventing or materially limiting the intensity of *delirium*. Delirium is a symptom of enfeebled and contaminated nutrition of the brain. It is to be looked for in all exhausting diseases, and in all acute maladies accompanied by high fever. You meet with it in the acute internal diseases, in the exanthemata, in erysipelas, in typhus and typhoid, in influenza, in the rheumatic and gouty fevers, and after severe burns, or compound fractures, or great surgical operations, after parturition, and in profuse hæmorrhage, from whatever cause. In all such cases, the timely administration of alcohol will *prevent* or mitigate the delirium, and will check it if it have come on early. It is also applicable to the treatment of delirium of the hysterical and epileptic types, although in such cases it will not in general be found to tell with such marked effect as in the delirium which accompanies acute disease.

The following is an interesting example of the latter form of delirium, occurring in connexion with one of the exanthemata, and of the good effects of alcohol, as well as of the evil of deferring the administration of a proper amount of support until an advanced period of the disease.

Case LXXXIX.—A middle-aged, pale, thin gentleman, a lawyer by profession, had an attack of small-pox, by no means severe, and without copious eruption, which was not confluent anywhere. He was treated by sudorifics and aperients, and kept low on slops without broth. Just before the complete maturation of the pustules, he became violently delirious. In his excitement one day, he jumped from his bed and made for the door, and on his way his foot slipped, he fell heavily and dislocated his shoulder.

The delirium was not improved by this accident, nor by a subsequent attempt, almost immediately after, to reduce the

luxation. He struggled violently and successfully against the surgeons, and, in his efforts, contrived to push himself close to a window, at which he aimed a violent kick, thrusting his foot and leg through the pane of glass, and inflicting a severe cut on the leg, which bled very freely. Soon after this, I was requested to see him in consultation with one of his medical attendants. Perceiving at once the nature of the violent delirium in which I found him, I requested that, while we were holding some conversation as to his previous history, he might have two glasses of port wine given him. This seemed to have a somewhat tranquillising effect, and when we came to examine him, we found him more rational, and less violent. It was agreed to give him port wine very freely; at first about two ounces every hour, and a little opium and carbonate of ammonia. In the course of a few hours he began to sleep; still we did not allow him to sleep continuously for more than two hours, but had him roused to take his port wine. When the delirious state was very much subdued, he was allowed to sleep longer. Solid food was given, and the port wine was gradually and cautiously diminished. In three or four days all trace of delirium had disappeared.

Now, in this case at first there was no indication of any great depression of vital power. The absence of adequate support, and the existence of considerable febrile disturbance, gave rise to the condition which favoured delirium. Had the supply been in the first instance better adjusted to the waste, delirium would not have occurred, and we should not have found it necessary to give alcohol so freely as was subsequently done.

Let me add one or two more examples to illustrate the application of this treatment to delirium in acute disease.

Case XC (vol. lvii, B. p. 105).—J. Parkinson, aged thirty-eight, a joiner, was in good health, and at work until July 30th, 1858, when he was attacked with sore throat, shivering, and headache. The throat became rapidly worse, so that he had great difficulty both in swallowing and breathing. A blister was applied over the larynx. About the fourth day he began to wander in his mind, and got out of a window on to the roof of

his house. His wife stated that, four years previously, he had had a very similar but less severe attack.

He was admitted into King's College Hospital, August 3d, 1858, the fifth day of the disease. He was then slightly delirious; there was great redness and swelling of the uvula and tonsils, and a furred tongue; fine crepitation was audible at the base of each lung.

On the 4th (sixth day), the fauces were observed to be very red, and the tonsils still swollen; and this condition was accompanied with great difficulty of swallowing. The patient appeared also to have some difficulty in speaking; every effort to do so was accompanied by a peculiar tremor of the lips and tongue preceding articulation. There was dulness on percussion, with some bronchial breathing, and a considerable amount of fine crepitation at the base of each lung behind, but rather more on the left than the right side. Pulse, 80; respirations, 18. Heart intermitting at every fifth or sixth pulsation. Tongue, brown and furred in the centre; urine, albuminous; the precipitate occupying about one-fifth of the depth of the fluid. Half a drachm of the aromatic spirit of ammonia, in camphor mixture, was ordered every four hours, half an ounce of brandy every hour, and turpentine stupes to the chest.

The next day, August 5th (seventh day of the disease), the patient was rather more delirious. The fauces were still red, with a quantity of dirty viscid mucus hanging about them. No cough or expectoration. Pulse, 84; respirations, 26. The dulness remained at the base of the left lung, but had disappeared from the right side, and there was much less crepitation. The urine contained only a trace of albumen.

At night he became more restless and delirious, and was with difficulty kept in bed. A castor oil enema was administered, as the bowels had not been moved since his admission, and the brandy was increased to half an ounce every half hour.

On the 6th (eighth day), the patient was sweating profusely. The throat was better, the fauces not so red, and the tongue cleaner. There was still some bronchial breathing at the base of either lung behind, and slight dulness on percussion remained on the left side, but crepitation had almost disappeared. The urine was free from albumen. Pulse, 78; respirations, 28.

On the 7th, there was still some delirium. Bronchial breathing had entirely disappeared. Pulse, 72; respirations, 26. Tongue clean. The brandy was reduced to half an ounce every hour; and, as the patient's bowels had not been moved, a draught of senna and aloes was given.

On the 8th (tenth day), an erythematous blush was observed, for the first time, on the left leg, extending between the knee and the ankle. Pulse, 75; heart still intermitting. The brandy was reduced to half an ounce every second hour. Two calomel and colocynth pills, to be followed by a black draught, were ordered.

On the 9th (eleventh day), the patient continued better and quite free from delirium. He was expectorating a little frothy mucus. The leg was still red and tender.

By the 10th (twelfth day), the erythematous blush had faded considerably.

On the 15th (seventeenth day), he was sufficiently well to be discharged.

Case XCI* (vol. lvii, B. p. 57).—Jane Brabbs, aged forty-two, a woman of intemperate habits, after drinking hard for two days without taking food, found herself trembling, unable to sleep, and haunted by spectral illusions. In this condition she was brought to the hospital, June 11th, 1858. She was then talkative and restless, starting up frequently in bed. A drachm of tincture of opium and two ounces of brandy were administered about four o'clock in the afternoon, and repeated at nine; and about half an ounce of brandy was administered every hour through the night (with beef-tea), but no sleep was procured.

The following day, June 12th (third of the delirium), there was less tremor, and the patient spoke rationally, although rapidly. A mixture of chloric ether and ammonia, with fifteen minims of tincture of opium, was ordered every four hours. In the afternoon chloroform was administered by inhalation, but failed to induce sleep.

Throughout the night brandy was given in doses of half an ounce every half hour, but was again reduced to half an ounce

* Reported by Dr. E. S. Thompson.

every hour on the morning of the 13th. The patient was then very talkative; but in the afternoon, after eating a chop, she went to sleep and slept for three hours. She awoke better, but not free from hallucinations, and again passed a sleepless night.

During the 14th (fifth of the delirium), the patient remained awake, although the pupils were contracted and the opium continued. At night she dozed, waking every hour for brandy. The next day, the 15th, she was much less talkative and had a fair appetite.

On the 16th (seventh day), as no sound sleep had been obtained, the dose of opium was increased to twenty minims, and in the afternoon chloroform was administered by inhalation. In the evening she became noisy and violent, and the chloroform inhalation was repeated; but when its immediate effect had passed off, she was so violent that it was found necessary to remove her to a ward by herself, and to put her under restraint.

On the 17th (eighth day), the patient was still in the same excited state, violent and vociferous; large doses of opium were administered at short intervals without any benefit. At two o'clock in the afternoon, half a drachm of chloroform was given by the mouth, and shortly afterwards the dose was repeated. But it was not until a third and double dose had been given that any sleep was obtained; she then slept from six to half-past eight.

At ten o'clock that evening, the brandy was again increased to half an ounce every hour, and the chloroform repeated. The patient slept from eleven o'clock to half-past five the following morning, the 18th, and then awoke much more rational, although still somewhat excited and trembling. The dose of chloroform was repeated.

At twelve o'clock that day, as she continued better, the brandy was reduced to half an ounce every hour, and the chloroform was ordered to be given in half-drachm doses every four hours.

Under this treatment our patient continued to improve. On the 19th (tenth day) the chloroform was reduced to half a drachm every six hours. She slept for a good part of the night, and awoke more herself on the morning of the 20th. The brandy was then reduced to half an ounce every second hour.

On the 21st (twelfth day), although very weak and restless,

she had a better appetite and less tremor, and was able to sleep for several hours consecutively.

On the 23d she still remained very weak, and slept but indifferently. The brandy was changed for a pint of porter.

On the 24th (fifteenth day), she seemed to be gaining strength, and now slept soundly. The chloroform was discontinued, as it appeared to make her sick. On the 28th (nineteenth day) she was discharged well.

The following is a case of epileptic delirium :

Case XCII (vol. lvi, p. 202).—Caroline Lewry, aged twenty-nine, single, was admitted into King's College Hospital, Feb. 25th, 1858, in a state of violent delirium.

We learned that she had been in poor health and subject to epileptic fits from childhood ; that these had recurred at intervals of about a month, always leaving her weak and exhausted for a day or two afterwards. Her mother stated that she had lately been living very badly, and that poor living always increased the frequency of the fits.

On February the 21st she had a fit, from which she recovered as usual ; but on the 24th had a second attack, and from that time became delirious, and at length so violent that she was brought to the hospital.

When admitted (second day of the delirium), she had a wild expression of face, and could with great difficulty be kept in bed ; she seemed to be haunted with spectral illusions, and was constantly singing. The skin was moist and cool ; the tongue dry and furred. Pulse 120, weak and compressible.

Two ounces of brandy were immediately given, and half an ounce ordered every hour.

The next day, February 26th (third of the delirium), the patient remained in very much the same condition, frequently starting from her bed, and fixing her eyes on some imaginary object ; but she was less noisy and violent. The pulse had fallen to 96. The brandy was continued, and a mixture containing carbonate of ammonia, with eight minims of tincture of opium in each dose, was ordered every four hours.

On the 28th (fifth day), the delirium was still of the same character ; the pulse had fallen to 80.

On the 1st of March (sixth day), the patient was better—no longer violent, and able to sleep a little. Pulse 76.

On the 2d (seventh day), the delirium seemed to be passing off, and she only started occasionally and complained of a mist before her eyes and slight headache. Pulse 76. Tongue clean.

By the 3d (eighth day), our patient was quite free from delirium, and slept well. She complained of some headache and a pain in the præcordia. The heart sounds were natural, and the urine healthy. Pulse 76. Brandy reduced to half an ounce every second hour.

On the 4th, a quinine mixture was ordered. She continued improving under a tonic treatment until the 13th (eighteenth day). The headache had then ceased; she slept well, and had a good appetite, and was shortly afterwards discharged.

Let me invite your attention to the tabular statement showing the gradual decline in the frequency of the pulse under the free administration of alcohol.

TABLE OF PULSE AND RESPIRATIONS.

	Date.	Pulse.	Resp.	
Brandy ℥ij immediately, and ℥ss every hour afterwards.	Feb. 25th	120		During the delirium.
	,, 26th	96		
	,, 28th	80	24	
	March 1st	76	24	
	,, 2d	76	26	
	,, 3d	76	26	
	,, 4th	78	30	Delirium ceased.
Brandy ℥ss every 2d hour.	,, 6th	80	30	
	,, 9th	78	28	
	,, 11th	80		
	,, 13th	80		

Case XCIII* (vol. xlix, p. 75).—Harriet Smith, aged thirty-eight, while residing in the same house with a person ill of fever, began to feel unwell, and was soon afterwards attacked with an intense headache. She quickly lost all consciousness, and now that she is well, remembers no further particulars of her illness.

* Reported by Mr. J. H. Thornton.

This patient was brought to the hospital, July 29th, 1855, about a week from the commencement of the malady, in a state of almost complete insensibility. When put to bed, she lay nearly motionless, picking, however, at the bed-clothes with her fingers, and occasionally moaning or muttering, or crying out if moved. When loudly spoken to, she appeared to be somewhat roused, but not sufficiently to answer questions. Both urine and fæces were passed unconsciously in the bed. A number of fawn-coloured spots were observed scattered over her body, especially on the chest and abdomen. Pulse 135.

A mixture of chloric ether and ammonia was ordered every third hour, an enema of ten grains of quina, in two ounces of beef-tea, three times in the day, and half an ounce of brandy, with beef tea, every hour.

On the 30th, the pulse and respirations were 115 and 28; on the 31st, they were 116 and 24. The patient remained in the same state of stupor, picking and muttering, but taking her food and brandy well. She was also perspiring freely. No diarrhœa nor relaxed motions.

On August 1st, about the eleventh day of the disease, she was rather more sensible; skin hot, but perspiring less; pulse, 120; respirations, 28.

On the 2d, the pulse and respirations were 114 and 24 respectively; on the 3d, 108 and 24. The patient was better, and had left off muttering and picking the clothes, but now began to vomit her food and medicine.

On the 4th (fourteenth of the disease), she was much better, and able to give rational answers to questions, but still passed her evacuations unconsciously, and the vomiting continued. Tongue cleaner; pulse, 120; respirations, 20.

On the 6th (sixteenth day), the pulse and respirations were 106 and 20; on the 8th, 100 and 20. Tongue much cleaner. The patient was still troubled with sickness.

On the 9th, a quinine mixture was substituted for the chloric ether and ammonia, and the following day the sickness entirely ceased. The patient now complained of pain in one knee. Pulse, 96; respirations, 24.

On the 13th (twenty-third day), she was still suffering great pain in her knee, and the pulse had risen slightly to 108.

By the 16th (twenty-sixth day), there was a very great improvement. Our patient was fairly convalescent, and able to eat and sleep well, and to get up for a short time. Pulse 84. The brandy had been gradually reduced. She continued to improve, and gain strength, until the 25th, when she was discharged well.*

You will ask, what are the signs that too much alcohol is being given, and what are the indications which should suggest to the practitioner that he ought to diminish the quantity, or to withdraw it altogether.

I must tell you, *in limine*, that it is far more dangerous to life to diminish or withdraw alcohol than to give too much. It is plain to common sense that, in the latter case, you may easily and quickly reduce the quantity, or suspend the use of alcohol altogether; but it is not so easy to make up for lost time, and to supply readily a want which had been felt for many days. Hence it sometimes happens (as in Case LXXXIX), that the withholding such a supply in the early period of acute disease leads to the employment of a much larger quantity in the latter stages.

The signs upon which I recommend you to place your chief reliance, as indicative of the supply being too much, are mainly referable to the digestive organs. They are such as show that the first stage of digestion—stomach digestion—is disturbed. Your patient will suffer from flatulence, frequent eructations, even sickness, dryness of the tongue and mouth, with some degree of sordes of the teeth and lips. Of course you will take pains to separate such symptoms as arising from too much alcohol, from similar ones as belonging to the disease.

It is important that you should watch the effects upon the nervous system. When alcohol is given as I have recommended, it calms the nervous system, promotes tranquil sleep, from which the patient may be easily aroused, and *averts* delirium. If given in excess, either by large doses at once or by too frequent doses, it tends to produce coma. In the first case, the quantity is disposed of partly by being appropriated to the nutrition of the nerve matter, whether in centres or nerves, and

* Other examples of recovery from a comatose state in fever, under large doses of alcohol, will be found at pages 146 and 137.

partly by combustion and elimination, as carbonic acid and water. In the other case, there being more than sufficient for the normal wants of the system, a large proportion remains in the blood as alcohol, or passes into the more simple secretions, such as the fluid of the cerebral ventricles, and acts as a poison on the nerve-fibre and nerve-cell, disturbing their nutrition, so as to produce the phenomena of drunken or alcoholic delirium, or paralysing their nutrition, and inducing coma.

The coma and the delirium produced by an excessive use of alcohol are often difficult to distinguish from these phenomena, as forming part of an acute morbid process, and as indicating rather the need of a more active exhibition of alcohol. Although over-stimulation is of very rare occurrence in our practice, you will do well to be provided with some signs to distinguish it when it occurs. The following points are, I think, deserving of your attention.

1. The coma of alcohol is not so profound as that of disease; and the patient who is suffering from the former may be more easily roused than in the coma from disease. It is a good plan in these cases, as a test between the two forms of coma, to sluice the head well with cold water. The patient with coma of alcohol will much more readily respond to this than when the comatose state is part of the disease.

2. By omitting the alcohol for two or more hours, and giving nothing but water for that time, and visiting your patient after the lapse of a short period, you will find the coma distinctly less if it have been excited by alcohol. But in such an experiment as this, you must watch the patient at short intervals, and be very cautious of withholding the stimulant for too long a time. I have often known patients sink irretrievably, through timid practice, when the alcohol had been too long withheld.

3. The persistence of the exhalation of the fumes of alcohol by the breath may be regarded as a sign that too much is being administered. When the just quantity is given, the fumes are not perceived in the breath, or at most for but a short time after a dose has been taken.

I shall conclude this already too long lecture with a few additional remarks on the case upon which it is founded.

Is it quite clear that all was done in this case that ought to have been done to restore the child; that nothing was omitted —nothing done too much?

The case was complicated a good deal by the vomiting which occurred towards the close of it. I do not see, that at the early period of the symptoms more could have been done than was done. At that time, the nerve-fibre and nerve-cell were paralysed by the overdose of alcohol, and it was necessary to wait until the spirit had been consumed or eliminated. After that, there still remained a comatose state, from the want of the renewal of the ordinary nutrition. We then ventured to give small quantities of wine. Were I to treat such a case again, I would give wine or brandy more freely than was done in this instance, and I would also administer, by the rectum, quinine with a small quantity of brandy. You will find this often a very useful practice where the powers of life are low, and it ought not to have been omitted in our little patient's case. But she showed so little power of reaction, that it is in the highest degree improbable that any further treatment would have been successful.

LECTURE XVII.

ON DISEASES OF THE URINARY ORGANS.

HÆMATURIA.

The subject of hæmaturia appears a suitable one for my clinical lecture to-day, gentlemen, because there are now in the hospital four cases exhibiting that condition of the urine. These will afford us a favorable opportunity for studying some points in the clinical history of this form of hæmorrhage.

Before entering upon the detail of these cases, let me premise that hæmaturia is only a symptom, and, like all cases in which the symptoms have given rise to the nomenclature, it will draw into its consideration all those conditions which are capable, as agents, of producing it. To illustrate to you how this may arise in various ways, and to point these out, will be the principal object of my lecture to-day.

I need scarcely remind you that the amount of blood found in the urine varies from a small quantity, occasioning scarcely any discoloration of the urine, or giving rise in a greater or less degree to that peculiar smoky hue, as if the urine were mixed with soot, which is generally very characteristic of the presence of blood in this secretion, up to such a quantity as gives the urine a more or less decided red colour. When there is but little blood, the reaction of some of the ingredients of the urinary excretion darkens the colouring matter, and causes the peculiar smoky hue; but when the blood is abundant, this change of colour takes place only to a very limited extent.

Nor need I dwell upon the means of detecting and determining the presence of blood in the urine; the various tests necessary for this purpose you will find detailed in all the elementary books upon this subject. I shall content myself with remarking, that the microscope affords at once the readiest

and the surest means of determining the point in question, especially when you can examine specimens quite recently voided.*

When the practitioner has determined that there is blood in the urine, his next and most important object of inquiry is—*what is the source of the blood?* How comes it to be mingled with the urinary secretion? It may come from the fountain-head itself of that fluid, and be poured out from the Malpighian bodies simultaneously with the watery element of the urine, or it may be mixed with the urine, merely from that fluid flowing over or near a bleeding surface: thus blood escaping from any other part of the kidneys than the Malpighian bodies, or from the ureter, the bladder, or urethra, will readily mix with the urine; and lastly, in women the catamenial blood, or any hæmorrhagic flux from the uterus, will occasion hæmaturia.

The last case we may dispose of first and summarily. In all cases of hæmaturia in the female, you will of course satisfy yourselves of the condition of the uterus, and if there be uterine hæmorrhage of any kind, you will have to suspend your judgment respecting the source of the blood until the flow from the uterus shall have ceased, unless indeed there be symptoms present which clearly and unequivocally refer to the bladder and kidneys as *likewise* a source of hæmorrhage. Fortunately, the internal remedies directed against the uterine hæmorrhage are likely to prove equally beneficial when the blood comes directly from the urinary organs themselves.

The most important point of diagnosis in hæmaturia is to determine whether the blood flows from the bladder or from the kidneys; in other words, we must distinguish *renal* from *vesical* hæmorrhage. This is sometimes difficult; but frequently it is sufficiently plain. If the blood come from the bladder, there is generally some local pain on pressure, or some disturbance of the functions of that reservoir, as denoted by pain before or after passing water, or by frequency or difficulty of micturition. The blood, in cases of this descrip-

* Dr. Johnson's valuable book 'On Diseases of the Kidneys,' Dr. Odling's 'Practical Chemistry,' the late Mr. John Bowman's 'Medical Chemistry,' and Dr. Beale's work 'On the Microscope in its Application to Practical Medicine,' ought to be in the hands of every student and practitioner.

tion, is apt to form clots in the bladder, which sometimes impede micturition; it is not uniformly diffused through the urine, as in renal hæmaturia; and in evacuating the bladder, the urine comes away, at first either of its natural colour or very slightly reddened, but the last portion of it is deeply coloured, and often seems to consist solely of blood: sometimes, indeed, pure blood, either liquid or in clots, or in both forms, comes away at the end of the micturition.

Again, what are the symptoms which will lead you to think that the blood comes from the kidneys? In such a case, you will naturally expect to discover some indications of renal affection—as pain in the lumbar region, particularly if felt over one kidney; you will likewise find the blood uniformly diffused throughout the urine; there will be often, though not always, an absence of clots, the urine being alike throughout, and that passed early being just the same as that voided at the end of the stream. There is an important exception to the absence of clot in renal hæmorrhage, and that is, where the blood flowing from the kidney, independently of the urine, coagulates in the infundibula or the ureter, so as to form moulds or casts, which are afterwards forced into the bladder, and thence expelled in micturition. Such casts sometimes cause considerable pain, like that of calculus, in their passage down the ureter. Further aid in the diagnosis between renal and vesical hæmorrhage is obtained by the use of the microscope. If the blood comes from the bladder, we may see more or less of vesical epithelium mixed with the blood-particles: this form of epithelium is flat and scaly, and sufficiently easily distinguishable from that of the kidney, which would be more likely to be present when the hæmorrhage originates in that organ; the renal epithelium being more or less globular, and frequently accompanied by, and entangled in, small casts of the uriniferous tubes.

When either kidney is the source of the blood, we often find, under the microscope, casts of the renal tubes, formed of coagulated blood—*blood-casts*, as they have been called. The presence of these affords unequivocal proof that the blood comes from that source. But such a sign is seldom obtained satisfactorily.

Every now and then you will meet with cases in which you

can get no conclusive evidence to enable you to determine whether the blood comes from the bladder or from the kidney. There will be no symptom pointing to either organ, and the urine will come away freely, thoroughly charged with blood, and without clots. In such cases the probability is that the bladder is the source of the bleeding, and that the blood pours from a highly vascular fungus growing upon some part of the vesical mucous membrane, or from that membrane itself, which may be unduly charged with blood, and so tender, that solutions of continuity are easily effected in it. The continuance of the hæmorrhage generally clears up the doubts in the diagnosis, by giving time for the development of decided disturbance of the functions of the bladder.

A source of fallacy may arise in the fact that blood freely poured from the kidney into the bladder is capable of giving rise to inflammation of that organ, and a free secretion of mucus and pus causing the urine to become alkaline. The occurrence of such symptoms is very embarrassing, and demands great circumspection with reference to the diagnosis.

Having said thus much on the general rules that are to be observed in our diagnosis of hæmaturia, let me now proceed to the details of the cases to which I have particularly to direct your attention to-day.

Case XCIV.—The first case is a remarkable one. The patient, William Burrowes, aged twenty-three (vol. xxiii, p. 209), was admitted into Sutherland ward on the 2d of June, 1848, labouring under rheumatic fever; and he is now (June 13th) suffering from that disease, accompanied with severe pericarditis. Almost from the commencement of the rheumatic symptoms, this man had passed blood in his urine; and when he was admitted, his urine contained, in addition to the blood, urates in large quantity. The hæmorrhage is gradually getting less, but not until the amount of blood lost has been very great. This I consider a very peculiar case—such a one, indeed, as is rarely met with; and from the large quantity of urates (evidence of an irritated state of kidneys), and the absence of vesical symptoms, I did not hesitate to say that the blood came from the kidney.

The diagnosis of renal hæmorrhage in this case is founded on

the absence of symptoms referable to the bladder, the non-existence of clots, and the uniform coloration of the urine; and it is supported by the history of the case, which shows that at the commencement of the attack of rheumatic fever the kidneys were the seat of, to say the least, a morbid effort in the formation and discharge of lithates in large quantity, and that the patient then experienced a considerable sense of weight referred to the region of the kidneys. It is probable that both kidneys were the source of hæmorrhage, because the sense of weight was referred to both sides, and since the exciting cause of the bleeding, which I shall presently explain, is more apt to affect both kidneys than one only.

The microscopic examination of the urine in this case afforded no help, for it revealed no more than numberless blood-corpuscles and granules of urate of ammonia.

Now what can be the cause of the hæmorrhage in this instance? You may remember that I have often told you in former lectures that it was common to find, in the course of certain diseases dependent on the presence of morbid matters in the blood, more or less irritation of the glands through which these poisonous substances are eliminated. The glands especially concerned in the elimination of the poison of rheumatic fever are the kidneys; and, in the present case, the irritation of these organs has been extreme. As a result of it, there takes place a great afflux of blood to them; and if the engorgement attains a certain intensity, the delicate vessels of the Malpighian bodies give way, and the blood escapes.

There are many circumstances which prove that hæmaturia may be caused by the irritation of the kidneys, excited by a substance which can reach these glands only through the blood. It is well known that both turpentine and cantharides will irritate the kidneys, when administered in large doses. Cantharides in small doses excites the kidneys, and increases the secretion of the urine; but the excessive excitement produced by large doses diminishes the flow of urine, the state of engorgement being too great to be compatible with the healthy functions of the organ; finally, the vessels yield, and hæmorrhage is the result. The effect of turpentine is very similar. And when you examine the kidneys of patients who have died after

taking turpentine, you find many of the uriniferous tubes and of the Malpighian capsules full of blood, the precise source of the hæmorrhage being thus clearly displayed.

In such a case as that of our patient Burrowes, the kidney is brought into a similar state of exalted nutrition, or hyperæmia, or active congestion, by, most probably, an eliminative effort. A morbid matter generated in the blood (lactic acid, if we agree with Prout) makes its way out of the system through various channels, of which the skin and the kidneys are the chief. The ordinary nutrient changes of these organs (for so we must call the skin in reference to its secretory function) are thus greatly disturbed; the sweat becomes excessive and highly acid, and it acquires a peculiar and characteristic odour; the urine diminishes in quantity, increases in density, its colour becomes much heightened, and uric acid alone, or in combination with soda and ammonia, is freely generated. No doubt the kidneys (which are among the most vascular organs of the body), if examined at this stage, would be found highly charged with blood, as the affected joints are. In certain cases, then, the walls of the minute blood-vessels, having too little power of resistance to withstand the pressure of the blood, give way, and more or less hæmorrhage ensues. Such I take to be the rationale of the phenomena in the case of Burrowes.

The principal indication for treatment in a case of this sort is to promote active elimination by other emunctories besides the kidneys, and so to relieve these organs as much as possible. Thus it will be necessary to excite the action of the skin by diaphoretics; of the bowels by purgatives. Counter-irritation over the region of the kidneys may also be advantageously employed; or, if the patient is robust, a small cupping may be of use; but the strength of the patient and the amount of the hæmorrhage should always be carefully taken into account prior to the application of this remedy.

In this case we should not have been justified in taking away blood, the patient having been already greatly anæmiated. Occasionally, however, the rapid and sudden abstraction of a small quantity of blood appears to put a stop to the hæmorrhage at once. This acts, perhaps, on the principle of revulsion, or of counter-irritation. If you have recourse to counter-irri-

tants, you must be careful to employ mustard, and to avoid turpentine and cantharides, the active principles of which, even when they are applied to the skin, are readily absorbed, and may exercise a pernicious influence on the urinary organs.

In the case of Burrowes I was content to deal simply with the rheumatic fever, and not to employ any special treatment for the hæmaturia. Regarding this symptom merely as one of many local irritations caused by the rheumatic poison, I felt that the general constitutional treatment ought to mitigate the renal irritation, as it was expected to diminish that in other parts. And the result justified this view; for, as the rheumatic symptoms declined under opium and alkalies, so, *pari passú*, did the hæmorrhage. It continued very abundant for several days, and then gradually diminished, until there was no further indication of blood than the well-known smoky hue. This lasted a considerable time, and did not entirely disappear until the patient was nearly convalescent.

Let me, *en passant*, call your attention to a circumstance in this case that is especially worthy of your notice; it is the obstinacy which the rheumatic symptoms exhibited, and probably will yet exhibit, as contrasted with the case of the woman I referred to in a recent lecture on rheumatic fever, in whom the eliminatory plan of treatment was exclusively adopted. That woman lost no blood, and, although she had active inflammation of the pericardium, and the fever was of a very intense character, she proceeded rapidly to convalescence, with scarcely a bad symptom. This man has lost blood in large quantities from his kidneys; the bleeding began at a very early period of the rheumatic attack, yet it has not sufficed to keep off a severe attack of pericarditis, in which not only lymph was deposited, but a large quantity of liquid effusion was poured out, accompanied by severe dyspnœa; nor has all this loss of blood saved our patient from swollen and exquisitely painful joints, into each of which copious effusion has taken place. On the contrary, the articular as well as the cardiac symptoms have been much less tractable than usual, and have resisted the treatment employed with an obstinacy which contrasts remarkably with the readiness with which similar affections, in the case already quoted, yielded to a similar treatment. Now I do not say that

the phenomena of this case justify us in at once inferring that they were due to the loss of blood. All that I wish to impress upon you is, that the early removal of blood has not in this case had the effect of arresting the rheumatic state; it has, in truth, appeared to aggravate and prolong the rheumatic symptoms. This patient remained in the hospital till July 22d, when he was discharged quite recovered.

Case XCV.—The second case is likewise well worthy of your attention, as an instance of hæmaturia accompanying and forming the sequel to a very formidable disease—namely, inflammatory dropsy, or dropsy arising from inflammation of the kidney.

J. Pickford, aged forty, a labourer (vols. xxiv and xxiv, A), was admitted into the hospital on the 15th of May, 1848. His habits have been temperate, but he seems to have been getting into a bad state of health for some time past, as he had abscesses in his axilla and elsewhere a fortnight before his admission. Soon after these appeared, œdema came on, first in his feet, but very soon afterwards in his upper extremities and face: finally, the effusion took place in his scrotum and abdomen; and when he entered the hospital he was universally dropsical. One of his most prominent symptoms was scantiness of urine, which did not amount to more than from three to four ounces in twenty-four hours; of specific gravity 1020, very much charged with blood, and becoming nearly solid by the addition of nitric acid: so small was the quantity of urine, that for two or three days I feared that a total suppression of the secretion was likely to occur. However, on the 17th, the quantity increased to eight ounces; on the 18th, it was six ounces, of specific gravity 1025; on the 21st, seven ounces; on the 22d, it rose to sixteen ounces, nearly half the normal quantity; on the 23d, he passed twelve ounces; and on the 24th, twenty ounces. Throughout this period the urine contained blood-corpuscles in great numbers; it was free from crystals of any kind, but contained a great number of fibrinous casts and particles of renal epithelium, which left no doubt as to the blood having its source in the kidney.

The state of the kidneys in this case was probably owing to

exposure to cold, but the attack could not be traced to any particular instance of exposure. Cold is the most common cause of this state of kidney, especially if accompanied with a rapid or sudden suppression of sweat. Under these circumstances, it is very reasonable to assume that some morbid material is retained in undue quantity in the blood, which irritates the organs through which it is eliminated; of these, the kidneys suffer most, partly from their highly vascular and delicate structure, but chiefly because, in all probability, the greatest part of the morbid matter makes its way out through them. In this way an irritated and inflamed state of the kidney is induced, which sometimes terminates in the destructive disease of the organ.

The indications for treatment afforded by a case of this kind are very obvious: they are to restore the defective action of the skin—to soothe and relieve the irritation of the kidney—and to promote the elimination of water from the system.

We have in the hot-air bath a very valuable and ready means of exciting the action of the skin: this was consequently used with our patient from the beginning, and with the effect of promoting sweating, while he was in the bath, as well as afterwards. After the hot-air bath has been frequently used, it produces a state of great debility; and this constitutes the chief difficulty in continuing it, in order to gain the greatest benefit from it. In this instance I carried into effect the practice of dashing the patient with cold water immediately upon his coming out of the bath. The effect in this, as in other cases in which I have tried the plan, was certainly to give the patient a greater tolerance of the remedy, and, at the same time, by the reaction which succeeded the cold dash, to cause more active sweating on his return to bed.

With the hope of relieving the active congestion of the kidney, our patient was cupped over the loins, and several ounces of blood were taken away. I cannot say that he derived any benefit from this; and, I must confess that in the treatment of similar cases I have been more frequently disappointed than satisfied by topical blood-letting, when the congestion of the kidney was active. I suspect that as long as the morbid matter is undergoing elimination through the kidney, and keep-

ing up irritation of the gland, local blood-letting does little or no good. If a particle of dust gets into the eye, it excites conjunctival inflammation; you may leech the eye, day after day, until your patient is blanched; yet active congestion of the conjunctiva will continue; but remove the particle of dust, and the congestion will quickly subside. So with the kidney,—you will do more to relieve the active congestion of which it is the seat, by opening new channels for the elimination of morbid matter—restoring and promoting the action of the skin, and increasing that of the bowels—than by the withdrawal of blood. But when these evacuations have been some time in action, and the congestion of the kidney has assumed a passive character, then the removal of blood by cupping, or by leeching, will often succeed in relieving the congestion.

The third indication, that of promoting the elimination of water from the system, is in some degree fulfilled by the sweating process; but the use of drastic and hydragogue purgatives supplies us with a very efficient means of getting away a considerable quantity of fluid through the intestinal canal. For the generality of cases you will find that which we employed in this case the most efficient remedy of this class—namely, the compound powder of jalap: it is a safe and sufficiently active medicine. In other instances, the compound gamboge pill will suffice; or elaterium may be resorted to, but this is a much more violent remedy, and often uncertain, owing to the difficulty of procuring it in a state of purity.

After the inflammatory condition of the kidney had been subdued, and the organ began to resume its secreting activity, as shown by an increase in the quantity of the urine, the bitartrate of potass was exhibited in diuretic doses. Now, this would be apparently an unscientific plan of treatment, if this medicine be supposed to exercise any direct stimulating influence upon the kidneys. It is probable, however, that its diuretic powers may be due to some chemical or physical change which it produces in the blood, whereby the exosmose of water through the Malpighian vessels is favoured. On this view no objection can exist to the employment of this remedy in inflammatory states of the kidney; and, indeed, experience tells so much in its favour, that we should not be justified in abstaining from em-

ploying it, merely on account of an hypothesis, which may or may not be well founded.

The renal hæmorrhage in this case appears to have been, up to the time when the kidneys began to act more freely, due to the state of inflammation into which the gland was thrown; as the urine began to flow, the hæmorrhage diminished considerably, but did not cease. On the 30th of May, the secretion of urine was fairly re-established; on that day our patient passed two pints and twelve ounces of urine; on the 31st, two pints and eighteen ounces; on June 1st, three pints: after this the blood began to appear in the urine in increased quantity; and on the 6th of June, the amount of urine passed fell to thirty ounces; on the 8th, to twenty-eight ounces. And now the hæmorrhage returned with greater violence than ever, so much so that the urine seemed almost like pure blood, and contained not only multitudes of blood corpuscles, but also peculiar casts of uriniferous tubes, such as we know are thrown off in the state of chronic nephritis. The condition of our patient at this time I viewed to be as follows:—The active congestion of the kidneys has passed away; the acute stage is over, and a chronic nephritic condition has been established. Moreover, he has fallen into a *hæmorrhagic state:* he has become pale, his blood thin, and its nutritive powers no doubt greatly impaired; in consequence of which the vessels of the kidney have become weaker, and, therefore, less able to maintain their integrity in opposition to the pressure of their contents. In accordance with this view, I determined on altering the plan of treatment, abandoning all antiphlogistic diuretic remedies, and having recourse to the use of stimulants, astringents, and support. I therefore gave him plenty of nutritious food and port-wine, and applied counter-irritation, by mustard poultices; and as a styptic he has taken gallic acid, in four-grain doses, three or four times a day. This treatment seems so far to have had a very good effect; the hæmorrhage has decreased considerably, and is fast disappearing.

[It was not until the 15th of August, fully three months from the commencement of the treatment, that this patient had recovered so completely as to justify my sending him out of the hospital. The blood had quite disappeared from his urine, and

there remained but a trace of albumen. All signs of anasarca had been absent for some time.]

Case XCVI.—I shall add another case, remarkable for the amount and duration of the renal hæmorrhage, which also originated in that inflammatory state of kidney, which favours the production of general dropsy.

Francis Speight, aged thirty-five (vol. xi, p. 218), a porter; by his occupation much exposed to the changes of weather. Admitted Oct. 9th, 1844. This man got wet a fortnight before his admission, and was affected with a pretty extensive catarrh. A week after this he found his face and both arms swollen, and in two or three days the swelling extended to the legs. He now became troubled with headache, and unable to attend to his business. He was bled, but without relief, and then sought for advice at the hospital.

On his admission he exhibited all the usual appearances of general dropsy; anasarca of the legs and face, and of the upper and lower extremities; of the scrotum to a considerable extent, as well as of the abdominal integuments; and a small quantity of fluid was likewise detected in the peritoneal cavity. The quantity of urine secreted in the day was very small; it had a decided smoky hue; its specific gravity was 1025, and it became nearly solid by heat and nitric acid.

Under the use of the hot-air bath, diuretics, such as acetate of potass, digitalis, bitartrate of potass, the urine increased in quantity and diminished in density; but from being smoky it soon became increasingly red, until, on the 24th, it assumed the colour of blood, so highly charged was it with that fluid. On that day its specific gravity was 1022.

The dropsical symptoms showing no marked tendency to increase nor yet to subside, and the patient becoming decidedly blanched from loss of blood, the restraint of the hæmorrhage appeared to me to be the most urgent point of treatment. Accordingly we began the use of various reputed styptics. Acetate of lead was first tried; this very soon produced a well-marked blue line upon the gums, but no other effect. After a week's trial it was abandoned for the tincture of sesquichloride of iron, which was given alone in water, in doses of fifteen

minims, thrice a day, for four days; after this, it was continued for three days in combination with tincture of digitalis and tincture of opium. These remedies seemed to have no effect whatever in restraining the hæmorrhage. The quantity of fluid passed from the bladder (blood and urine) is described as copious; unfortunately it was not accurately measured. On the 9th of November, the hæmorrhage being as great as ever, and the dropsy undiminished, I resolved to give tannin, and at the same time to administer port wine freely. He was ordered three grains of tannin in an ounce and a half of decoction of uva ursi thrice daily, and he was allowed six ounces of port wine. He had beef tea and farinaceous food liberally. The bowels were kept open by occasional enemata, and a dose of compound powder of jalap now and then.

On the 11th the dose of tannin was increased to six grains, and on the 14th to ten grains. At this date eight ounces of port wine were allowed. The patient was quite blanched, and had a decided hæmorrhagic look, and the dropsy continued much the same.

The reports, however, soon began to be more favorable. The œdema became less; and while there was no diminution in the quantity of the urine, its colour became paler, and the quantity of blood seemed to diminish. On the 21st the specific gravity of the urine was 1007; its quantity copious, and it contained less blood and albumen. The dose of tannin was now increased from ten to fifteen grains.

From this time to the 7th of December a steady diminution of the hæmorrhage took place; and on that day the tannin was given up; and, in consequence of the extreme anæmia of the patient, citrate of iron was administered in doses of five grains thrice a day. The anæmia was now so great that, in addition to the usual blanched appearance of the skin and mucous membranes, venous murmurs were audible, and the patient was greatly troubled with buzzing noises in his ears.

After another month's treatment of this kind the hæmorrhage had ceased, the dropsy was quite gone, urine, of specific gravity 1013, was abundantly secreted, and it contained a very slight quantity of albumen. He had gained so much in general health that I consented to his leaving the hospital on the 4th of

January, after a sojourn in it of nearly three months, during two of which he was passing blood in large quantities from the kidneys.

It is important to mention, in concluding the narrative of this case, that the patient became an inmate of the hospital three times in the four subsequent years for various complaints, and that opportunity was thus afforded for examining the urine and investigating the state of the kidneys, and that on each and all these occasions no evidence of disease of the kidneys could be obtained. The urine was found to be perfectly normal.*

Case XCVII.—A fourth case is that of a boy named James Taylor, aged four years, who is labouring under dropsy after scarlet fever (vol. xxiii, p. 214). I call your attention to it now because it affords another instance in which irritation of the kidney, from the presence of a poison in the circulation, some of which is being eliminated through the gland, gives rise to renal congestion and hæmorrhage, and causes albumen to appear in the urine, even after the red particles of the blood have ceased to show themselves in it. We now and then find it produced in the early stage of scarlet fever, even when the eruption is well out; but in general it does not appear until the desquamative stage, *i. e.*, whilst the poison is passing out of the system. It is in the cases in which the eruption has been trifling, and has not come out freely, or at all, that the kidneys are most apt to be affected, the work of elimination being thrown chiefly upon them and very little on the skin. Often the patient may have got well through the fever, and may have had a good convalescence: he goes out, is exposed to cold, and the next day dropsy appears, bringing in its train all the other symptoms of renal congestion.

* Four years, however, after the last admission of this patient into the hospital, he again entered it, in January, 1852 (vol. xxxv, p. 130), labouring under general dropsy and albuminous urine, brought on by a severe wetting. He left the hospital free from dropsy, and with his urine slightly albuminous, after a sojourn of two months. He was again admitted in the spring of the present year (1856) for a similar attack, and nearly died from suppression of urine. He left the hospital, however, free from dropsy, but with urine still albuminous, and with indications of renal degeneration.

This little boy does not appear to have had any rash. He was admitted with general dropsy on the 3d of June, and the following history of him was obtained. During the last month his brothers and sisters have had scarlet fever; but, although constantly with them, he did not seem to take it; he has had no sore throat, nor rash, nor any other symptom of the fever, until three days ago, when his abdomen was observed to be much larger than usual, and his ankles, legs, and face swelled. On his admission, fluid could be detected distinctly in his abdomen, and the anasarca was general. The urine was very scanty, and contained blood and albumen.

Now the treatment in such cases, and which we adopted in behalf of the boy Taylor, is to be conducted on much the same principles as in the case which we have last considered—inflammatory dropsy; namely, to relieve irritation and promote the elimination of urine, and by the cautious use of warm baths to increase the action of the skin. I do not advise you to have recourse to early local bleeding in these cases; but if the congestion of the kidney linger after the greatest quantity of the poison may be supposed to have been eliminated, then you will often find benefit from the local abstraction of blood in quantities so small as not to affect the general strength of the patient. The urine continued to present the smoky hue, and to contain blood-discs, up to the 17th, although it had increased in quantity. Two leeches were then applied to the loins, and immediately afterwards the smoky colour of the urine began to diminish, and very soon disappeared. This boy was discharged cured on the 4th of July.

Case XCVIII.—The fifth instance of hæmaturia to which I shall refer to-day is a remarkable case. It was especially difficult to assign a cause for the hæmorrhage, unless it may have been vicarious of the menstrual discharge, the kidneys being already in a morbid state. The patient, Mary Parsons, sixteen years of age, having never menstruated, was admitted into the hospital May 22d (vol. xxiv, p. 208). Two months previously, blood began to appear in the urine without any apparent cause, and she experienced pains in her limbs, supposed to be rheumatic, which shifted from one part to another: from

this time she has continued to pass blood in increasing quantity, so that the urine soon came to be quite of the sanguineous colour. By the loss of so much blood she has now become blanched. A venous murmur was audible in the neck, but we did not hear a bellows sound over the base of the heart, or in the arteries. There were no vesical symptoms; the blood was uniformly mixed with the urine, and came away as freely at the commencement as at the termination of micturition, nor did the urine contain any clots.

The uninterrupted discharge of the large quantity of blood which this girl had been passing for so long a period, without a symptom referable to the bladder, or to any other neighbouring organ, left in our minds no doubt that the kidney was the source of the hæmorrhage. The microscope discovered multitudes of blood-corpuscles and numerous crystals of oxalate of lime. How long these latter existed we were unable to determine. Their presence in the urine betokened an irritated condition of the kidneys, which would have received full confirmation, had casts of the tubes and epithelial particles been observed.

It was difficult to fix upon a cause for this hæmorrhage. Renal calculi do not occur at so early an age. But there were two circumstances in the case which arrested attention: one was, that she had scarlet fever two years before, although we could not learn with certainty that dropsy existed; the other, that the catamenia had not yet appeared. Now it is possible that the kidneys may have been the seat of a hæmorrhage vicarious of this function, as the stomach and bowels often are, while the chronic irritation of the kidneys—the sequela of scarlet fever—may have determined the hæmorrhage to these organs. After the hæmorrhage has once been established, especially in an irritated organ, it is not difficult to conceive how it may be readily kept up: the blood becomes poor, and the vascular system weak, and a general hæmorrhagic tendency is established, while the derangement which the kidney has suffered from the original attack determines that organ as the seat of the continued bleeding.

From the long duration of the hæmorrhage in this case (two months), and the state of extreme debility to which the girl was reduced, I was anxious to improve the state of her blood as

much as possible, and to check the bleeding. She was well supported, had a moderate allowance of port wine daily, and took astringents—lead, gallic acid, tannin, the tincture of the sesquichloride of iron. The hæmorrhage, however, showed no disposition to stop; it diminished for a little while, but returned again with as much intensity as before. She is now again taking gallic acid, in large and frequently-repeated doses. If the hæmorrhage does not soon cease, I intend to try some of the terebinthinate medicines, which are supposed to exercise a styptic influence. There is no doubt that they act favorably as styptics in intestinal hæmorrhage, but in renal hæmorrhage their power is much less certain, and they are, moreover, very apt to excite irritation.*

The cases which I have now detailed to you afford good examples of a common and an important form of renal hæmorrhage, and have an interesting bearing on the general doctrines of hæmorrhage.

In all the cases, the primary cause of the hæmorrhage appears to have been an inflammatory or, at least, an irritated state of the kidney. In the case of Burrowes (Case XCIV), you have hæmorrhage due to simple irritation; Cases XCV, XCVI, and XCVII, afford examples of an inflammatory state of the kidney; whilst in XCVI and XCVIII you find an inflammatory state ending in a cachectic condition, which favours the continuance of the hæmorrhage.

These cases likewise indicate very distinctly two ways in which hæmorrhage may occur, and so far they serve to illustrate the general doctrines of hæmorrhage. Whatever produces a state of hyperæmia predisposes to hæmorrhage. That altered state of nutrition, which we call inflammation, would, therefore, take a high place among the causes of hæmorrhage. Inflamma-

* The necessity for great caution in the use of these remedies is shown by the sequel of this girl's case, which terminated, some time after this lecture was delivered, in her death. The hæmorrhage resisting all the ordinary remedies, five drops of spirits of turpentine were given three times a day. In the course of two days this remedy was followed by strangury, and a great diminution in the quantity of the urine. This was followed by a low febrile state, with oppressed brain, in which the patient died. The kidneys were in a state of chronic nephritis.

tion, consisting, as it does essentially, in an exalted *vis à fronte*, attracts blood largely to a part, and so fills the capillary vessels, in some places to the actual bursting of their walls, and the consequent escape of blood. Pneumonia furnishes a good example of this form of hæmorrhage, and the sputa in this malady acquire their rusty colour by the intermixture of the escaped blood with the mucus of the air-passages. So also an inflammatory state of kidney, by inducing active hyperæmia, causes effusion of blood, which mingles with the urinary secretion. These, then, are examples of *active* hæmorrhage. The cachectic state which follows a long-continued hæmorrhage, as in the case of Speight (XCVI) or of Parsons (XCVIII), by impoverishing the blood and weakening the coats of the vessels, impairs the inherent forces of the capillary circulation, and induces local passive hyperæmia, or passive congestion, and passive hæmorrhage, from the giving way of the weak and ill-nourished walls of the vessels.

But, in using the term passive hæmorrhage, I must beg you not to understand me as admitting a distinction which some pathologists draw, namely, that it is a hæmorrhage which takes place by the *filtration* of the blood through the coats of the capillaries without their rupture. Such an hypothesis as this I have always considered and long taught to be quite untenable: were it possible for the blood-corpuscles to pass through the coats of the capillaries, the latter would have pores, which would be visible by the microscope. This distinction I would, however, make: in *active* hæmorrhage the rupture of the vessels arises from the attraction of an inordinate quantity of blood to them; in *passive* hæmorrhage the same rupture arises not simply from the quantity of the contained blood, but rather from its depraved quality, and the ill-nourished and weakened condition of the coats of the vessels themselves, which give way on the slightest increase of pressure. And I would especially direct your attention to the fact that the hæmorrhage of the *active* form may, by long continuance, or by any cause tending to impoverish the blood and to weaken the general nutrition, degenerate into that of the *passive* kind.

Not to make this lecture too long, I shall postpone to another occasion the remarks which I have to offer on other forms of hæmaturia, and on the treatment of this affection.

LECTURE XVIII.

DISEASES OF THE URINARY ORGANS.

ON HÆMATURIA.

Gentlemen,—In the last lecture I detailed to you narratives of cases to indicate and illustrate a most important form of renal hæmaturia, namely, that which depends on an irritated or inflammatory condition of the kidney. And we also illustrated, and, I think, established, the fact that the *active* hæmorrhage excited in the irritated and inflamed organ may, by impoverishment of the blood, and by weakening of the coats of the vessels, degenerate into a *passive* hæmorrhage of a very chronic kind, which is often exceedingly difficult to cure.

It is an interesting subject of pathological inquiry to ascertain why, in the various cases to which I referred in my last lecture, the kidney should be the seat of so much irritation. These cases occur in individuals who have been much exposed to cold or wet, and who may, therefore, be reasonably supposed to have experienced a decided check to or suppression of the action of the skin. They occur notoriously in patients who have imbibed the poison of scarlet fever. These are the two conditions—namely, exposure to cold and wet, either or both, and the influence of scarlet fever—which most commonly produce the form of hæmaturia under our consideration, with its consequences—albuminous urine, and dropsy, and inflammation of the uriniferous tubes of the kidney. Cases of this kind, therefore, may be grouped together as typical of a special kind of hæmaturia. Analogous cases are met with under analogous states of constitutional disturbance. Now and then, hæmaturia, with signs of irritated kidney, but without dropsy, is met with in typhus fever. In the preceding lecture I have given an

instance of hæmaturia associated with rheumatic fever.* Irritated kidney, too, is a frequent concomitant of acute gout, and even of the gouty diathesis, although not always, nor very often, accompanied with hæmaturia; it will occur sometimes in small-pox, and I have already referred to the well-known fact, that certain poisons—irritants of the kidney—when introduced into the stomach, will pass off by the former organ, and in their escape irritate it and cause it to bleed.

With these facts before us, it seems no very difficult matter to frame a reply to the question I have suggested, namely, why, in the group of cases under consideration, the kidney should be the seat of irritation? The appropriate answer seems to me to be the following: that in these cases the blood is the seat of a foreign material, which, having an affinity for the kidney, is drawn to that organ and acts upon it as an irritant, just as cantharides or turpentine would. The scarlet-fever poison, or the poison of typhus or of small-pox, may operate in this way; and from its affinity, especially that of the scarlet-fever poison, for the gland, it may use the kidney as one of its channels of elimination from the blood. So also the rheumatic and the goutty matters irritate the kidneys, and escape from the system through these organs. And in cases of inflammatory dropsy, due to exposure to wet or cold, and perhaps checked perspiration, nothing is more likely than that, under the influence of the arrested or impeded secretion of so important a gland as the skin, some element of the perspiration may be retained in the blood, or some new compound formed in it, which may operate as an irritant poison upon the kidneys.

But, important although this discussion undoubtedly is, and glad as I should be to go more freely into it with you, I must not pursue it further, since my object in these lectures is to bring before you matters of clinical rather than of pathological interest. Still, we must on no account entirely exclude questions of pathology from our consideration when studying clinical medicine: if you do so, you run the risk of becoming blind routinists and careless and aimless practitioners; and your attention will be wholly directed to the tedious minutiæ of symp-

* Case XCIV, p. 345.

tomatology, or to the framing of tables of statistics, and you will be led to neglect the true aim of the physician, namely, diagnosis and treatment.

Let me now proceed to some other forms of renal hæmaturia. And first I shall allude to those which accompany an inflammatory state of the kidney.

There are two forms of inflammation of the kidney. In the one, the seat of morbid action is the uriniferous tubes. This is, undoubtedly, the most common and the least destructive form, and that from which recovery takes place most frequently and most completely. It is analogous to the inflammation of the mucous membrane of the infundibula and pelvis of the kidney and the ureters, which is known by the name of *pyelitis*. Of the hæmorrhage connected with this form of renal inflammation I have already given you examples in the previous lecture. The inflammation of the kidney from exposure to cold, that from scarlet fever, and from other poisonous influences, all belong to this category.

The second form of renal inflammation is that which may be called phlegmonoid. It is like the phlegmonoid inflammation elsewhere. It seizes upon a patch, or even two or more patches, of the organ, and rapidly destroys it or them, involving all the tissues—tubes, blood-vessels, connecting tissue, mucous membrane—in one common slough. This is its main character, that it rapidly passes into the sloughing and suppurative process. The separation of the slough leaves a pus-secreting cavity, whence successive quantities of pus are discharged with the urine.

You will find, in practice, that the phlegmonoid inflammation either follows the impaction of a renal calculus, which acts like an irritant foreign body, or that it occurs, much as a furuncle or carbuncle is formed in the subcutaneous areolar tissue, without any assignable cause, as if some powerful irritant and destructive agent were arrested in the circulation at the points affected.

There is nothing in pathology which seems to be more mysterious, as to its causation, than a common boil. A man, in apparently perfect health, finds a small point on his arm or thigh become irritable; he is led to examine it, and finds a minute vesicle containing a straw-coloured fluid, and surrounded by a faint blush. In a few hours this becomes hard and painful, and

by-and-by the centre, which had been the seat of the vesicle, becomes prominent, thins, and looks yellow, and ultimately a slough issues from it, leaving a cavity which soon contracts, and a cicatrix is formed, marking the position of the destroyed tissue.

It seems highly probable that when sloughs and suppurating cavities are formed in internal parenchymatous viscera, they occur under pathological conditions and with morbid phenomena similar to those of the common boil. This seems especially to apply to abscesses of the liver, of the brain, and to those of the kidney, formed as I have just described. There is, however, this difference between the sub-cutaneous boil and that of the internal viscera, that, in the former, the separation of the slough is the signal for the commencement of a contracting and healing process; but, in the latter, owing, perhaps, to the difficulty of ensuring the complete and rapid evacuation of the slough, and of the matter formed with it, this healing process does not generally take place, but the cavity left by the slough continues to secrete pus, emptying itself from time to time, as in the kidney, when it communicates with the ureter, and becoming refilled to be again emptied; or, as in the liver or brain, finding an exit through some outlet, which in the former tends often to save, in the latter generally to destroy, the patient.

Let me now remark (excusing, as I hope you will do, this digression), that the hæmaturia which accompanies this phlegmonoid inflammation of the kidney is not at first distinguishable by any special feature, as regards either the blood or the urine, from that which is met with in conjunction with tubular nephritis. Your diagnosis must be determined by the history of the case, and by general symptoms. You must keep in view that the phlegmonoid disease is of gradual formation, does not come on rapidly, is not generally traceable to any special exposure to cold or wet, and, as a rule, is never accompanied by dropsy. The phlegmonoid inflammation generally attacks one kidney only; and the pain, when it is a prominent symptom, is referred to one side, and serves to point out the seat of irritation. This is also further indicated by the presence of some degree of fulness of that region, with, perhaps, tenderness on deep pressure, especially when the loin is pressed from behind forward. When the inflammation has fairly passed into suppuration, the

hæmaturia will cease, and the blood will be replaced by pus: at first you find that the urine presents, instead of its usual clearness, an opaque appearance from the moment it is voided, the opacity being caused by pus-cells diffused in vast numbers through the urine; but, ere long, the pus accumulates in larger quantity, and falls as a heavy sediment to the bottom of the vessel, and may be easily recognised by the usual tests. The presence of pus in the urine following hæmaturia, with more or less completely the train of symptoms I have described, would render the diagnosis quite certain.

The fever which accompanies this form of inflammation is allied to intermittent or remittent fever, and simulates those of marsh origin very exactly. After this stage, a new feature sometimes occurs. A tumour is found in the lumbar region, extending upwards into the hypochondrium, and to a considerable extent downwards, sometimes almost to the iliac fossa. The tumour may be felt, and is often very defined, both through the abdominal parietes and from behind. On the left side, it may so closely resemble the spleen, as to lead to its being mistaken for that organ. Indeed, it is sometimes a question exceedingly difficult of solution to determine whether a large tumour in the left hypochondrium be spleen or kidney. The renal tumour extends lower down, and not quite so high up as the splenic. The anterior edge of the enlarged spleen is generally very defined and readily felt, and you may often insinuate your finger beneath it; it is likewise distinguished by the characteristic notch or notches. When the tumour is splenic, the percussion of the posterior part of the left lateral region of the chest is quite dull for some way up, often as high as the fifth or even the fourth rib; signs of pleuritic fluid or of condensed lung being wanting. An enlarged kidney does not rise so high into the chest; it is more free to enlarge downwards. The reasons of these differences are to be found in the normal anatomy of the two organs. The spleen lies upon the inferior ribs, and is tied to the stomach and to the spine by the gastro-splenic omentum; it is lodged quite up in the concavity of the diaphragm, and its first increase of size would be in the upward direction. The kidney is situated lower down; it is kept *in situ* only by loose areolar tissue and blood-vessels, and its highest point is lower down than the

spleen. It needs considerable enlargement to reach the concave surface of the diaphragm.

It rarely falls to the lot of the practitioner to watch a case of this kind through all its stages from the beginning; and we are forced to discover the clinical history of the malady, by putting together the scraps of various cases which come before us at different periods of the disease.

Case XCIX.—A case which I witnessed in the spring of 1855, along with Mr. Wakefield, illustrates the concurrence of hæmaturia with phlegmonoid inflammation. The patient was a very stout, full man, of plethoric and gouty habit, sixty years of age. He had had, on previous occasions, attacks of pain in the kidney and hæmaturia, and had voided calculous matter. The first symptom of his last illness was hæmaturia, with some dull pain, referred to the region of the right kidney. The hæmaturia was considerable, and under it, on the third day, he became quite prostrate, his face and tongue assumed a typhoid aspect, and he was slightly delirious. On the fourth day, in consequence of the delirium and his weakness, it was determined to administer stimulants freely. He now began to have a distinct and severe rigor every evening. The hæmaturia, it had hitherto been supposed, was due to the displacement of a renal calculus, as on former occasions. But the occurrence of these febrile symptoms with the quotidian rigor, for three days in succession, led us to expect some inflammatory affection, with suppuration, or tendency thereto. One evening he succeeded in getting out of bed, despite of the remonstrances and opposition of his attendants; and he had no sooner got on his feet then he fell in a faint, from which he could not be recovered.

The post-mortem inspection showed phlegmonous inflammation of the areolar and adipose tissue around the right kidney, and a circumscribed abscess, large enough to contain a walnut; pus was diffused through other parts of the areolar tissue of the kidney. The organ itself was very large and much congested, but showed no sign of a suppurative process; it doubtless participated in the inflammation which seemed to have begun in the surrounding areolar tissue, but it had not advanced beyond

the earliest stage of active hyperæmia. The tubes contained more epithelium than in health.

I have said enough to indicate how inflammatory irritation of the kidney will give rise to hæmaturia.

By far the most common cause of bloody urine is the disturbance of a calculus formed and lodged in the kidney. So long as the calculus remains in the position in which it was formed, there is often no symptom to indicate its existence in the organ; but, immediately it has undergone any displacement, it excites irritation of the gland, congestion, and bloody urine; or its displacement may have caused the rupture of a greater or less number of blood-vessels, from the torn mouths of which blood will escape with more or less freedom. There is no direct proportion between the amount of hæmorrhage and the size and number of the calculi. In other words, a small calculus may excite a profuse hæmorrhage, while a large one, or a great number of small ones, may escape with only a very trifling hæmorrhage. I have as many as forty oxalate-of-lime calculi, which were discharged at one effort, so to speak, from the kidney of a gentleman, and scarcely an ounce of blood was lost. On a previous occasion, the same patient had expelled five calculi, similar in size and composition, and yet there was considerable hæmorrhage for a period of three or four days.

The position which the calculus occupies in the kidney has, no doubt, much to do with the amount of the bleeding. If the stone be lodged near one of the infundibula, it may make its way into it without any material injury to blood-vessels, however large or numerous the calculi; but if the stone be lodged in one of the cones, or in the cortical substance, its displacement must necessarily lacerate the parenchyma, and cause the rupture of a great number of capillary vessels. One would expect the greatest amount of hæmorrhage when one or more Malpighian bodies are involved in the laceration.

The diagnosis of hæmaturia caused by a calculus in the kidney is not so simple as it would *à priori* appear. Although the symptoms of stone in the kidney are often sufficiently definite, there are other morbid states affecting that organ or its annexed structures which are capable of generating symp-

toms of a like kind; and it is not often easy to distinguish between them.

A slow hæmorrhage from the kidney, when the oozing of blood is sufficiently gradual to allow of the moulding of clots in the infundibula, may give rise to the symptoms of the passage of a calculus, when one or more of these clots escape into the ureter, and pass down that tube into the kidney.

Case C.—A remarkable instance of this was witnessed by me in a case of cancerous enlargement of the kidney, in a gentleman sixty-two or sixty-three years of age. The tumour, which could be distinctly felt through the abdominal parietes in the right hypogastric region, had probably existed for some time without the patient's knowledge. Although of great size, it caused no uneasiness or inconvenience whatever, and, indeed, was not discovered until an attack of hæmaturia, unattended by pain, rendered an examination of the abdomen necessary. Soon afterwards clots began to pass, and their passage down the ureter was accompanied by pain in the course of this tube; and on one occasion, a clot seemed to be arrested so distinctly at the vesical extremity of the ureter, creating great irritability of the bladder, as to excite for a time the suspicion that a calculus had become impacted at the end of the ureter. These and other symptoms were, however, soon explained by the progress of the case; and all the irritation of ureter and bladder was found to arise from the renal hæmorrhage, and from the passage of clots of blood through the ureter, and their accumulation in the bladder exciting inflammation of the mucous membrane of this organ, with copious purulent secretion. As soon as the hæmorrhage had ceased, and all remnant of blood-clots had disappeared, the symptoms referable to the bladder and kidney ceased also, notwithstanding that the cancerous tumour had steadily increased in size. On a recurrence of the hæmorrhage, the same symptoms reappeared, ceasing with the discontinuance of the bleeding; and, although the patient survived for some months the last attack of hæmorrhage, neither these symptoms, nor, indeed, any symptom referable to either kidney or bladder, again recurred. He passed urine freely and in natural quantity; nor was it possible

to discover in that fluid, by repeated physical and chemical examinations, any departure from the healthy state. At no period of the case, indeed, did the investigation of the urine afford any assistance to the diagnosis. The patient died of the effects of an extensive effusion of fluid into the left pleural cavity; and on inspecting the body, it was found that, in addition to immense cancerous deposits in the right kidney, giving this organ a size three times its natural one, there were masses of cancer in both pleuræ and in the mediastinum.

An inflammatory affection of one of the ureters may excite symptoms very much like those of the passage of a renal calculus. In such cases the urine contains more or less of pus, with or without blood; the purulent matter has its source in the inflamed mucous membrane of the ureter and bladder, or it comes from the pelvis or infundibula of the kidney, or from a renal abscess.

The severity of the symptoms in the following case seems more to be attributable to the complication of an inflamed and ulcerated ureter, than to the renal disease alone.

Case CI.—W. Denny, aged thirty-four, a butler, admitted August 9th, 1850. This man's illness began with a sudden seizure of severe lancinating pain, referred to the right loin, and accompanied by constant and intractable vomiting. Two days previous to the attack, he had observed that his urine was of a dark colour, and that upon standing, a thick sediment formed, which he described as half solid, and resembling dark-coloured *blanc-mange*. This was probably pus, derived from the kidney or ureter, or both.

Very active treatment, such as bleeding, fomentations, and various internal medicines, affording no relief to his pain or vomiting, he was removed to a large metropolitan hospital, where the bladder was sounded by an eminent surgeon and pronounced free from calculus. He remained in the hospital ten weeks, suffering from paroxysms of pain and vomiting, his urine being always thick and very offensive. His symptoms becoming mitigated, he left the hospital for the country, where they soon returned, and he was compelled to seek admission again. After a month's treatment, he was again discharged, and went to the

sea-side, when the pain and vomiting gradually diminished, the right loin remaining very tender to the touch. He continued free from any paroxysm for four years, when he found the urine become thick and offensive in smell, the pain in the loin increased very much, and he suffered from vomiting. He described the urine as being of a dark chocolate colour, thick and offensive, and depositing a reddish gravel. He now suffered from repeated paroxysms of pain, with rigors, and was forced to seek relief from one hospital to another. By one able physician he was told that he had a mulberry calculus in his right kidney. The pain was so severe at times, that he was made to inhale chloroform for its relief. After a further sojourn in the hospital for some weeks, he again went into the country, whence he returned to become an inmate of King's College Hospital with a renewal of precisely similar symptoms.

Whilst under our observation here, his sufferings appeared to come on in frequent paroxysms of most acute pain, referred to the right loin, and extending down the ureter, with violent vomiting and retraction of the testicle. The attacks seemed quite to indicate the attempted passage of a foreign body from the kidney along the ureter. But no calculus could be detected, nor was ever observed at any period of his illness to pass away in the urine, and the periods of total remission of pain, which he used to enjoy often for so long a time as three or four weeks, and since his admission into hospital for as long as a week, rendered it improbable that a calculus had become lodged in the bladder.

The examination of the urine showed that it contained pus in variable quantity, increasing with the paroxysms, and diminishing as they subsided; but no calculous matter could be discovered in the urine, or in the deposit from it, save microscopic crystals of lithic acid and some of oxalate of lime. There were no indications of blood in the urine.

Under the frequent recurrence of the attacks of pain and vomiting, with rigors, and the weakening influence of the purulent discharge, this patient soon exhibited signs of decided failure of strength. His debility was much increased by a severe attack of diarrhœa, and he gradually sunk, and died

on the 17th of October, upwards of two months from his admission.

At the post-mortem examination, the most careful search was made for a calculus; but no sign of one could be discovered. A small abscess had destroyed part of the right kidney, but that organ was not enlarged nor diseased in any other part than around the abscess. The ureter was thickened throughout the greatest part of its course, and deposits of lymph adhered to its mucous membrane. The left kidney and ureter were healthy, and the bladder was likewise free from disease.

The severe symptoms in this case could not have been due to a calculus, for no such body was found. If it had ever existed, it must have been evacuated before death; and then there would have been some marked modification in the symptoms; nor is it likely that a calculus of such a size and nature as to excite symptoms so severe as this patient laboured under would have been evacuated without attracting his attention. I am inclined to think that the severity of the symptoms was due to the inflammatory affection of the ureter; and in this view I am confirmed by a case in which symptoms of precisely the same kind occurred in connexion with a similar inflammatory condition of the ureter, proceeding from the bladder to the kidney, and extending to the pelvis of the latter organ, but not involving its substance.

The affection called *pyelitis*, by Rayer, is chiefly known as caused by the irritation of a renal calculus, and its clinical history has not been described saving as inflammation of the infundibula and pelvis of the kidney. As such cases are almost invariably associated with calculus, it is impossible to say how far the symptoms are due to the foreign body, and how far to the inflammation of the mucous membrane.

I cannot doubt that an inflammatory state of the ureter, either of its mucous membrane or of its muscular coat, or of both, occurs, generally, in connexion with gout, but sometimes independently of it, without any evidence of calculus. In one case of this kind, the patient being a man of highly gouty diathesis, there were intense pain and irritation along the course of the ureter; but there was no retraction of the testicle, and the urine,

while it presented no sign of the presence of blood, was slightly opaque from diffused purulent matter. This patient also suffered from most severe and prolonged hiccup, which, after the failure of most of the ordinary sedatives, was relieved by very free purging under the influence of a few large doses of calomel. In another case, the symptoms, although very definite, were less severe. They were pain in the course of the right ureter, without retraction of the testicle, and a muddy state of urine from diffused pus. This state of urine gradually disappeared as the local pain diminished. There was no blood. The early appearance of tolerably pure blood in a fresh state, accompanied with retraction of the testicle and pain distinctly localized, will generally indicate the presence of a calculus, and the diagnosis may be regarded as certain if the patient has passed a stone or pieces of gravel on any previous occasion. But it is well known that calculi may pass even in numbers without any material discharge of blood.

I have said enough to show you that you must not be hasty in attributing the hæmaturia which is associated with pain referred to the region of the kidney and the course of the ureter to the presence of a renal calculus. That it is so in a large proportion of cases, there can be no doubt; but you must bear in recollection that there are at least five affections which may give rise to most of the symptoms: these are—1, phlegmonoid inflammation of the kidney; 2, pyelitis, involving more or less of the ureter; 3, gouty irritation or inflammation of the ureter; 4, malignant disease of the kidney; 5, inflammation of the ureter, whether spreading to the bladder or communicated from that organ. Hæmaturia is sometimes found in hospital practice to be due to an impoverished state of blood, from insufficient and inappropriate food. In the genuine scurvy, hæmaturia is of rare occurrence, so far as I know: it is more prone to happen in those states of blood which produce purpura, or which tend to give rise to œdematous or dropsical swellings; blood impoverished in a state of constitution tainted with struma, or with some other *materies morbi*, as that of rheumatism, gout, or cancer.

The following case illustrates this form of hæmaturia coming on in the depressed state of system which accompanies erysipelas

of the fauces, and also shows how readily it gets well when the depressing influence of that poison has been removed.

Case CII.—A man, aged sixty, was admitted into King's College Hospital, suffering under well-marked symptoms of erysipelas of the fauces. The cases was well noted by Mr. Liveing (vol. xxxviii, p. 77). His illness began with shivering, fever, loss of appetite, sore throat, and some difficulty of swallowing. This last symptom increased so rapidly, that in three days deglutition was almost impossible, although the fauces were open and unobstructed. The urine was found to contain blood in considerable quantity; blood-corpuscles in abundance were seen under the microscope, and numerous tube-casts, composed of blood-corpuscles, which had doubtless escaped from the rupture of one or more of the minute vessels which are composed of the Malpighian tufts.

He was treated by the free administration of quinine and beef-tea by the rectum, wine, and the local application of nitrate of silver to the throat. No attention was paid to the hæmaturia. Four days after this treatment had been commenced, the fauces were restored to their natural condition, and the blood had disappeared from the urine.*

I must not conclude this lecture without referring to the hæmaturia which has its source in the bladder. This occurs under forms very similar to those of renal hæmaturia.

For example, you will find an inflammatory condition of the mucous membrane of the bladder a very frequent cause of this bloody discharge. This cystitis may be due to a constitutional cause, very often *gout*, or to a mechanical one, such as the presence of a calculus in the bladder, or the influence of some peculiar irritant, as cantharides or turpentine.

A second cause of cystic hæmaturia is the retention of the urine in the bladder, and the generation of carbonate of ammonia by the decomposition of the urea, which is its principal organic constituent. Such retention may arise from a stricture of the urethra, which does not allow the bladder to be ever completely evacuated by the natural expulsive effort. But it more

* See on this subject Lecture IX, page 179. This affection of the throat, which is very apt to be fatal, is not, I fear, generally known and appreciated.

frequently comes under the observation of the physician as the result of paralysis of the bladder, due in the great majority of instances to disease of the spinal cord, which likewise causes more or less of paraplegia.

It may be laid down as a general rule, that in this form of cystic hæmaturia the urine contains more or less of muco-purulent matter, assuming, when this secretion is alkaline, the peculiar viscid character which has long been known among practical writers as "*ropy mucus.*" This name, although you will find it adopted by high authorities, is nevertheless an incorrect one. The substance in question is not *mucus*; it is *pus*, altered by the presence of an alkali—altered as you often see it when in the wards we test for its presence by liquor potassæ. Under the influence of this agent, or of ammonia, the liquid pus becomes viscid, stringy, or ropy, just like the white or albumen of an unboiled egg. The reaction is probably due to the presence of oily matter in the pus, which, uniting with the alkali, forms a soap.

A third form of cystic hæmaturia is that which depends upon the presence of a fungoid growth from the mucous membrane of the organ. It is remarkable how insidiously this affection will come on. A fungus of considerable size may exist in the bladder, for a long time, without exciting any symptom calculated to arrest the attention of the patient or of his medical adviser. The presence of blood in the urine is generally the first indication that all is not right; and, in some instances, there is no other symptom than the frequent recurrence of the hæmorrhage, occasionally to a fearful extent, but always to such a degree as, sooner or later, to blanch the patient and exhaust his strength.

In other cases the fungus will excite inflammation of the mucous membrane of the bladder, and render that organ irritable and impatient of its contents, and giving rise to frequent micturition, and to the increased secretion of mucus, and afterwards of pus. In other instances, again, the expulsive power of the muscular coat of the bladder is materially impaired; and although the bladder becomes irritable, and micturition very frequent, it seldom succeeds in completely emptying itself: small quantities of urine remain, therefore, after each effort of micturition, which, undergoing decomposition, generate carbonate of ammonia, and add to the existing irritation and inflammation.

Such is the history of that formidable malady, fungus of the bladder, which I may illustrate to you by the following cases:

Case CIII.—Anne Tapner (vol. xlii, p. 149), aged sixty-seven, a widow, mother of six children. Had not suffered from any serious illness till the occurrence of the symptoms for which she sought admission into the hospital in July, 1854. I need not enter into the full detail of her previous history as obtained by my clinical clerk, Mr. Straker, who took the notes of the case, excepting to state that no evidence could be obtained of any hereditary disease in her family. The catamenia ceased at fifty.

Seven years prior to her admission she passed blood in her urine, but not to such an extent as to interfere with her ordinary avocations. Since that time she has been subject to a frequent recurrence of the bloody discharge, with intervals of three or four months of perfect freedom from blood; but the urine has been apt to emit an offensive smell, and has rarely been free from sediment. At Christmas, 1853, she had pain in the loins, which lasted for two or three weeks, and then ceased; during that time there was a copious jelly-like sediment from her urine.

The illness for which she entered the hospital began, three weeks before her admission, with severe pain before, during, and after micturition. The urine was bloody, and continued constantly so during a fortnight immediately preceding her admission. The pain in the bladder has been constant during this time, but she was free from pain in the loins. She has been obliged to pass her water every quarter of an hour, otherwise it "flew from her" involuntarily. The whole quantity passed was not more than natural. Sometimes the urine would stop suddenly in its flow, as if something blocked up the passage.

The urine for the first week of her sojourn in the hospital was of a deep-red colour, precisely that of blood; it contained several clots, each of which was as large as a nutmeg; it had a very offensive ammoniacal odour, and was highly alkaline.

The patient's complexion was very anæmic; her countenance haggard and expressive of suffering; the tongue and mucous membrane of the mouth were likewise pale, and indicative of a general anæmic condition.

I thought it expedient to have the bladder searched for a calculus. This was very carefully done, but no evidence of the presence of a stone could be obtained. The interior of the bladder felt rough, and nodulated to the extremity of the sound.

The urine deposited a large quantity of viscid matter, which adhered to the sides of the vessel (the so-called ropy mucus), and was deeply stained with the colouring-matter of the blood. This was carefully examined with the microscope, and found to contain abundance of pus and blood-corpuscles, but no trace could be discovered of any peculiar cell indicative of cancerous disease.

The lumbar regions were carefully examined, but no evidence of enlargement of the kidneys could be obtained.

From the absence of all symptoms distinctly referable to the kidneys, the escape of large round clots, and the extreme pain and irritation, being referred to the bladder, it was concluded that the hæmorrhage was from that organ, the mucous membrane of which was likewise probably the seat of considerable inflammation, indicated by the large quantity of muco-purulent secretion which came away with the urine. And as no trace of stone could be found, looking to the age and cachectic condition of the patient, and the frequent attacks of hæmorrhage with varying intervals of cessation, it was inferred that the source of the hæmorrhage was the mucous membrane of the bladder, which was probably the seat of malignant disease.

Under the use of gallic acid, with occasional doses of acetate of lead and opium, the hæmorrhage subsided very much, and the vesical pain was somewhat relieved by the use of enemata of starch with laudanum: the patient, however, continued to sink rapidly, being worn out by great pain and very frequent micturition. She died in nine days after her admission.

On opening the bladder, there was found growing, by a thick pedicle from the posterior wall and the inferior fundus of the bladder, a globular tumour, of the size of a greengage plum. On section, the basement half of this tumour was found to consist of white medullary cancer; the apex, or free portion, was of a dark-red colour, extremely vascular, and the whole surface well calculated to discharge the large quantity of blood which had been found in the urine.

The kidneys were pale and flabby, rather large, and seemed somewhat fatty; the ureters were healthy; the other organs were free from disease, but the whole body was remarkably anæmic.

It seems highly probable that this malignant tumour was of long date, and of very slow growth, extending over the whole period of seven years, during which the patient suffered more or less from vesical symptoms. I shall add another case, to show how a fungus may exist in the bladder for a considerable time without exciting any symptom to arrest the attention either of the patient or his medical attendant.

Case CIV.—A gentleman, aged thirty-five, had spent some years in India, and afterwards in Germany, living freely, and following field sports pretty keenly. In December, 1852, he suffered greatly from pain in the stomach, and such irritability of that organ that it could scarcely be brought to retain any kind of food. Three weeks elapsed before he recovered this attack. He suffered from a second similar attack early in August, 1853, while at Homburg: from this he had scarcely recovered, when he had to encounter a third similar one at Schwalbach, whither he had removed. This lasted fully three weeks. He then returned to England; and early in October, owing to some indiscretion in diet, his symptoms returned with great severity, and he suffered much from pain after eating, and incessant vomiting. He became rapidly emaciated, and put on very much the aspect of a person labouring under malignant disease of the stomach. After an illness of six weeks, notwithstanding the most judicious treatment, no improvement took place. At this time I first saw him, at his uncle's house, in one of the midland counties. I found him emaciated to the last degree, and suffering from the most distressing irritability of the stomach and constipated bowels, but no indication of any disturbance of the urinary organs. The aspect of the patient was quite enough to create a strong suspicion of malignant disease; but, on a careful examination, no tumour or other evidence confirmatory of such suspicion could be obtained. By my advice, he was limited strictly to small portions of arrow-root made with water; he was allowed to drink water in very small quantity; aperient

medicine was prohibited, and morphia with creosote was given in small and repeated doses. From this time the sickness was completely controlled, but the bowels became very much confined. After ten days, some little difficulty was experienced in micturition; but this was found to be due to the impaction of a large quantity of fæces in the rectum, the bowels not having acted during that time. On the removal of these, the difficulty in micturition ceased, and he made a steady progress to recovery. In January, 1854, he called upon me perfectly recovered in every way, and he had grown fat. From this time to the beginning of March, he continued quite well at Brighton, with the exception of an occasional slight attack of asthma, to which he was subject. On the evening of the 7th of March, he dined with a friend in London on pea-soup, smelt, and a mutton cutlet, and went to the theatre afterwards. While at the theatre, he was seized with diarrhœa, followed by vomiting, and now all his old symptoms returned—pain after food of all kinds, excessive irritability of stomach, confined bowels, and rapid emaciation. In a fortnight, he had gone back to the state of nearly complete marasmus in which I had first found him. No new symptom arose to throw any additional light upon the nature of his malady. By dint of very careful feeding with an almost exclusively farinaceous diet, and the frequent use of various sedatives, bismuth, creosote, morphia, and alkalies, his condition had begun to improve in about six weeks, when a new symptom showed itself: the bladder became irritable, and he was troubled with great frequency of micturition; and now blood began to appear in the urine in considerable quantity, giving a bright scarlet hue to the excreted fluid, and numerous clots were expelled. So decided a hæmorrhage coming on in his state of extreme emaciation and exhaustion very quickly undermined his slight remaining vital power, and he died on the third day from the onset of the hæmorrhage.

The amount of lesion of the stomach, as revealed by post-mortem inspection in this remarkable case, was scarcely proportionate to the severity of the symptoms under which the patient laboured. Nevertheless, there were some very remarkable alterations in its mucous membrane, showing that it had been the seat of a morbid process during life, which must have

occasioned considerable disturbance in the functions of the organ. It would be out of place here to enter upon any description of these, of which, thanks to the patient examination of Dr. Conway Evans, I have a very full account. Brunner's glands in the duodenum were much enlarged, as if that viscus had been also the seat of much irritation.

There was no disease of the kidneys; but the bladder was imbedded in a mass of scirrhus, which occupied the space between it and the rectum, and was of such magnitude as nearly to fill the whole true pelvis. It seemed to be incorporated with and to have sprung from the wall of the bladder. A red fungoid mass projected into its cavity, and from this oozed the blood, the loss of which had, no doubt, materially hastened the patient's death. The morbid mass was found, by microscopic examination, to consist of a fibrous network, containing in its meshes a multitude of nucleated cells, of various shapes and sizes.

It was a curious point in this case, that, until the occurrence of the hæmaturia, no symptoms of disturbance of the functions of the bladder should have presented themselves.

It is one more instance, in addition to many others which are recorded, to show how little an adventitious mass will interfere with the functions of the organs among which it is placed, when its growth is very slow and gradual. Such, probably, was the case here, judging from the extent of the tumour. It seems not improbable, likewise, that the existence of a growth like this in the pelvis, by affording some obstruction to the passage of the contents of the bowels, and perhaps also by nervous sympathy, may have tended to keep up the excessive irritability of the stomach.

There is yet one other form of hæmaturia to which I shall briefly allude. It is endemic in the Isle of France, and so common there, as I am informed, that few of the male population escape it. In the single case which I have had the opportunity of fully investigating, I came to the conclusion that the hæmorrhage, which was always very small in quantity, was derived from the bladder.

Case CV.—The young man (p. p. 1847), who was the subject

of the case to which I have referred, enjoyed excellent health, and, with the exception of frequent micturition, often to a very troublesome extent, and the occasional discharge of blood, all the functions were quite natural. He had resided for five years in the Mauritius shortly before he came under my observation, at which time he was about twenty-five years of age. The blood was always very small in quantity, never so much as to discolour the great bulk of the urine; it came with the last portion only, quite at the end of micturition, discolouring slightly, and a few drops of apparently pure blood escaping at the last. Sometimes small clots were discharged, without any definite shape. After standing, the urine would deposit a sediment of a whitish or reddish mucus. This, when subjected to the miscroscope, was found to contain bladder epithelium, very numerous octohedral crystals of oxalate of lime, with a few cells, which presented all the microscopic characters and reactions of those of pus- and blood-corpuscles. There was no appearance of casts of tubes, or of renal epithelium. The source of the hæmorrhage was doubtless the bladder, and the disease seemed to be essentially a catarrh of that organ with occasional hæmorrhage. Could the great abundance of oxalate of lime crystals in the urine have acted as an irritant to the bladder, exciting the mucous secretion as well as the hæmorrhage? Under the long-continued use of nitric acid and gallic acid, the hæmorrhagic tendency diminished, and the patient improved very much.

Let me conclude this already too long lecture by a glance at the means to be used in the treatment of hæmaturia.

It is obviously of the first importance to distinguish between the inflammatory and the non-inflammatory forms of hæmaturia, whether vesical or renal; and not less so to determine the immediate exciting cause of the inflammation, as, especially in vesical hæmorrhage, whether that cause be purely mechanical, and the inflammation kept up by the presence of a calculus.

If such a cause exist in the kidney, and excite renal hæmorrhage, the best single remedy for the discharge of blood is rest in the horizontal posture, the patient being advised to lie on the side opposite that whence the blood flows, if you are able to

determine with precision which is the affected kidney. To this may be added free dilution, the use of some vegetable astringent, dry cupping over the loins, or the abstraction of a little blood if there be reason to fear much congestion, or counter-irritation by mustard. Other treatment must be suggested by the peculiar diathesis of the patient; and it must be such as will promote the solution of existing calculi, or hinder the formation of fresh ones. And it is important also to promote the discharge of these offending substances. I can speak most favorably, from the experience of several cases, of the influence of the long-continued use of a mild non-irritating diuretic, such as Vichy water, in promoting the expulsion of renal calculi, even of those composed of oxalate of lime, which it could not influence chemically.

With reference to that form of renal hæmaturia which accompanies inflammatory dropsy, or that dependent on tubular nephritis, I need not do more than refer to the cases with which I have endeavoured to illustrate the previous lecture. The remedies to be chiefly relied on in the early periods of this affection are purging and sweating; but in the use of these, as of other reducing remedies, I cannot too often nor too strongly warn you to be cautious not to carry them so far as to induce that impoverished state of blood which is in itself favorable to hæmorrhage.

The vesical inflammation which gives rise to hæmorrhage, and which is not dependent on the presence of a calculus, is best subdued by soothing means, warm fomentations, hip-baths, opiates chiefly as enemata, hyoscyamus, and in some instances by the cautious application of leeches to the perinæum or above the pubes. But from these last means you must not expect much; they are on the whole better avoided, and ought not to be employed at all if there be any reason to suspect that gout has any share in exciting or promoting the inflammation.

The urine in these cases should be kept in a moderately acid state. It seems quite certain that alkaline urine is an irritant to the bladder, more especially if its alkalinity be due to the decomposition of urea within the organ. The best remedies for this purpose are the nitric acid and the benzoic acid. Either may be used, in combination with uva ursi or the Pareira

brava, or may be often much better given in distilled water, and may be carried with great benefit to a very large dose. The dilute nitric acid, in doses of half a drachm, in two ounces of the strong decoction of Pareira brava, three or four times a day, is an invaluable remedy in cystic inflammation.

It is after the inflammatory state has been subdued that a cachetic state is apt to supervene, which favours the continuance of the hæmorrhage. And this condition will be inevitably augmented, if the so-called antiphlogistic remedies have been previously used too zealously, and if the general nutrition of the patient has not been sufficiently upheld.

In this stage we must seek the aid of styptics; and our present knowledge affords us nothing better than the tannic and gallic acids, or the astringent vegetable infusions, decoctions, or tinctures which contain them. Either the tannic or the gallic acid may be given in very large doses; indeed, I know no limit to their use, but that which the stomach and taste of the patient will readily oppose. The former is preferable to the latter, as being more soluble; but as, in its passage through the system, it is changed into gallic acid, according to Wöhler and Frerichs, and probably would act upon the seat of hæmorrhage as that substance, the latter is the preferable drug to be administered internally. As Dr. Garrod suggests, a dose of gallic acid ought to act more powerfully as a remote astringent than an equal weight of tannic acid. In all cases of hæmorrhage, whether of hæmoptysis, hæmatemesis, hæmaturia, or any other form dependent on hæmorrhagic tendency, I have used gallic acid with the greatest advantage, and I am therefore disposed to regard it as the best styptic we possess.

Circumstances might render it advisable to inject an astringent solution into the bladder. In such a case, it would be better to use the tannic rather than the gallic acid, inasmuch as the former has the more energetic chemical action on albumen, gelatine, and fibrine.*

* Pereira, 'Mat. Med.,' vol. ii, part 1, p. 1231.

LECTURE XIX.

ON DISEASES OF THE URINARY ORGANS.

ON THOSE DISEASES OF THE KIDNEY WITH WHICH ARE ASSOCIATED AN ALBUMINOUS CONDITION OF THE URINE AND DROPSY.

I WISH, gentlemen, to bring before you, to-day, and in a subsequent lecture, a connected view of the principal points in the clinical history of those forms of diseased kidney with which are associated albuminous urine and dropsy. Both the albuminous urine and the dropsy in these cases must be regarded as undoubtedly symptoms of great importance; but the latter is of the greatest moment in the eyes of the patient.

With reference to the connection of dropsy with an albuminous condition of the urine, you will find that the cases naturally divide themselves into two great classes. Of these, the first includes those cases which are distinctly and decidedly dropsical —in which the dropsy is, at once, the most marked and the most urgent symptom, which both patient and physician watch from day to day, each alike anxious for its reduction and entire disappearance; the second consists of those cases in which the dropsy attracts but little notice, is very variable in amount, being in some cases considerable, but never excessive, while in others it may be very slight indeed, or even altogether absent; and in some instances the disease may go on for a long time, and, possibly, destroy the patient's life without giving rise to the least œdema. All the cases of both these classes exhibit the common feature of a more or less albuminous condition of the urine; there being an extensive range as regards the quantity of the albuminous element, from those cases in which the urine on boiling becomes almost as solid as the serum of the blood when similarly treated, down to those in which only the very slightest precipitate is attainable by heat, nitric acid, or other tests.

Occasionally, and I now allude more particularly to some of those instances of renal disease in which the kidneys are apt to become shrunken and contracted—a pathological condition which I described many years ago under the name of *gouty kidney*—notwithstanding the existence of most serious morbid states of the kidneys, the urine may be perfectly free from albumen. I should perhaps rather say, that it now and then happens that, after there has been distinct evidence of renal disease existing for some time, all traces of albumen may disappear from the urine. This is especially the case in the intervals between the paroxysms of the gouty attack in some of those instances of gout in which the kidneys after a time become damaged, and their secreting structure more or less destroyed. And in some of these cases, when you examine the urine, you may occasionally fail to detect the slightest trace of albumen, although at the time of your examination the kidneys may be in a decidedly diseased condition; but even in such instances, if you carefully test this secretion from day to day, you will generally find that after a time, and particularly just before or during the next attack of gout which your patient gets, albumen will make its appearance in the urine in greater or less quantity.

I must ask you to look at all these cases in a *clinical* point of view with reference to the general symptoms which they exhibit; in other words, with reference to the nature and amount of disturbance which the disease excites in the functions of the body. The condition of the circulation, especially of that in the capillary system; the position and amount of the dropsy, if it exist, and the period at which it came on; the state of the blood; the colour of the skin and the complexion; the quantity and quality of the urine, and the nature of its sediment; the condition of other organs, especially the heart, the liver, and the spleen;—these are the main points to which your attention should be directed. And from the results of such an investigation you will be generally able to form a sound estimate of the actual state of the kidneys. Add to this a careful and repeated examination of the urinary sediments under the microscope, and you will obtain data sufficient to enable you often to make your diagnosis quite certain. Let me, however, caution you against relying too much on the

information derivable from this last source, and against your beginning your investigations of the case by examining the sedimentary deposits from the urine. I have traced many mistakes in diagnosis and in practice to a too exclusive reliance on the physical or even the chemical examination of the urine: the mind is apt to acquire a bias from too eager attention to a comparatively easier and simpler investigation, which leads it to overlook or to misinterpret other important phenomena in the case. And I may add, that while it is clearly your duty not to neglect any means of observation and investigation, it is desirable that you shall be as little as possible dependent on means which are not always at hand, and which it does not fall to the lot of every eye and hand to use with readiness and skill.

In the first of the two classes to which I have alluded—namely, that which comprises those cases which are distinctly dropsical, and in which the dropsy forms an early and an urgent symptom—the disease is, generally speaking, *acute;* it runs a rapid course in its early stages, and the dropsy becomes very quickly developed. Of this great class there are two varieties, viz., 1st, the *acute dropsy* specially so called, and which generally arises from exposure to wet and cold; and, 2d, that form of dropsy which is apt to follow scarlet fever, and to which I shall allude more particularly in a subsequent lecture. You have had the opportunity of seeing many cases of this affection in the hospital during the past winter months.*

The acute dropsy is very similar in its characters to that which follows scarlet fever, and the peculiarities of the urine in both these diseases are very much alike. In both instances the dropsy comes on rapidly, and its principal features bear a close resemblance to each other in the two affections; but there is this difference between them, that in the dropsy following scarlet fever the colouring-matter of the blood is remarkably diminished in quantity, and perhaps also altered in quality, while this does not necessarily nor so decidedly obtain in the case of the acute dropsy properly so called, although sooner or later it suffers changes both in quality and quantity.

* For cases of acute dropsy, see Lects. XVII, XVIII, and XXIV; and of dropsy after scarlet fever, see Lects. XXII and XXIII.

The history of an ordinary case of acute dropsy is this:—A man is much exposed to wet and cold, or to cold alone—perhaps he has had a thorough wetting, or it may be he sleeps in the open air all night: a few days afterwards, it may be as many as twenty days, he finds that his kidneys do not act properly; perhaps his urine contains blood, which he distinctly recognises as such; or, as more frequently happens, he passes blood in his urine, but is unacquainted with the circumstance, though any one at all skilled in these matters would readily detect it. Then his eyelids and face become puffy and swollen; after a little time, the limbs, scrotum, and body generally also become œdematous; and often there is more or less dropsical effusion into the peritoneal cavity. This is the most frequent order in which the dropsy comes on; but you will meet with many exceptions to it, as it sometimes begins in the legs and the belly, and goes upwards.

The second class of cases, viz., that in which dropsy is not a prominent feature (though in some of these it, now and then, happens that the dropsy may, in the more advanced stages of the disease, become very urgent and distressing), admits of being divided for clinical purposes into three varieties, according to the quantity of urine daily excreted, together with its density and the amount of albumen which it contains. These three varieties are as follows:

1st. In some of the cases of this class, the urine is moderate in quantity, or less copious than it should be, of a high specific gravity, ranging between 1015 and 1030, sometimes even as high as 1035, rarely so low as 1010, and so highly albuminous as to become almost solid when boiled; or at least containing a very considerable quantity of albumen.

2d. In the second variety, the amount of urine passed daily is moderate, but it has a tendency to have a lower density than healthy urine—varying, perhaps, from 1005 to 1015—and it contains a moderate quantity of albumen.

3d. In the third variety of this class, the urine is generally very abundant, clear and pale, of low density—rarely above 1010, and generally even lower than this—and containing only a small quantity of albumen, which, upon the application of

heat, forms flocculent particles, at first diffused through the fluid, but which soon sink to the bottom of the test-tube, forming a flocculent or granular precipitate.

In some of the forms of this second class, the dropsy is considerable; in others, this symptom is entirely, or almost entirely, absent; but in all those instances in which there is dropsy at all, this condition is more or less *chronic*, and it is this which constitutes the essential difference between these cases and those of the first class, which are decidedly *acute* in their nature.

There is yet another condition, which may be regarded as somewhat exceptional, although I have no doubt it occurs far more frequently than is generally supposed. A person may have been passing a considerable quantity of urine containing a slight amount of albumen, perhaps only the merest trace of this substance, without dropsy, and with few or no symptoms of ill health; in short, he may have been labouring under disease of the kidney without being aware that anything was wrong, when he becomes exposed to cold or gets a wetting: soon afterwards a consider amount of general dropsy rapidly supervenes, the quantity of urine secreted in twenty-four hours undergoing a material diminution, but the albumen acquiring a decided increase. The patient now comes into a hospital, or is subjected to proper medical treatment. He is kept in bed, has a few hot-air baths, a little purging, and some non-irritating diuretic, and the dropsy subsides; and in the course of ten days or a fortnight, or perhaps a little longer, he leaves the hospital quite free from dropsy, but his urine still containing a small quantity of albumen. In the great majority of these cases, however, the dropsy is of gradual origin and slow development; but it may, of course, go on until it becomes extreme.

The following appears to have been an example of an acute affection supervening upon chronic renal disease, probably fatty disease of the kidney, of the existence of which the patient seemed ignorant.

Case CVI.—William Higgins, aged twenty-two, was admitted November 16th, 1848 (vol. xxvi, A). He was a railway porter, lived chiefly in the country, declared himself a man of temperate habits, and previously quite healthy. A fortnight before his

admission, he got wet through, and was obliged to remain all night in his wet clothes, by which he was thoroughly chilled. Two or three days after this, his face swelled; and very soon afterwards, the swelling extended to his upper and lower extremities, and to the abdomen; and from that time the dropsy steadily increased till he came into the hospital.

At the time of his admission, the dropsy was universal, and there was a considerable amount of fluid in the sac of the peritoneum. The eyelids were much swollen. The patient stated that he had been out of health for six months, and had frequent colds, to which of late he had been particularly liable; and for some time before he came to the hospital, he had passed very little water. For the week previous to his admission, he had suffered from dyspnœa, especially at night, and the quantity of urine excreted had been much diminished, and he had not perspired at all. There was no evidence of any morbid state of heart or lungs; nor in his previous history was there anything to explain the tendency to renal disease, excepting, perhaps, the exposure to vicissitudes of weather incident to his vocation.

The examination of the urine, which was frequently made during the first week of his sojourn in the hospital, gave the following result:—Quantity much below the natural amount, of acid reaction, opaline, smoky, of specific gravity 1030—1035; a deposit is formed on standing, in which are detected by the microscope numerous transparent casts, renal epithelium, several casts containing oil, *fat-cells* in abundance, crystals of uric acid, covered all over with sharp spines (urate of soda?), a very few crystals of oxalate of lime, and some blood-corpuscles.

This patient was subjected to a diaphoretic treatment, by the action of hot-air baths; he was occasionally freely purged with compound powder of jalap, and the bitartrate of potash was exhibited in half-drachm doses, and he was kept constantly in bed. Under this treatment the dropsy quickly diminished; on the thirteenth day it had all disappeared, excepting some œdema of the thighs and ankles; the ascites was quite gone; the urine had increased considerably in quantity, had become slightly alkaline under the influence of the bitartrate, and its specific gravity was 1020. It was still highly albuminous. The urinary

sediment contained blood-corpuscles, vesical epithelium, and fat-cells, which, however, had decreased considerably.

On the seventeenth day of treatment, the œdema was limited to the ankles, the urine (of specific gravity 1018) was smoky, contained albumen in diminished quantity, and the deposit consisted of blood-corpuscles, vesical epithelium, and a few large round fat-cells.

On the twenty-fourth day of treatment, the dropsy was quite gone; the albumen also was much diminished, heat and nitric acid rendering the urine simply opaque. A few casts containing oil, which had disappeared for nearly a fortnight, were now found again in the deposit, with fat-cells and blood-corpuscles.

On the twenty-seventh day, he was directed to take ten minims of the tincture of the sesquichloride of iron three times a day, and he was put upon a diet which excluded as much as possible all food tending to produce fat, consisting of lean meat, a very small quantity of green vegetables, and water, with six ounces of bread. After two days he gave up the bread of his own accord, and was allowed fifteen ounces of meat in the day.

He did not remain more than five days in the hospital; probably not liking the diet. Under it the urine became paler in colour, of specific gravity 1025, slightly albuminous, with very little deposit, which contained some vesical epithelium and a very few fat-cells. There was no trace of dropsy.

In this condition he left the hospital: the primary chronic state of renal disease probably remained, but the acute condition had disappeared. It would have been interesting to have traced the subsequent history of this patient; but nothing has been heard of him since—a period of seven years.

From what I have said, you will at once perceive that the clinical features in these cases exhibit a good deal of variety. The dropsy varies in amount, and in the time and manner of its invasion; the urine differs very much in the several cases as to its quantity, its specific gravity, the amount of albumen it contains, and we shall see other varieties of symptoms as we go on. The question, then, naturally arises, to what are these variations of clinical features attributable? Is it possible that they may exist with only a single morbid condition of the kidneys? or are we not bound to infer that there are varieties of pathological

states of these organs, just as there are varieties of trains of symptoms to which they give rise? That there are different varieties of morbid conditions of the kidneys, is a reasonable inference on *à priori* grounds; but after a careful consideration of all the facts of the case—of the variations in the clinical features, and of the different characters of the urine, which the cases themselves present—this conclusion cannot, I think, be doubted. And, as it seems to me, it is rendered quite certain by a reference to the morbid anatomy of the kidney, from which we learn that at least two essentially different conditions of this organ are associated with the clinical phenomena which I have described;—the one, a state in which the organs acquire a greater or less *increase* in size and weight; and the other, in which they present the appearance of having shrunk more or less, and in which they experience a *diminution* in bulk and weight.

Of the first of these classes there are at least two forms: one, in which the pathological condition of the kidneys comes on very quickly, while the symptoms to which it gives rise are consequently *acute;* the other, in which the morbid process is very gradual, and the clinical features which it produces are essentially *chronic.*

Under acute enlargement of the kidney, I would class that state which is concurrent with the acute dropsy consequent on exposure to wet and cold, and also with the dropsy which follows scarlet fever. In both these diseases, the kidneys, when examined in the early stage, exhibit enlargement, though this is never very considerable; they are larger and plumper than healthy kidneys, and contain much more blood and epithelium (the uriniferous tubes being filled with epithelial cells, shed, perhaps, by a process somewhat analogous to that of cutaneous desquamation) than are found in these organs in a state of health. It is very difficult to distinguish the kidney of a patient who has died of acute dropsy from exposure to cold, from that of one in whom death has occurred in the dropsy which succeeds scarlet fever; but, generally speaking, the whiteness of the cortical portion of the organ, and the extreme redness of the medullary cones, or, in other words, the distinction in colour between the cortical and pyramidal portions, although well-

marked in the former malady, are much more so in the latter. Indeed, I would venture by these anatomical characters of the kidneys alone to pronounce upon the nature of the disease from which death had resulted.

Under the head of acute enlargement of the kidney, I may refer to that which occurs from phlegmonoid inflammation of the organ. This often reaches a great size, such that the kidney can be felt through the integuments. It is the more apt to occur when two or more lobules are the seat of inflammation. I shall not dwell on this, however, as it does not belong to the forms of disease which are specially associated with albuminous urine.*

Under chronic enlargement the kidney acquires a considerable increase of size, to the extent of being one third or one half as large again as the natural gland. There are two kinds: one being that which first attracted the notice of Dr. Bright—the large, mottled kidney, the peculiar features of which the discoveries of late years have shown to depend upon the deposition of fatty matter in the epithelium of the uriniferous tubes; the second, in which the enlargement is due to the deposit of a waxy-looking, fibrinous material, also in the uriniferous tubes, giving rise to what has been termed *waxy degeneration*. This condition is shared by the kidney in common with the liver and spleen, and all these glands generally acquire their increase of size simultaneously. The enlargement in both these cases is exceedingly chronic, but we have not as yet, I think, accumulated sufficient evidence to enable us to state how long it will take to make either a large fatty or a large waxy kidney. My own experience would incline me to think it a matter of many months, and often of some years, before the deposit takes place to so great an extent as to interfere materially with the normal functions of the organ. Indeed, a considerable amount of deposit may exist in the kidney without greatly interfering with its functions. Many of you, no doubt, will remember a patient who was in Fisk ward, a few months ago, in whom there appeared to be good reasons for believing that extensive waxy degeneration of the kidneys, liver, and spleen existed (the two latter organs

* See Lect. XVIII.

could be felt very much enlarged). This man enjoyed a tolerable share of health, and was, in fact, able, after some weeks, to leave the hospital; since which time, I regret to say, we have been unable to hear anything of him.

By-and-by, when you come to deal with these diseases in patients among the wealthier classes of society, who are enabled to surround themselves with all the means and appliances which are essential to their comfort, and which aid much in counteracting or retarding the effects of disease, you will be more struck with the prolonged duration of these maladies, and the wonderful manner in which a fair enjoyment of life may be attained under a condition which produces a daily drain of albumen in greater or less quantity. The patients who come before us here are very differently situated: of many we lose sight; but of many we see the end hastened by exposure to hardships and cold, and by intemperate habits. Let me, in conclusion, relate to you the details of a case illustrative of each of these forms of chronic enlargement of the kidney. And, first, of the fatty enlargement.

Case CVII.—Charles Tiedemann, aged fifty-five (vol. xxxv, p. 124), a copperplate printer, and by the nature of his vocation subject to great vicissitudes of temperature. He denies being an intemperate man, but admits that his habit has been to drink about two quarts of porter and a quartern of gin daily. Has never had rheumatism or gout, nor have any of his family suffered from either of these complaints.

About three months before his admission, he observed that his breathing was not so good as usual, that he would puff on making any exertion, and that he could not lie down in bed without some disturbance of his breathing. His feet and legs began to swell soon after this, and the swelling gradually extended to the thighs and scrotum; the urine became scanty in quantity. After six or seven weeks' medical treatment, the action of the kidneys was slightly improved as regards the quantity of urine excreted; but the other symptoms not yielding, he obtained admission into the hospital on the 28th of January, 1852.

When this man entered the hospital, he was found to be suffering from a general condition of anasarca, which chiefly affected

the upper and lower extremities, and the integument of the trunk, especially that of the chest. The anasarca was considerable, and caused tension of the skin, which pitted on pressure. It varied in amount from one side to the other as the patient lay on the right or left side, and was always greatest in the most dependent parts. On palpation of the abdomen, it was evident, from the sense of fluctuation, that there was fluid in the peritoneum. The venous system in general, especially that of the neck, was full, and appeared to indicate some obstruction to the free return of the venous blood to the heart. The sounds of the heart were natural, but feeble. A slight rhonchus was audible in the anterior parts of both lungs; but there was no other indication of disturbance of these organs. There was no evidence of enlargement of the liver; but the presence of ascites, although not in large quantity, favoured the suspicion that the circulation through that organ was not quite normal.

So large an amount of dropsy, and of such long duration, affecting the upper as well as the lower parts of the body, denoted *primâ facie* disease of the kidneys. This suspicion was confirmed by the absence of other causes likely to conduce to such a result, such as disease of the heart or of the liver; and it became a certainty after we had investigated the condition of the urine.

It was found that the quantity of this fluid excreted in twenty-four hours was much below the normal amount, being not more than a pint, of specific gravity 1017, acid, loaded with albumen, as shown both by heat and nitric acid. It had the smoky colour which characterises urine into which the colouring matter of the blood has found its way in very small quantity, and when allowed to stand it deposited a light flocculent precipitate at the bottom of the glass.

At various times this precipitate was examined by the microscope, and found to contain blood-corpuscles, which were most numerous when the urine exhibited the smoky hue; numerous granular casts, some of which were of great delicacy, other casts containing oil-globules and oil-cells entangled in them; fat-cells, containing large oil-globules, urate of ammonia, many small crystals of uric acid, and some epithelium. In all the examinations, the oil-globules and particles of fatty epithelium

were present; but the blood-corpuscles were frequently absent, and latterly they were only very few in number when they did appear.

On one occasion, Dr. Beale made an examination of the urine, with a view to the separation of the fatty matter. Twenty-four pints of urine was the quantity operated on, and the amount of fatty matter separated was found to weigh ·47 of a grain. This, Dr. Beale states, consisted almost entirely of cholesterine, which was readily obtained in a crystalline form.*

It would serve no good purpose to go through the daily reports on this case, which extended over a period from the 58th of January till the 25th of September. During the whole of this time the prominent symptom was the dropsy, which from time to time yielded to the influence of the various remedies adopted, but again increased as the power of the drug employed declined. He suffered likewise from many other trying symptoms. Of these, the first in importance was a variable amount of dyspnœa and cough, accompanied with a viscid expectoration. These were, probably, due to more or less irritation of the bronchial tubes, and a somewhat œdematous state of the lungs. Both lungs were affected equally. Like the dropsy, these symptoms yielded from time to time to the influence of remedies, and rose or fell with the fall or rise of the power of the remedy.

On the 1st of June, our patient was seized with a sharp pain in the right side, beneath and to the right of the mamma, and a friction sound was distinctly heard over the painful spot. Under local counter-irritation and increased diuresis, induced by infusion of digitalis and the spirit of nitric ether, this symptom disappeared in a few days. At various times he complained of muscular or neuralgic pains, sometimes referred to the joints, especially the knee-joint of the right side; and he often suffered from pain in the loins, in the region of the kidneys.

These pains, and the bronchial irritation and pleurisy, were doubtless due to one and the same cause—namely, the depraved state of the blood, and the presence in this fluid of some

* 'Archives of Medicine,' vol. i, page 8.

abnormal material, which so far deranged the nutrition of the nerves, muscles, and the pulmonary mucous membrane, as to induce enough irritation to excite the symptoms described.

A large proportion of our patient's suffering was due to a highly irritable state of the stomach. The least provocation brought on nausea and vomiting. The appetite was very defective, and the amount of food taken very small. Solid food, especially of an animal kind, was not borne by the stomach. On one occasion he craved very much for some mackerel, and I was induced to allow it him; but he suffered for many days afterwards from nausea and vomiting, the oily fish having probably proved an additional irritant to the gastric mucous membrane.

This man exhibited another remarkable symptom, but one of rather unfrequent occurrence in similar cases — namely, a partially amaurotic state, limited at first to one eye. Our attention was called to this symptom on the 7th of June, and the following note was made: "At this time the dropsical swelling of the trunk is considerable, and the face puffy. On examining the eyes, each cornea exhibits a large and well-marked arcus senilis. The sight of the right eye is much impaired, and everything appears of a dusky colour and indistinct. This affection of his vision came on as a dark spot before the eye; next day this was surrounded by a red circle, which has since died away, leaving everything dusky. The left eye seems sound. Both pupils contract under the influence of light." This symptom continued unchanged until three hours before his death, some months afterwards, when he became totally blind.

To what are we to attribute this amaurotic state? Is it an affection of the retina or of the brain? Its limitation to one eye, in the first instance, indicated an affection of the retina of that eye. There was no other cause for the blindness as far as regards the eye itself, for its dioptric media were essentially sound. The first appearance of the impaired vision as a dark spot was like one of those subjective phenomena to which changes in the state of the retina are apt to give rise, and was no doubt caused by a disturbance of nutrition, dependent upon the abnormal state of the blood.

The nausea and vomiting were also probably caused by an irritated state of the gastric mucous membrane, arising from the diseased blood. It is, indeed, to this poisoned state of blood, resulting from the formation or retention in it of substances which would have found a ready exit through the kidneys, had they been in a sufficiently healthy state, that most of the symptoms from which this patient suffered were due. In this respect all forms of diseased kidney are much alike; all tend to produce similar secondary phenomena; whether large or small, the diseased organ fails to excrete the normal elements of the urine; and these, or certain of them, accumulating in the blood, affect all the tissues and organs more or less, and at length exert their baneful influence on the nervous system, disturbing its functions in the most serious manner.

Our patient remained under observation for eight months. Dropsy, varying in amount, was always present; and to diminish this, and promote the action of the kidneys, was the aim of our practice. Those students who watched this case throughout had a good opportunity of witnessing the trial, in succession, of various diuretic remedies. Those which seemed to exercise the best influence were the infusion of digitalis, the bitartrate of potass, the benzoate of ammonia, and lemon-juice. The compound powder of jalap and the compound gamboge pill were often had recourse to, as drastic purgatives, with the view of carrying off water from the system.

These remedies were employed with very variable success. At length the dropsy seemed to gain ground in a very decided way. The scrotum became enormously swollen, and the lower extremities likewise. A small slough formed at the bottom of the scrotum, from which in a few hours two pints of fluid drained away, affording great relief to the extreme tension of this part. After this the fluid continued to flow only in very small quantity.

As the dropsy was increasing, and the quantity of urine secreted was rather on the decrease, and the patient exhibited some degree of drowsiness, it was determined to give the extract of elaterium. It was administered in the form of a pill, in doses of a quarter of a grain every morning: it created some purging and great sickness. It was now tried in combination with

hydrocyanic acid; its emetic action was thus partially restrained without impairing its purgative influence, and it was borne well for the greater part of thirteen days, and a large quantity of water flowed away per anum. Towards the end of this time it again began to create very troublesome vomiting, and produced so much distress to the patient that it was found necessary to give it up.

A new drain was now established from a wound in the left leg, which was caused by the patient accidentally striking his leg against an iron bedstead. A slough formed in this spot, and from it a large quantity of fluid drained away: after several days, I thought it advisable to enlarge the wound by an incision, and thus give free exit to the accumulating fluid.

Notwithstanding all this, the dropsy increased during the month of August and the first days of September. On the 6th of the latter month, his breathing became very much affected. Moist crepitation and rhonchus prevailed over both lungs. The patient was unable to lie down. The dropsy was everywhere much increased; and the urine was highly smoky, and became almost solid upon the addition of nitric acid.

An incision of about an inch and a half in length was made through the skin and areolar tissue of the right leg, behind the inner malleolus. From this a large quantity of fluid drained away; between two o'clock on the afternoon of September 7th, and the same hour on the 8th, as much as three pints nine ounces were collected. The fluid thus obtained was clear, of a pale-yellow colour, specific gravity 1010, slightly acid, but very soon becoming alkaline. The addition of nitric acid rendered it almost solid. During this period the kidneys excreted two pints of urine, specific gravity 1025. The effect of this great drain of fluid was, that on the 8th, the day following that on which the incision was made, the dropsy was everywhere much reduced, and the chest-symptoms were very much relieved—indeed, they had almost disappeared.

On the 9th three pints, and on the 10th, two pints of fluid oozed from the leg, the dropsy became still further diminished, and the chest-symptoms did not show any disposition to return; but the patient was evidently weaker, and the areolar tissue near the incisons had become erysipelatous and sloughy. Fluid

continued to ooze in diminished quantity from both legs, and large sloughs formed. The strength gradually failed. On the 24th he became quite blind, and about twenty minutes before three o'clock his left arm became convulsed. At three he died, the intellect remaining clear to the last, but he was very drowsy for some time before death.

The post-mortem examination was made on the 25th, about twenty-four hours after death. The body presented the appearance of great general anasarca. The belly was the only part which was examined.

Upon opening the abdominal cavity, the intestines were observed to be universally of a leaden hue, and covered more or less completely with layers of rough lymph, which had all the appearances of having been recently deposited. The peritoneal sac contained a considerable quantity of a somewhat turbid serous fluid; and towards the region of the liver there was about a teacupful of healthy-looking pus; but whether this was free, or whether it was contained in a kind of thin sac of false membrane very easily broken down, was doubtful. The liver itself was apparently quite healthy, as also was the spleen, except that the last-named organ was somewhat larger than natural. The kidneys were large, and presented exceedingly good specimens of fatty degeneration of these organs, exhibiting, under the microscope, the usual characters of this form of renal disease.

Of the waxy enlargement of the kidneys the following case affords a good illustration:

Case CVIII.—Charles Adams, aged twenty-five, a tailor, admitted into Fisk ward (vol. xlii). He was born in the country, but has passed the last seven years of his life in London. When he first came to town, and for the four succeeding years, his habits were highly intemperate, for he usually drank seven or eight pints of porter in the day, in addition to ten or a dozen glasses of gin at night; but at the end of this time he took it into his head that so much drinking had affected his constitution, and he therefore left it off entirely. About this time, *i. e.*, three years prior to his admission into the hospital, he contracted gonorrhœa and syphilis, for the latter of which affections he was put under the influence

of mercury, by which his mouth was kept sore for some time. Notwithstanding this, the chancre was followed six months afterwards by secondary symptoms; which, under sarsaparilla, soon got well. Eight weeks subsequent to this, one of his testicles became much swollen; and this affection was so severe as to cause him to lie up for more than two months. Last winter he suffered much from what he calls *rheumatism*, but which rather appears to have been some further consequence of the syphilitic poison. At this time both knees and and ankles were slightly swollen; and he suffered very much from pain in the forehead, and in the bones of the legs and fore-arms. This pain was always worst at night, and the affected parts were exceedingly tender to the touch.

Soon after he recovered from the inflammatory affection of the testicle, *i. e.*, about eighteen months before his admission into the hospital, he was one day seized with violent pain in the belly, extending from the right hypochondriac region across to the left. This pain he describes as having had the character of that which attends cramp, and after its first occurrence he noticed that his abdomen seemed to be larger than before. The pain continued to recur in paroxysms daily for a week, and the abdomen appeared to swell more and more after each attack; and at the end of this time it attained the size which it had on his admission. He appears to have suffered much from looseness of the bowels at this time; but he states that he has never been jaundiced, and that he never noticed any puffiness or swelling of his face. His urine has generally been high coloured, and he has usually passed about the normal quantity.

He is a pale, sallow, rather light-complexioned man, exceedingly thin and emaciated, having a slightly œdematous condition of the legs, and a belly of considerable size. A careful examination showed that the increased size of the belly was caused by an enormous enlargement of the liver—this organ appearing to extend quite down into the iliac region on the right side, and a considerable way into the lumbar on the left—and also by the presence of a moderate amount of fluid in the peritoneal cavity. There is much tenderness all over the region of the liver; the superficial abdominal veins are distended;

there are no indications of nodes in any part of the body. Heart and respiratory sounds normal; the bowels are much relaxed, and he states that a short time ago his motions had very much the appearance of plaister of paris; pulse 76. Urine rather more than two pints, of the natural colour, clear and transparent, of acid reaction, of specific gravity 1012; depositing on standing a slight, cloudy sediment, and highly albuminous, so as to become almost gelatinous when boiled. The sediment, when examined under the microscope, is seen to be composed of faint, transparent, fibrinous casts, of large size, in very small number, with still fewer casts of a waxy character, of small size. In addition to the waxy-looking casts above mentioned, what appear to be the fragments of broken-up epithelial cells, together with a few cells much resembling those of pus, are seen scattered over the field. Occasionally some of the above-described epithelial *débris* can be seen entangled in a tube-cast.

Without reading you all the details of this case, which extended over a long period, I shall confine myself to stating that this patient remained in the hospital for nearly seven months, when death took place. The symptom from which he suffered most was diarrhœa, which in a greater or less degree was almost constant during the whole of this time, and which nothing appeared to hold in check for more than a few days.

The quantity of urine passed varied from a pint to more than four pints in the twenty-four hours, the average being about two pints, and the variation being, in some measure at least, dependent on the amount of aqueous fluid discharged by the bowel. The amount of albumen in this secretion varied slightly from day to day, but was always great, the urine when boiled often becoming almost solid; the density of the urine ranged between 1010 and 1016, the average being 1012. The sediment deposited by the urine, even after standing twelve hours, was always exceedingly scanty; and, although very often examined, it invariably exhibited, both to the unaided eye and with the help of the microscope, the characters above recorded, being always remarkable for the paucity of casts it contained, a careful examination on some days failing to detect even a single one.

At first this patient was treated with krameria and opium,

and for some time an ointment containing mercury and iodine was rubbed in over the region of the liver; then sulphate of copper and acetate of lead, each combined with opium, were tried in turn, but without any material benefit resulting. After a time, the compound infusion of catechu was given, and enemata of starch and laudanum regularly administered; these, too, failing to check the diarrhœa, infusion of matico was had recourse to, and opium suppositories were introduced into the rectum. Tannic acid, also, was exhibited, but without producing any good effect; and the only thing which materially relieved the purging, though this was only for a few days, was the docoction of Indian bael.

On the whole, perhaps, had it not been for the diarrhœa, this patient would not have suffered very much during the greater part of his stay in the hospital; but he occasionally had attacks of severe pain over the region of the liver, which were usually relieved by sinapisms; once a blister was applied in this situation. After he had been under treatment some time, it was noticed that his motions, which, though always watery, often varied much in colour (being sometimes almost white, at others very dark, and generally horribly offensive), contained some joints of a tape-worm. Some oil of male fern was, therefore, given him, and the parasite (five or six yards long) was expelled, but without in the least degree lessening the diarrhœa.

At length the œdematous condition of the lower extremities increased, and the enlargement of the abdomen also became greater, while the amount of urine passed occasionally fell as low as a pint in the twenty-four hours; and whenever this occurred, he became drowsy and sleepy, and evinced a tendency to fall into a comatose condition, from which state, however, he on two or three occasions roused up again. Under these circumstances, we endeavoured to promote the action of the kidneys by the exhibition of diuretics; and those which we employed for this purpose were the bitartrate of potash, the compound decoction of broom, and the benzoate of ammonia—all with more or less success. The diarrhœa, however, baffled all our efforts, exhausting the patient to the last degree; and on December 5th, he was seized with an epileptic fit of a most severe character, which lasted more than three hours, and then

left him in a comatose condition; but from this he roused again the following day. From this time, however, he gradually sank, and died, worn out and exhausted, on December 18th.

The following is the report of the examination of the body of this man, as drawn up by Dr. Conway Evans:

The body was examined on December 20th, about thirty hours after death. It was greatly emaciated, and the lower extremities were highly œdematous.

On opening the belly, the peritoneal cavity was found to contain a considerable quantity of a clear serous fluid, and a liver of enormous size. The latter was of a light fawn colour, and of a waxy appearance; its surface quite smooth, but undulating, and studded with occasional yellowish-white spots which varied in size from that of a pin's head to that of a cob-nut. The capsule of Glisson was not in the least thickened, but, on the other hand, perfectly smooth and transparent. On taking out the liver, this organ was found to weigh 8 lbs. 6½ oz.; and its cut surface presented an irregular intermixture of dirty white and red, with great numbers of yellowish-white (not encysted) tumours of a cheesy consistence scattered throughout its entire substance. These tumours were, generally speaking, larger than those which were visible on the exterior of the organ, and varied in size from that of a pin's head to that of a walnut. The thinnest sections which could be obtained, when subjected to microscopical examination, were invariably found to be exceedingly opaque, so as to render the exact structure very difficult of determination. All traces of cell-radiations, or rather of radiating ducts with their contained cells, were (apparently) almost obliterated; while the bulk of each lobule between the interlobular plexus and the intra-lobular (hepatic) vein appeared to consist of masses of pale, whitish, unorganized, fibrinous-looking matter, with here and there patches of oil-globules. This appearance was, no doubt, due to numbers of the liver-cells lying in many planes, and superimposed, as it were, one over the other viewed *en masse*. The liver-cells generally were very pale, smooth, and white, and of very small size—much smaller than those of a healthy liver—and their outline was very indistinct. Most of them were entirely devoid of oil, and none of them had

anything of a bile-tint; nor did they contain any granular matter, as in health. The nuclei in most of the cells were also indistinct, and, indeed, in a large proportion altogether invisible. Some of the cells, on the other hand, were crammed with oil. The whitish tumours, when examined under the microscope, were found to consist of finely-granular amorphous matter, interspersed with numerous dark-looking oil-cells.

The stomach and small intestines appeared healthy. There was no ulceration, thickening, redness, nor apparently any other morbid condition of Peyer's glands; nor could any portion of a tape-worm be discovered. In the large intestine, the mucous membrane generally appeared to be somewhat thickened, and the solitary glands were here slightly enlarged. The spleen was of large size and exceedingly firm consistence: it weighed 15 ounces. The lungs appeared to be healthy; they were freely crepitant throughout; there were no pleuritic adhesions. The heart was exceedingly small, and very flabby; it weighed barely 5½ ounces; all its walls appeared equally atrophied. The fibrous tissue of all the valves (on both sides) was somewhat opaque; in other respects the valves seemed healthy. There was no atheromatous state of the arteries.

The kidneys were very large; equal in size, and alike in appearance; one of them weighed rather more than 8 ounces. The capsule was readily separable, and the surface was then seen to be of a pale fawn colour, smooth, and waxy-looking, but raised into largish, though not prominent, undulations. The lobular markings were entirely obliterated, but there was nothing of the granular appearance of a *granular fat kidney*. When examined under the microscope, the tubular structure could be made out only with considerable difficulty; and in some places, indeed, this was quite impossible. All the epithelium which could be seen appeared very far from normal, of a yellowish colour, and very flat, and looking as if shrunken, though still firmly adherent to the tubular walls; but from a great number of the tubes all traces of epithelium had entirely vanished, nothing remaining save the basement membrane and matrix, which were highly glistening, and often appeared to be thicker than natural, and in many parts studded with minute

globules of oil. None of the tubes appeared to contain any of the fibrinous substance which was observed in the urine during life; neither was there any appearance of desquamation nor of crumbling of the epithelium, so as more or less to fill up the tubular cavities. The Malpighian capillaries were much thickened, and very opaque; and the coats of the minute renal arteries, particularly the longitudinal fibres, were greatly hypertrophied. In many parts these vessels were exceedingly tortuous; and occasionally the canal of a minute afferent artery could be seen filled with a mass of highly refracting globular particles (oil, or phosphate and carbonate of lime, or both), and the Malpighian body to which it led atrophied and shrunk up.

In conclusion, let me again remark, that it seems to me quite clear, looking simply at the organic alterations which the kidneys themselves undergo, that the several pathological conditions of these organs which I have just mentioned to you cannot be classed as one and the same; and when we take into account the clinical history, this conclusion seems still more likely to be the just one. What can be more different than the respective clinical histories of the case just related, and of that to which I first called your attention in this lecture; or of those cases of acute dropsy with hæmaturia referred to in former lectures, or of others which I shall presently bring before you? Can it be possible that different clinical phenomena may coincide with one and the same pathological condition?

Just as well might we affirm that all the enlargements and contractions which the liver is apt to experience are due essentially to one and the same morbid state. And I may here observe, that there is a close analogy between the diseases of the kidney and those of the liver, and that a comparison between the various diseased conditions of these two organs tends to explain their true pathology in a remarkable manner. Thus, for example, enlargements of the liver, like enlargements of the kidney, are of two kinds—*acute* and *chronic*. The acute enlargements of the liver are connected with inflammatory states of that organ, just as acute enlargement of the kidney is associated with inflammatory conditions of that organ. So, also, of the *chronic* enlargements of the liver, we have the *waxy* de-

generation and the *fatty* degeneration; both essentially analogous to similar conditions of the kidney.*

I shall proceed with the consideration of this subject in my next lecture.

* The following table exhibits the classification of the affections of the kidney referred to in the lecture, which seems most consistent with clinical observation and experience:

A.—Cases in which dropsy is urgent and acute, and albumen abundant.	Acute dropsy. Dropsy after scarlet fever.	Acute enlargement of kidney.
B.—Cases in which dropsy is not a prominent symptom—is very variable in amount—chronic—and may be absent. Albumen variable.	Chronic enlargement of kidney.	Fatty disease (Bright's kidney). Waxy disease.
	Chronic contraction of kidney.	Chronic nephritis, or chronic wasting kidney. Gouty kidney.

Although it is the rule that the fatty enlargement of the kidney remains without diminution during the life of the patient, still there are, as Dr. Johnson informs me, a few rare and exceptional cases in which this state of kidney assumes the wasting tendency, and becomes very much reduced and contracted, the urine exhibiting the granular and large waxy casts which are characteristic of that chronic state.

LECTURE XX.

ON DISEASES OF THE URINARY ORGANS.

ON THOSE DISEASES OF THE KIDNEY WITH WHICH ARE ASSOCIATED AN ALBUMINOUS CONDITION OF THE URINE AND DROPSY.

I CONCLUDED my last lecture with the details of two cases illustrating the two forms of chronic enlargement of the kidney—namely, the fatty kidney and the waxy kidney.

In forming your diagnosis, which is sometimes difficult, you will have to keep in view the distinctive features of the fatty enlargement of the kidney, as compared with those of the chronic contracted kidney and of the waxy enlargement. The contracted state is much more frequent than that of enlargement, whether fatty or waxy. The fatty condition is generally attended with dropsy, much greater in amount and of a more persistent and obstinate character than either of the others, which, unless accompanied by some diseased or enfeebled state of heart, are frequently quite free from dropsy, and generally have it only to a slight amount. The presence of abundant fat-cells, fatty-casts, and free oil, with albumen in large quantity, in the urine, would indicate fatty kidney, although, for a time at least, such a state of disease may exist without these appearances; whereas, in the contracted kidney, fat-cells or fatty casts are either not present in the urine, or exist in but small number, and only occasionally, and in its more advanced stages, and the albumen is never by any means so abundant as to render the urine nearly solid under nitric acid and heat.

The waxy kidney exhibits clinical phenomena sufficiently distinct from those of the fatty disease. Instead of the white anæmic complexion, with puffy face, which accompanies the latter malady, you will find the patient looking sallow, and, generally speaking, free from any swelling of the face. Dropsy

either does not exist at all, or is very trifling. It does not show itself until the disease has advanced considerably, and it rarely, if ever, is so prominent and chronic a symptom as in the fatty disease, nor is it often as much as in the contracted kidney. In a well-marked example of the disease, of which I lately witnessed the fatal termination, in the person of a medical man, there was no sign of dropsy throughout his long illness of two years. In most of the cases the peculiar waxy degeneration is not limited to the kidneys, but affects the liver and spleen, causing enlargement of these organs. The increased size of these viscera, therefore, becomes an aid to the diagnosis of this affection, in addition to those signs which may be obtained from the altered urinary secretion.

The condition of the urine resembles that found in the waxy disease as regards the quantity of albumen, which is generally large. But fat-cells are not found, nor the fatty casts; transparent fibrinous casts and the *débris* of epithelium are the most common appearances. But these may be absent; and in both forms of enlarged kidney this absence of all sediment is not uncommon.

There is quite enough evidence to show that this disease is a peculiar cachexia, allied perhaps to scrofula, which shows itself mainly in the diseased states of kidney, liver, and spleen. Whether the occurrence of the waxy deposit in the kidneys alone (the liver and spleen being quite free) is to be regarded as an indication that the malady is only in an early stage, I cannot say. You will find in Dr. Johnson's book an example of this disease limited to the kidneys, and some other cases illustrative of the general cachexia.*

Let me now proceed to the second class of cases, viz., that in which the kidneys experience a diminution in size, and are decidedly smaller than they shall be. The different varieties of contraction of the kidneys are due, so far as our knowledge at present enables us to state, to one and the same pathological

* On the subject of the waxy disease, see a paper by Drs. Gairdner and Drummond, in the 'Edinburgh Monthly Journal' for May, 1854, and an article by Dr. Parkes in the 'British and Foreign Medico-Chirurgical Review' for October, 1854.

condition, which, I need scarcely say, is totally different from any of those which lead to great enlargement of these organs, and which is essentially slow and chronic in its nature. In this state the kidneys often suffer a remarkable diminution in their size; they shrink up, and become more or less fissured or lobated; their capsule is generally remarkably thickened, and peels off readily; and when they are divided longitudinally, the remarkable shrinking is seen to have taken place mainly at the expense of the cortical substance, so as to reduce this portion of the kidney to the thickness of a mere rind, extending along the bases of the medullary cones. With all these characters, if the disease be sufficiently advanced, there are very often, scattered in greater or less numbers throughout the cortical substance, cysts of various sizes. These cysts vary in dimensions from exceedingly small, microscopic objects—probably, as Dr. Johnson suggests, simple dilatations of uriniferous tubes—up to such as are as large as a nut, or even larger.* The elements of the kidney can generally be found in cases of this kind; but their characters are very different from those which they exhibit in a state of health. The epithelium is seen to be more or less wasted, and to have undergone, or to be undergoing, disintegration; many of the tubes are found to be denuded, so that the basement membrane and matrix only are left: some of these denuded tubes are found to be dilated into small cysts, while others appear to be undergoing a process of atrophy, and are very much smaller than natural; the coats of the minute arteries, particularly their longitudinal fibres, are often more or less hypertrophied, and these vessels themselves frequently very tortuous (the canal of a minute afferent artery being sometimes blocked up with oil, while the Malpighian body to which it leads is wasted and shrunk up); and the Malpighian capillaries are generally thickened and opaque. In some kidneys of this kind,

* I am not prepared to enter into the *great cyst-controversy*. From all I have seen, it would seem to me that the simple and natural explanation offered by Dr. Johnson may be regarded at least as one way, and by far the most frequent way, in which these cysts are formed. But I can conceive it quite possible that cells may occasionally form in the uriniferous tubes, as suggested by Mr. Simon, which, by enlarging, produce those remarkable renal cysts or pseudo-hydatids which have so long attracted the attention of morbid anatomists.

you will find distinct streaks of a whitish material following the direction of the tubes of the cones: these you may pick out with the point of a knife or a needle, and upon examination you will find them to consist of lithate of soda, which had remained in the tubes. When this deposit is met with in the kidneys, the same salt will be found deposited, in greater or less quantity, in other parts of the body.

We meet with very different degrees of contracted kidneys: nevertheless, as I just now mentioned, they are all associated with one and the same pathological condition, and the rationale of the morbid process by which the contraction is effected may probably be explained in some such manner as the following: Some causes or other come into operation which excite disturbances of the nutritive processes to a greater or less degree, and interfere with the normal development of the blood, this fluid becomes contaminated, and some or all of the contaminating ingredients are conveyed to the kidneys to be eliminated by these organs. In their passage through these glands, these poisonous elements create a highly disturbed state of their nutrition—a state, possibly, in some degree inflammatory, but chiefly atrophic, the tendency of which is to cause the organs to waste and shrink.* The kidneys, thus injured, are rendered unable to carry off in due quantity some of the elementary constituents of the urine; and these, accumulating in the blood, become a further source of mischief, in fact, a further source of poisoning, not only to the kidneys, but also, secondarily, to almost all the other organs of the body.

Now, one state in which this train of symptoms is very likely to occur is that condition of the system which we call *gout;* a peculiar state, in which some morbid material—uric acid, per-

* The evidence of an inflammatory process having any share in the production of this state of kidney appears to me to be very unsatisfactory; and on that account I cannot adopt the term applied to this disease by my friend Dr. George Johnson, "*chronic nephritis.*" If the essential pathological change in these kidneys be inflammation, that process produces results of a very different kind to what we notice elsewhere. The pus-globules which are sometimes observed in the urine of patients labouring under the disease do not necessarily come from the tubes, but rather from the mucous membrane of the infundibula, pelvis, and ureter; and they are probably due to the irritating quality of the urine.

haps, or some compound of uric acid, or, at all events, something very nearly allied to this substance—becomes developed in abnormal quantity in the blood, operates as a poison upon the joints, and likewise irritates the kidneys, and thus tends to keep up a gradual process of retention of morbid matters in the system, which ultimately leads to the destruction of these organs. You will not suppose that I limit the causation of this contracted state of kidney solely to gout: there are many cases in which we find no trace of gout; yet there is a general constitutional condition analogous in many respects to that which gives rise to gout, where the assimilative processes are much at fault, and where the blood is ill supplied and poor.*

Let me here again refer to the analogy between hepatic and renal diseases. In the contracted, fissured, and granulated liver (the true cirrhosis—*Anglicè*, hob-nail liver), which is often met with in hospital practice giving rise to ascites, you find the analogue of the contracted kidney. The two diseases often occur simultaneously in the same person, apparently owing their origin to one and the same cause—namely, irregular and intemperate habits. Cirrhose is an atrophic condition of the liver from a deficient and depraved nutrition: many of the lobules and ducts of the organ become obliterated and wasted; much of its vascular system experiences the same fate; and here and there occur accumulations of cells loaded with fat. There are but few lobules which escape injury and retain their healthy character; the capsule of Glisson is thickened and condensed in structure, and shows a tendency to shrink, as the material of a cicatrix in the skin or mucous membrane does. The diminution in size of these livers is due partly to waste of substance, and partly to this shrinking or contracting tendency of the diseased Glisson's capsule.

Some able pathologists are content to believe that all these effects are due to chronic inflammation, and ascribe this to the direct poisoning influence of alcohol frequently indulged. Plausible as is this doctrine, I confess myself unable to assent to it; for I have no doubt the disease occurs where the habitual indulgence in fermented liquors has never existed, and unquestion-

* See Lect. XXVIII, on Gouty Kidney.

ably very many for years accustomed to the free use of such fluids escape all indications of the malady.

It is likewise the opinion of some that the contracted liver is always preceded by a stage of enlargement. This I think a very doubtful view. Without question, there are large livers, in which may be found patches affording the appearance of the hob-nail structure, and others exhibiting the fatty condition, and others again that of healthy liver. It seems to me very doubtful that such livers ever attain the condition of the contracted liver. Ascites develops itself very slowly over a period of many months; but general nutrition goes on fairly, and a good quantity of bile is secreted, the patient suffering chiefly from the bulk of the liver and the distension of the belly. I have seen cases in which such livers have become reduced in size, but never to such an extent as to be otherwise than still enlarged.

The enlargement in these cases appears to me to have been due to the fatty disease affecting parts of the liver, while other parts, either previously or subsequently, have been the seat of cirrhosis. Whether there is any strictly analogous condition of kidney with this of liver, I am unable to say with any confidence.

I am not able to illustrate the state of chronic contracted kidney with the full history of any case recently in the hospital; but I think we may derive instruction from investigating a case now under treatment, respecting the diagnosis of which some difference of opinion may and does exist among those who have watched its progress.

Case CIX.—The case to which I refer is that of a woman named Catherine Henry, now in Augusta ward (vols. xl, p. 59, and xlii, p. 88). Our patient has gone through a considerable amount of wear and tear, partly from several severe illnesses which she has had, and partly in consequence of intemperate habits. She tells us that she is thirty years of age, and married; that she has lived in London the last ten years, following the occupation of a waistcoat-maker; and she confesses that her habits have been intemperate, particularly during the last five years, her chief beverage having been brandy-and-water. Her

serious illnesses appear to have begun about three years ago with an attack of rheumatic fever, which laid her up for two months. I may here remark, that when rheumatic fever occurs in persons between the ages of twenty-five and fifty, it borders very closely upon gout in its characters, and is very difficult to distinguish pathologically from an attack of acute gout affecting a great number of joints. You have the same profuse acid sweats, and the same pain, tenderness, and swelling of the joints, in the two cases; but there is this difference between these two diseases, viz., that in acute gout there is far less liability to affections of the heart—endocarditis or pericarditis—than in acute rheumatism.

About four months after the first attack of rheumatic fever, our patient had a second; and about six months subsequent to this, a third; and from the time of the first attack of this disease, she became subject to shortness of breath and palpitation on the least exertion. Possibly there may have been pericarditis at this period, followed by adhesion between the two opposed pericardial layers. There does not now appear to be any decided valvular disease. But, although there is now no distinct bellows-sound, indicating valvular imperfection, some of you may have observed that there is some degree of roughness accompanying the first sound of the heart, and best heard at the apex. This is by no means unfrequently the case in persons who have had rheumatic fever once or twice, in consequence of the fibrous structure, which enters so largely into the formation of the valves of the heart, having lost its suppleness and flaccidity, but yet not to so great a degree as to render the valves incapable of closing their respective orifices.

Two years ago, this woman noticed that her eyelids and face were puffy and swollen, generally in the morning when she got out of bed: about the same time, too, she became subject to more frequent calls to pass her water than usual, especially at night, being disturbed frequently, and obliged to get out of bed for this purpose. This symptom, in the absence of clear indications of bladder affection, is highly suggestive of chronic disease of the kidneys. I am unable to give you any very good explanation of this phenomenon: the kidneys being assumed to be irritated, the bladder may be supposed to partake in the irri-

tation sympathetically, and thus give rise to frequent micturition; or the urine itself may be irritating to the bladder, and thus excite this viscus to empty itself frequently.

The next point in the history of this patient is the occurrence of swelling of the ankles, soon followed by more general dropsy, affecting the legs, thighs, upper extremities, and belly. The quick development of general dropsy ought always to excite suspicion of diseased kidneys, and especially when the face is involved, whether first or last. The dropsy most liable to be confounded with that dependent upon renal disease is that which arises from disease of the heart; but the distinction between them is, generally speaking, simple enough; for whereas in cardiac dropsy the parts most dependent are the first to become swollen, except under peculiar circumstances, in renal dropsy the converse of this commonly prevails, the highest parts of the body, as the face, eyelids, and scrotum, being usually the first to become œdematous.

The enlargement of the belly, as noticed in our patient, clearly arises from the accumulation of fluid in the peritoneal cavity. In the absence of any distinct peritoneal affection, this dropsy must be caused by some morbid condition of the liver; and looking at the intemperate habits of this patient, it seems reasonable to suspect such disease in her case. I have already alluded to the analogy between, and the frequent coexistence of, cirrhosis of the liver and the chronic disease of the kidneys, which ultimately leads to the wasting and atrophy of these organs. But the morbid processes in the liver and in the kidneys, though they may be similar, if not identical in their nature, may not go on *pari passú*—that is to say, one may advance much more rapidly than the other; and this is probably the case in our patient, the disease of the kidneys being in advance of that of the liver. However this may be, there seems good reason to believe that, in the early stages of chronic renal disease, a dropsical accumulation taking place pretty rapidly into the abdominal cavity is an indication that the liver is apt to be suffering from a diseased state analogous to that of the kidneys, and creating obstruction to the portal circulation.

About twelve months ago, this woman was admitted into the hospital under my care; she was then suffering from consider-

able general dropsy, which was attributed to exposure to cold, an acute attack supervening upon the more chronic disease. She remained in the hospital some time, and she was then passing from two to three pints of urine daily, varying in density from 1010 to 1015, and containing a considerable quantity of albumen. The treatment then adopted consisted at first in free purging (the hydragogue purgatives being those which were employed), and subsequently in the exhibition of the tincture of the sesquichloride of iron, as well for its diuretic properties as for its power of improving the quality of the blood. Gallic acid was also given, with the view of restraining the flow of albumen from the system. Under this plan, together with the occasional employment of the hot-air bath, she left the hospital in August (1853) comparatively well; but her urine still contained a minute quantity of albumen, though it did not exhibit under the microscope any tube-casts or other indications of renal irritation.

After leaving the hospital, she remained tolerably well, having, however, a little œdema of the feet and ankles occasionally, and "taking a little inward medicine now and then, such as cream of tartar and salts;" and following out, no doubt, her old habits of intemperance for about eight months, when a similar train of symptoms, viz., those of renal dropsy, again occurred. Patients with chronic disease of the kidneys are very apt to have dropsy develop itself rapidly if they get an attack of bronchitis, or any other affection which either embarrasses the pulmonary circulation or checks the action of the skin. The most probable explanation of this is that, in consequence of the impediment to the circulation through the lungs, or in consequence of the interference with the cutaneous secretion, the blood, already deviating considerably in its composition from the healthy standard as a result of defective renal action, becomes still further altered, and partly owing to the impoverished state of the blood, and partly to impediment to the general capillary circulation, the escape of the *liquor sanguinis* through the walls of minute blood-vessels becomes a matter of very ready accomplishment.

On May 20th (1854), this woman was again admitted into the hospital, and the following description of her then condition was entered in the Case Book:—"A pallid, sallow, puffy-faced,

rather thin woman, with black hair and grayish eyes; having considerable œdema of the lower extremities, with, perhaps, a small quantity of fluid in the peritoneal cavity; complaining of pain underneath the left breast and about the lower angle of the left scapula, and of heaviness in the head, being constantly very drowsy and sleepy; having a very distressing cough, with slight mucous expectoration, and breathing rather hurriedly (twenty-four times a minute), the pulse being 76. Much rhonchus and large crepitation are audible throughout both lungs; her appetite is bad; her tongue is coated with a thick, whitish fur, and her bowels are habitually confined. She passes daily between two and three pints of pale urine, of low specific gravity (1009—1010), which contains a considerable quantity of albumen, roughly, about one third its bulk."

Sallowness of complexion is not amongst the least significant of this woman's symptoms. It is a very frequent, almost constant concomitant of the contracted kidney; so much so, that one is often led, in passing through the wards and elsewhere, to fix on a case of this kind from the complexion alone, and with a correct result. In other forms of renal disease the complexion is different: in the dropsy after scarlet fever the face is swollen and peculiarly blanched; with fatty kidney the skin has a pale, waxy look; in the acute dropsy the face is pale and white, but less so than in that which follows scarlet fever; in the waxy enlargement of the kidney there is, with great emaciation, a greater amount of sallowness than we witness in cases of contracted kidney.

It is a good clinical exercise for you to watch the physiognomy of disease, the peculiar expression of the countenance, and the complexion; and in no class of diseases will you find this practice more useful than in renal affections. How often will a practised eye detect, by the peculiar sardonic expression, incipient pericarditis in rheumatic fever! How often does the face suggest the tubercular cachexy, or a cancerous affection, or some form of abdominal disease!

In addition to the dropsy of the extremities, &c., there was a more or less œdematous state of both lungs, and the history indicated that this had come on after a recent exposure to cold. This congested state of lungs, therefore, served to increase the

dropsy elsewhere, and I was led to regard the case as one of general dropsy in a certain degree dependent upon an acute irritation of the kidneys, the acute affection having supervened upon a chronic diseased condition of these organs; which, together with the embarrassment to the pulmonary circulation, would explain the phenomena exhibited by this patient. But you will not lose sight of the fact that embarrassed pulmonary circulation and bronchial irritation are often *effects* of the poisoned state of blood induced by chronic renal disease; a point of great interest with reference to the pathology of bronchitis.

The sediment which the urine deposited on standing, when examined under the microscope, was found to consist of granular casts and some waxy casts of rather large diameter, some of which contained one or more fat-cells entangled upon them, with here and there a few free oil-globules. There were also numerous pus-cells and much vaginal epithelium. Pus-cells and vaginal epithelium, I should tell you, are very common in the urine of women who are suffering from more or less irritation in the uterine organs, accompanied, perhaps, with leucorrhœa; and the appearance of pus in the urine under these circumstances has often puzzled the practitioner, and led him to imagine that some suppurative process was going on in the kidney. And a slight leucorrhœal discharge will often give rise to a considerable quantity of pus in the urine.

What, then, is our diagnosis in this case, and on what does it rest? It is plain, from the history, that our patient has been the subject of renal disease since a time prior to her first admission into the hospital—that is, more than a year. It is highly probable that, during all this time, she has been passing albumen in her urine, and, owing to attacks of cold and catarrh, she has experienced no less than three aggravations or exacerbations of the symptoms; the dropsy supervening and subsiding with the access and relief of the colds and of the bronchial attacks.

Now, it is not likely that this woman's symptoms could be caused simply by a succession of acute attacks affecting the kidneys. The amount of dropsy on each of these occasions was too small for that form of disease, nor did the urine quite corres-

pond with that which usually accompanies it; it was too copious, free from blood (which is generally present in acute dropsy), and contained too few epithelial casts and particles, which in such cases are usually thrown off in considerable quantity.

Is there a fatty or a waxy enlargement of the kidneys, or does the disease consist in a contracted state of these organs? The weight of evidence seems to me to favour this latter conclusion. In the former disease there is usually much more pallor and much more dropsy than our patient exhibits; the dropsy has subsided too readily and completely between the attacks, and the amount of albumen in the urine has fallen too low, for this slowly progressive disease. The occurrence of fat-cells in the urine would seem to favour this diagnosis; but it is well known that such cells as were observed in the secretion in this case are frequently generated in the more advanced stage of chronic contracted, wasting kidneys.

I must give the negative, also, to the supposition that this patient labours under the waxy enlargement of the kidney. The emaciation is hardly sufficient for this disease; and the absence of enlarged liver and spleen are opposed to this view of the case. The dropsy, also, is rather too much for that disease, and the fact of its subsidence and recurrence is not favorable: the urine, moreover, is less albuminous and less dense than is usual in the cases of waxy kidney, and the sedimentary matters are not of the kind met with in that disease.

I come, therefore, to the conclusion that this is a case of chronic contracted or contracting kidney. The urine being sufficiently abundant, pale, low in specific gravity, and, except during the severity of the acute attacks, containing albumen in small quantity, confirms this view of the case, which is likewise quite consistent with the long continuance of the disease and its occasional exacerbations.

Our patient's symptoms have on this, as on the former occasions, yielded sufficiently readily to the treatment adopted, which has consisted in pretty free purging, and the use of the non-irritating diuretics, such as bitartrate of potash first, and afterwards the benzoate of ammonia. The catamenia had been suppressed for three months; and this led me to give the sulphate of iron with sulphate of magnesia, with apparent good

effect in the restoration of this function. Twice this patie n suffered, while in the hospital, from rather profuse epistaxis; and she showed a disposition to purpura on her legs. Both disappeared under the use of the iron in combination with purgatives.

Epistaxis is a symptom of not uncommon occurrence in renal disease. According to my experience, it belongs more especially to the chronic wasting and contracting kidney, rather than to the other forms; and it appears to mark an advanced stage of the disease, when the blood has been much injured in quality, and the eliminating power of the kidneys for organic matters has been much diminished.

This woman exhibited, for a considerable time after her admission into the hospital, a very oppressed state of breathing, and a marked tendency to coma. Both these states were due to one and same cause—viz., a poisoned state of blood, disturbing the nutrition, and deranging the functions of the respective organs of respiration and of the consciousness. The disturbance in the lungs assumed a more serious form than that in the brain, owing, doubtless, to their exposure to other morbific influences—cold air, irritating matters in the air, and also to the accumulation of fluids secreted in increased quantities by the mucous membrane of the air-passages, under the influence of noxious blood. This is, as I believe, the true pathology of the so-called *renal bronchitis*, which is apt to occur in the acute affections of the kidney as well as the chronic, as in the acute renal dropsy after exposure to cold and after scarlet fever. We find an analogous condition of lungs from poisoned blood in typhus fever; a state which still less deserves the name of *bronchitis* than that from diseased kidney. In pyæmia, too, a similar affection occurs.

The organic disturbance of the brain which accompanies and causes the comatose tendency is, as I have already remarked, much less than the pulmonary affection. There we find nothing which the most zealous morbid anatomist could call inflammation; and, except the patient may have died in convulsions, we do not even find congestion—that most fertile of causes with a school of pathologists which is, I hope, fast disappearing. Indeed, the brain is generally anæmic; and the disturbed function consists in an almost paralytic condition of the cerebral

hemispheres (the centre of consciousness and intellectual action), from impoverished and depraved nutrition. Sometimes, as you know, this depraved nutrition will give rise to epileptic convulsions; and sometimes one or more of the small and weakened vessels will give way, and a true apoplectic coma will be established.

The existence of the comatose tendency is another point in favour of the diagnosis which I have given. For this symptom is much more prone to occur as a consequence of the contracted kidney than in any other form of renal disease. The peculiar state of nutrition which induces the constant state of waste, with wholly disproportioned renewal, is unfavorable to the elimination of the organic matters peculiar to the urinary secretion. And it is the accumulation of these matters in the blood which favours the development of coma and other derangements of brain-function.

That you might judge how imperfectly the kidneys acted in this case, I requested Dr. Conway Evans to make a careful analysis of this woman's urine; and I shall now give you the details of it. The quantity of urine which the patient usually passes in the twenty-four hours is about forty ounces; and, on the day on which it was analysed, the whole amount secreted, from ten o'clock one morning until the same hour the following morning, was collected and mixed together, and a portion of the entire quantity submitted to examination. The following were the results obtained:

Urine in 24 Hours.

Water	17129·016 grs.		
Solid matter	531·984	Organic matter .	435·555 grs.
		Fixed salts . .	96·429
Non-albuminous solids	399·121		
Albumen		132·863 grs.	
Urea		104·994	
Extractive, uric acid, ammoniacal salts, &c.		197·698	
Alkaline salts		87·334	
Earthy salts		9·095	
Chloride of sodium		30·800	
Sulphuric acid		17·837	
Phosphoric acid		15·930	

Now, taking the amount of solid matter ordinarily excreted

by the kidneys of a healthy adult in the twenty-four hours at about 650 grains, or in the case of a female, particularly when deprived of exercise, at considerably less than this—say 550 or 580 grains—it appears from this analysis, that the quantity of solid materials eliminated by this patient's kidneys in the same period is not so very much below the normal standard. But, upon further examination, you will find that of the 532 grains of solid matter excreted in the urine of this patient, nearly 133 grains consist of albumen; thus reducing the *non-albuminous solids* to rather less than 400 grains. And, if you look still further into the analysis, you will find that the amount of urea thrown out of the body daily, instead of being about 240 or 250 grains, is barely 105 grains; thus showing very clearly, I think, that one of the most striking features in these cases is the deficient excretion of urea and other urinary solids.

Had an analysis been made of the blood in this case, there can be no doubt that we should have found a notable quantity of urea in it, and we might have thus accounted for, at least, a portion of that which ought to have appeared in the urine. Some years ago there was in the hospital a man named Armstrong, forty years of age (vol. xxix, p. 72), who had well-marked symptoms of contracted kidney, complicated with imperfect mitral valve and a weak and dilated heart. Owing to this complication, and the consequently enfeebled capillary circulation, the albumen in the urine was more abundant, and the dropsy greater, than usual. This patient died comatose, and the post-mortem inspection revealed contracted kidneys with numerous cysts. In this case urea was found in the blood, and also in the serum obtained from a blister. Dr. Beale, who made the examination, reported that he obtained from half an ounce of the blister-serum ·54 gr. of urea. He also detected it in considerable quantity in the brain.

In the very general sketch which, in this and the preceding lecture, I have attempted to draw of the various affections of the kidney which give rise to albuminous urine, and likewise very frequently to dropsy, it has been my aim to bring under your notice chiefly such points as best deserve your attention in clinical investigation, and which may afford aid in diagnosis. It is quite beside my purpose to attempt any pathological dis-

cussion, although a more fruitful source of such discussion could scarcely be found. Let it be enough for you at present clearly to understand the clinical history and the morbid anatomy of these affections, and to watch the results of treatment.

Before we part, I must ask you to look through the several microscopes which are arranged on the table, under which are placed specimens of the various kinds of tube-casts which indicate these morbid conditions of the kidneys. Under this microscope, for instance, are placed *epithelial casts* and scattered *renal epithelial cells*, such as are often met with in the acute forms of renal dropsy; as in that which follows scarlet fever, and in that which sometimes results from exposure to wet and cold. Here you will see *granular casts*, with a few *waxy casts*, such as are generally found in the urine in that form of chronic renal disease which ultimately produces wasting and shrinking of the kidneys, and from which, I believe, the patient who has occupied so much of our attention to-day is suffering. Under these two microscopes, you will see *tube-casts* which occur in the urine in those cases of that form of chronic disease which tends to the permanent enlargement of the kidneys, rendering them either *fatty* or *waxy*. Here are *fatty casts* and *cells;* and here are what are termed *large waxy casts*. I use the nomenclature of Dr. Johnson.

Here, again, are tube-casts which may occur in the urine under a variety of circumstances; whenever, in fact, severe hæmorrhage takes place in the kidney. These are *blood-casts;* and they frequently result from a poisoned state of blood, which, as I just now remarked, may be produced by very different causes. An instance in which these casts were recently (only last week) detected in the urine was that of an old man in Rose ward, whom most of you must recollect, and who was labouring under chronic bronchitis and emphysematous disease of the lungs. In this case the impediment to the breathing was so extreme, that it led to such great congestion of the kidneys as to produce rupture of certain of the Malpighian capillaries, and thus gave rise to the formation of *blood-casts*, such as you see under this microscope.*

* For a fuller account of these various microscopic objects I refer to Dr.

In conclusion, let me caution you to keep in view the complications which are apt to accompany these renal diseases, and which more or less interfere with the development of the phenomena as I have explained them. Of these complications, diseased or weakened heart is one of the most frequent as well as one of the most serious. Contracted liver is also an important complication, and tends to determine the dropsy to the peritoneum. No complication is in itself more distressing to the patient than bronchial irritation; by embarrassing the pulmonary circulation it tends to aggravate the dropsy, and by favouring congestion to increase the albumen. The existence of any or all of these complications tends to increase the difficulty of the diagnosis, by destroying the clearness of the line of demarcation, as regards clinical features, between the several forms of renal disease, which give rise to albuminous urine and to dropsy. Much of the difficulty of the diagnosis of the case which we have had under consideration is due to its complications with heart and bronchial affection, and with some degree of hepatic disease. They have induced a much greater amount of dropsy and much more albumen in the urine than are found in uncomplicated cases of contracted kidney.

Johnson's book, and also to Dr. Beale's work on the Microscope in its application to Practical Medicine.

LECTURE XXI.

ON DISEASES OF THE URINARY ORGANS.

DROPSY.

In my lecture to-day, gentlemen, I shall make some remarks on the general doctrines of dropsy. This subject is naturally suggested by our having had under observation for some time past in the hospital many cases of dropsy, from various causes. Thus we have, and have had, many examples of dropsy after scarlet fever, of which there has lately (1848) been a considerable epidemic. There have, also, been two cases of ascites connected with diseased liver: of these patients, one has, within the last day or two, left the hospital improved; the other is still here, but he goes out in a few days, all his dropsy having disappeared. We have likewise under treatment a well-marked example of universal anasarca, commonly called *acute* or *inflammatory dropsy*, which is very nearly allied to the dropsy which follows scarlet fever; and within the last two or three days, a woman has been admitted with dropsy of the lower extremities, and, in a less degree, of the upper extremities and face also, which conditions are associated with distinct indications of some cardiac disease.

These are examples of the principal typical forms of dropsy. I propose, in this and some following lectures, to examine the pathology and treatment of this morbid condition, and to avail myself of these and such other illustrations as may hereafter arise.

You are, doubtless, aware that dropsy is but a symptom, an indication of a disturbed state of the circulation—such as permits a portion of the serum of the blood, or of the liquor sanguinis—very frequently, if not, indeed, most frequently, the

latter—to transude through the parietes of the small blood-vessels, and thus to escape into some serous sac, or into the areolar tissue, according to the situation and nature of the disturbing cause.

The simplest form of dropsy may be described as that which accompanies local derangements of the circulation. Women of weak constitution, whose tissues are lax and muscles flabby, and whose blood is probably deficient in some of its essential principles, or more watery than it ought to be, are prone to have swelled legs after standing or walking; the subcutaneous areolar tissue about the ankles becomes puffy and swollen, and pits on pressure; and this condition is always aggravated towards the close of the day. In such instances, the erect posture deranges the circulation of the legs and ankles; an effect which may be produced in strong persons by a very long continuance of the same cause.

A bandage applied too tightly, if left on for a sufficient time, will produce a swollen and dilated state of the veins below the point of application, and ultimately œdema and dropsy of the areolar tissue (anasarca) below the line of pressure of the ligature. The derangement of circulation which gives rise to this dropsy is a retarded return of the blood through the veins of the part, occasioned by the pressure of the bandage; the blood accumulates in the capillaries, which are, to a certain extent, relieved by the transudation of its liquid portion through their parietes into the interspaces of the surrounding areolar tissue.

When the general nutrition of any member or organ of the body is materially depressed, dropsy may ensue in it. You have frequent opportunities of observing an illustration of this fact in the anasarcous condition into which paralytic limbs are apt to fall. The limb which has suffered most in its nervous power is in general that which exhibits the greatest amount of dropsy. Hence, in the hemiplegic paralysis, especially in that of old persons, you often find the upper extremity anasarcous, but the lower not at all so. The occurrence and amount of dropsy, in either, will be very much promoted by the limb being kept in a dependent position; indeed, a limb, thus enfeebled in its circulation and nutrition, may be made dropsical in a few hours, by being allowed to hang down, or kept otherwise below the level

of the body. In such cases, the circulation is at fault, in consequence of the weakening of the proper forces of that portion of it which passes through capillary vessels. This is manifest from other phenomena which occur in these limbs, such as the wasting and flabbiness of the muscles, the softness and pallor of the skin, and the failure in the heat-producing function, which leaves these limbs almost always colder than the others.

To a similar enfeebled condition of the capillary circulation may be referred the congested and anasarcous state of the dependent parts of internal organs in low diseases, such as the posterior parts of the lungs in typhus or other analogous maladies.

In phlegmonous and erysipelatous inflammations of the skin and subjacent tissue, we find a state of œdema affecting the inflamed tissue, and also extending a greater or less distance around it. This is a form of dropsy caused by an effusion of serum or of liquor sanguinis into the areolar tissue; and this fluid can find its way thither in no other way than by filtration through the parietes of the blood-vessels. Examine carefully a patient labouring under erysipelas of the face, and you will find that the swelling is entirely due to the state of the subcutaneous tissue; and, in those parts in which the erysipelatous redness has not yet fully developed itself, that there is, nevertheless, swelling, and that the skin on pressure gives a doughy feel, and pits: the redness depends on the inflammation affecting the true skin, the swelling on the dropsy of the areolar tissue. This œdema or dropsy originates, likewise, in a disturbed state of the capillary circulation. But the disturbance is essentially different from that which occasions the form of anasarca to which I last alluded; in the former the forces of the capillary circulation are weakened, in the latter these forces are exalted —the blood is attracted to the skin and areolar tissue in increased force and quantity—and the same cause which gives rise to the increased redness, likewise produces the œdema.

In like manner, when blood is determined in an unusual degree to certain membranous surfaces, dropsical effusions are apt to take place into the sacs which these membranes enclose; hence the accumulations of serum or of liquor sanguinis which so often occur in the sacs of serous membranes after pleurisy,

pericarditis, or peritonitis. It forms an interesting topic of inquiry and discussion, why these effusions should take place after some pleurisies, &c., and not after others—why some pleurisies will afford no other effusion, save that of a small quantity of liquor sanguinis, yielding more or less of plastic lymph, while others will pour out—and that in a very short time—a sufficient quantity of fluid to fill the chest and compress the lung. I dare not digress into this interesting and important field of inquiry, but must content myself with stating my belief that the proneness to liquid effusion in the one case, and the absence of it in the other, is due to a difference in the physical constitution of the blood, which in the latter instance is more liquid, and contains a larger amount of water, and less fibrin, albumen, and red corpuscles, than natural; while in the former case the blood is either normal in its constitution, or contains an excess of fibrin. And I cannot forbear the expression of my opinion, that the mode of treatment used in these serous inflammations exercises a decided influence upon the occurrence or non-occurrence of such effusions, and that the practice of bleeding—especially of venesection—contributes much to the production of them. I can certainly state that, in cases of pleurisy, thoracic effusions have been of rare occurrence of late years in my own practice; and I can only explain it by the fact, that I have, to a great extent, abandoned the mode of treating these inflammations by large sanguineous depletions, and now content myself with adopting other means, which, although less showy and less bold, are much safer and less trying to the patient, and ultimately more satisfactory both to him and his physician. It is to *large* venesection that I object; and, indeed, my experience enables me to express the opinion that general bloodletting is unnecessary, and therefore superfluous, in most cases of pleurisy. If blood is to be taken away at all, let it be done locally, by the application of leeches, or, what is still better, by cupping, which, for the relief of pain or dyspnœa, may be sometimes used with advantage.

You observe I make use of the expression "blood determined to a part." This is a phrase much employed by medical men, and one which I think we may continue to use without disadvantage. It implies that the blood-vessels of a part obtain

more than their usual share of blood, but expresses nothing as to the cause or the mechanism of the determination. Undoubtedly, local determinations may arise from very different causes, and by very different kinds of mechanism. The problem, "Why a particle of dust adhering to the conjunctiva makes and keeps the conjunctiva as red as velvet," has not yet received a clear and definite solution. My own belief upon the point is, that local determination such as this—due clearly to the presence of some irritating agent, whether in the blood-vessels or external to them—cannot be satisfactorily explained without assuming the existence of a force which operates directly upon the blood in the capillaries,—a *vis à fronte*, which, by its attractive power, assists and regulates, by distributing in due proportion, the *vis à tergo* of the heart. An enfeebled condition of this force, and an augmented power of it, may equally tend to the production of very similar phenomena, often to be readily detected by a practised eye, but which sometimes cannot be distinguished except by experiment. Under both conditions you may have determination of blood, congestion, or hyperæmia: in the one case it will be a passive, in the other an active congestion: in both cases you will have dropsy, *i. e.* effusion of serum through the walls of the overcharged blood-vessels; and this dropsy will be in the one case active, in the other passive; though in both the proneness to dropsy will be favoured by the fluidity of the blood, its aqueous character, and the diminished amount of its red particles.

In addition to other instances of dropsy from local causes already referred to, I may mention the following, as illustrative of the fact that dropsies are the result of retardation of the circulation in some part of its course.

Swelled leg, from the adhesive form of venous inflammation, or what is called *phlegmasia dolens*, is dependent on a plugging up of the femoral and other veins of the leg by the results of the phlebitis, whereby the circulation through the limb becomes more or less obstructed, and the flow through the capillaries is so delayed as to produce an œdematous state of the whole lower extremity. This is a condition very common in puerperal women, and it also occurs in men, sometimes after fever, sometimes from other causes. The researches of Mr. Davis, Dr.

Robert Lee, and Mr. Arnott have given us the clue to the true pathology of these affections, and the explanation of the physical conditions upon which the swelling depends.

Pressure on a venous trunk or trunks will cause dropsy on the distal side of the pressure. This was illustrated long ago by the well-known experiment of Lower. This celebrated anatomist and physiologist tied the jugular vein of a dog, and found that the areolar tissue of the head and neck became enormously distended with serous effusion. Andral gives a case of dropsy of the peritoneum—ascites—caused by a tumour pressing on the vena portæ; the liver in this case was healthy, and the mechanical obstruction produced by the tumour was the sole cause of the effusion. Dr. Watson, in an admirable Essay on Dropsy communicated by him to the Library of Medicine (which I recommend to your perusal), mentions a case of dropsy of the upper half of the body, which was occasioned by an aneurism pressing on the vena cava superior, the lower half of the body having been free from all effusion.

A good example of this form of dropsy occurred in this hospital, and I shall refer to it more particularly, not only as a good illustration of the production of dropsy by pressure on a venous trunk, but also to show you how a knowledge of this fact afforded material assistance in forming a correct diagnosis.

Case CX.—The patient, a man of forty-three years of age, was admitted for an anasarcous state of the right upper extremity, with a similar condition of the left, although to a much less degree. The neck and face were very much swollen and puffy, and their veins large and distended with blood; and these phenomena were more developed on the right side than on the left.

There was no dropsy elsewhere; the lower extremities, which would have been involved had the dropsy been cardiac, were quite free from swelling.

The marked enlargement of the veins of the neck and of the upper extremities, especially on the right side, and the absence of any such enlargement in the veins of the lower part of the body, pointed to some obstacle to the return of the blood from the upper part of the body to the heart.

If this obstruction had existed in the heart itself, it would

have affected the veins of the lower half of the trunk to a greater or less degree. These, however, were intact. If, moreover, it had existed in the right vena innominata, the right side of the neck and right upper extremity would alone have suffered; but although they were the parts chiefly swollen, the left were also involved. The obstacle must, therefore, have been such as would involve the superior vena cava, affecting, at the same time, but to a slighter degree, the right vena innominata.

Another point in this case aided in indicating the locality of the obstruction. The right lung was evidently much congested and the source of a pulmonary hæmorrhage, the left lung being in a perfectly natural state. As there were no signs of any cardiac disease which could explain this phenomenon, it was interpreted as indicative of pressure on the right pulmonary veins by the same cause as that which produced pressure on the vena cava. The passage of the air into the right lung was obviously much impeded, and, no doubt, by a similar compression at or near its root.

These and other signs led to the diagnosis that the phenomena depended on the presence of a tumour near the right vena innominata and the root of the right lung, compressing that vein, the bronchial tube, and the pulmonary veins. The post-mortem examination showed that an aneurism, about the size of an orange, sprang from the posterior part of the ascending aorta just above the sinuses of Valsalva. The superior vena cava was imbedded in the right wall of this tumour, and the right pulmonary veins and bronchi were compressed by it.*

Hydrocele is no doubt due to some local disturbance in the circulation of the spermatic veins, caused probably by disease of their tunics.

There are some interesting cases mentioned by Tonnellé of extensive arachnoid effusion from obliteration of the sinuses. Several cases of intraventricular effusion, brought on by pressure on the venæ magnæ Galeni at their exit from the third ventricle of the brain have come under my observation, and I am disposed to think that a large number of the cases of water in the brain in

* For a detailed account of this case, the reader is referred to a Clinical Lecture published in the 'Medical Times' of Jan. 13, 1855.

children, which are associated with a strumous diathesis, are due to an impediment to the circulation through these veins in some part of their course. All these are very good examples to show how the local retardation of the circulation may produce dropsy; the amount of the dropsy varying with the extent of the circulation upon which the obstructing cause exercises its influence.

Those cases of dropsy of the peritoneum, or ascites, which occur independently of inflammation of that membrane, and which are by far the most numerous, are due to retarded circulation through some part of the portal system of veins, either of its intestinal part, its trunks, or its hepatic ramifications. It is when these last vessels are impeded, that ascites occurs in the most decided manner; and the dropsy is most abundant when the impediment to the portal circulation is most complete. Hence the small contracted livers produce the largest amount of dropsy; the large and hard liver causes the next greatest quantity of effusion; and the livers which are enlarged from cancerous or other deposits, creating partial obstructions here and there, will produce a still less degree of peritoneal dropsy. The pressure of a large spleen, of a peritoneal tumour, of cancerous glands, or of an enlarged pancreas, may likewise give rise to ascites.

Cardiac Dropsy.—To the category of dropsy from obstructed circulation must be referred all effusions that appear to result from enfeebled or deranged circulation through the heart, or what is specially called cardiac dropsy. This may arise, first, from *simple weakness* of the heart, as in cases where the nutrition of the whole body is languid, and the heart shares in the general weakness of the muscular system. You may often see, as an accompaniment to this condition, and not unfrequently indeed as its cause, a watery state of the blood; and this very nature of the blood favours that transudation of which its tardy circulation is the immediate exciting cause. The disturbance of the circulation falls most heavily upon the returning portion of the blood —that which moves with least force, and by which the *vis à tergo* is most feebly felt, *i. e.*, the circulation in the veins. Whatever may be the cause of inefficient circulation through the heart, the

veins and capillaries are sure to feel it most, because in them the blood moves most sluggishly, and under least pressure from behind; and it is, therefore, these parts of the circulating system which are most amenable to obstructing causes.

The most common form of cardiac dropsy arises from a physical obstruction in some part of the heart's mechanism.

Suppose the obstruction to be situate at the aortic orifice, the problem is to explain how this will cause dropsy. At the early periods of such an obstruction, the excited nervous and muscular energy of the heart is sufficient to master it, and to drive the blood with its wonted force through the various channels of the circulation. By-and-by this exaltation of these vital forces no longer suffices, and, notwithstanding that the wall of the left ventricle may have acquired increased thickness, there is evidence in its state of dilatation that the backward pressure of the blood, under the influence of the obstruction, has been gradually increasing to a degree which the contracting force of the ventricle is unable fully to counterpoise; hence the yielding of the ventricular wall, and the gradual augmentation of the ventricular cavity.

But soon this great backward pressure ceases to spend itself on the ventricle alone; the outflowing blood from the auricle now encounters the obstacle, and is retarded in its course by it. Thus pressed back, it excites the auricle to greater muscular efforts, and gradually creates more or less of dilatation of its cavity. Ere long the influx of blood into the auricle feels the obstacle, and the pulmonary circulation begins to encounter difficulty. To overcome this, the right ventricle is excited to increased efforts; but it too passes through the same series of changes as the left ventricle, its walls acquiring increased thickness, and its cavity gradually becoming dilated.

This state of dilatation at length extends to the auriculo-ventricular aperture and to the right auricle. The valves of the former (the tricuspid valves), never perhaps very perfect as counteracting the reflux from ventricle to auricle, and not enlarging in proportion to the dilatation of the aperture, become quite insufficient for their office, and allow of free regurgitation of blood into the auricle.

A new obstacle is thus set up to the onward flow of the

venous blood. At every systole of the ventricle a backward wave is established, which the inflowing blood from the veins has to encounter. For a time this does no more than keep the veins near the heart—the innominate and subclavian veins, and the jugulars—in a distended state; but ere long the backward wave sensibly influences the current in them, and venous pulsation is established. This regurgitant venous pulsation is a certain index of a considerably dilated state of the right cavities of the heart, and of imperfection of the tricuspid valves.

The occurrence of this direct and decided obstacle to the return of the venous blood is the signal for the more or less rapid development of dropsy, if, indeed, this symptom have not already shown itself.* The dropsy begins in the feet and ankles—parts which are most dependent—and gradually creeps up, affecting the thighs, penis, and scrotum, which become swelled to an enormous extent, and ultimately the abdominal integuments, the face, and the upper extremities. The obstacle to the return of the venous blood to the right auricle throws it back upon the hepatic veins; the result of which is, that often, in but a very few days, the liver acquires a great increase of size, which appears to be due to the immense accumulation of blood in it.

Such is the sequence of phenomena which usher in dropsy when the circulation is obstructed as it flows from the heart. The cardiac circulation may, however, be otherwise obstructed than at the aortic orifice. Thus, the cardiac obstruction may be due to disease of the mitral valves; and it matters not what the nature of the mitral lesion, whether it permit regurgitation from ventricle to auricle, or whether it create direct obstruction to the flow of blood from narrowing of the mitral chink. In either case the blood flows with difficulty, and against a greater or less opposing force from the auricle to the ventricle. The obstruction is immediately felt by the pulmonary circulation and by the right ventricle, and so also by the right auricle and the systemic veins.

Again, you may have the left side of the heart quite free from disease, and the obstruction may be in the pulmonary circula-

* Loveland, xxvi, 1849, 179—201.

tion. We have many opportunities of witnessing this in cases of asthma, or of chronic bronchitis of long standing. In these cases, the lungs become emphysematous, the air-cells are dilated, their walls stretched, the capillary network upon them altered in form and partly obliterated. The result of all this is an impeded capillary circulation through the lungs, requiring for its maintenance increased exertions on the part of the right ventricle. Out of these increased efforts, and the great backward pressure on the wall of the ventricle, arise the hypertrophy and dilatation which it soon exhibits, the impediment to the systemic venous circulation, and the dropsy.

It may be laid down that, except under some peculiar condition of the blood, dropsy will not develop itself in effect of cardiac disease, unless the right auriculo-ventricular opening and the right auricle be so dilated as to offer a decided impediment, through regurgitation, to the flow of the venous blood into the heart.*

And it may also be stated, as a rule pretty general in its application, that the nearer the cardiac lesion is to the right side of the heart, the more likely is dropsy to appear, so far as regards lesions of like nature. Thus dropsy will more slowly follow an obstruction in the aorta or the aortic orifice than one at the mitral orifice, or affecting the pulmonary artery. In mitral disease there is one link less in the chain which connects the obstruction and the right cavities than in aortic disease; and in obstruction of the pulmonary artery, there are two links less.

Renal Dropsy.—There is yet another form of dropsy, arising partly from an altered state of blood, and partly from impaired force of the capillary circulation throughout the body, the circulation in the kidney being that primarily deranged. This form of dropsy is general. It affects the areolar tissue everywhere, develops itself rapidly, and is not influenced by position in the marked way in which cardiac dropsy is. It very often appears first in the least dependent parts, as in the face and

* Dr. Blakiston's remarks on this subject in the 11th chapter of his very practical book 'On Chest Diseases,' deserve attentive perusal.

eyelids; sometimes it shows itself in the upper parts of the body simultaneously with the lower. The face and eyelids are often among the earliest parts in which the dropsical swelling occurs.

This form of dropsy is often described as general dropsy, or acute dropsy, and as such attracted the attention, many years ago, of Blackall and others. More correctly, perhaps, it may be termed *acute renal dropsy,* from its rapid supervention and its constant association with an acute affection of the kidney.

We need not wonder that a deranged and a more or less obstructed state of the circulation through the kidneys should cause dropsy. A large portion of the blood of the descending aorta must pass, in an incredibly short time, through the complicated circulation of these organs, where it has to traverse two distinct capillary systems. The short and wide renal arteries are at but a trifling distance from the aortic orifice, and any serious impediment encountered at the kidneys, more especially if quickly developed, would soon react upon the heart and upon the whole circulation.

The dropsy after scarlet fever belongs to this category of *acute renal* dropsy. And, as it is of very frequent occurrence, and exhibits the clinical features of the disease in a very exquisite manner, I propose to take it as typical of this kind of dropsy. We are at no loss to illustrate it, especially in cold seasons when an epidemic of scarlet fever has existed.

Not to carry this lecture beyond proper bounds, I shall resume the subject of dropsy after scarlet fever when we next meet.

LECTURE XXII.

ON DISEASES OF THE URINARY ORGANS.

ON DROPSY AFTER SCARLET FEVER.

The present, gentlemen, is a convenient time to make some remarks on the dropsy which frequently follows scarlet fever; inasmuch as we have lately had a considerable number of cases of this affection in the hospital. Although it seldom happens that any one who keeps up a regular attendance in the wards fails to see at least half a dozen sufficiently well marked examples of this disease during the winter session, still we rarely have as many as this number in the house at any one time. The malady is very rife this winter, there being just now a prevailing epidemic of scarlet fever, in consequence, probably, of the extreme coldness of the season—for this has been one of the most severe winters that we have had in this country for several years (1853), and a low temperature is one of the conditions most favorable to the development of this disease.

Many of you are, no doubt, well aware how this affection comes on: nevertheless, for the benefit of those who are not quite *au fait* on this subject, I will briefly describe its usual course. A person gets scarlet fever—that is to say, the poison of scarlet fever in some way or other enters the system; sore throat comes on; and then the rash makes its appearance over the body. The latter soon disappears, and a process of desquamation took place, the whole occurring in the space of about ten days, and from this time until about the twenty-first day the patient becomes convalescent; but if the urine be daily examined during this period, it will very often be found, at some time between the fourteenth and twenty-first day, to present certain appearances which are characteristic of irritation in the

kidneys.* These indications of renal irritation may disappear, and the patient may get perfectly well; but, on the other hand, they may increase, and general dropsy will supervene—a dropsy so general as to involve the whole of the subcutaneous areolar tissue, and very often the peritoneum, and sometimes the pleura or pericardium, or the areolar tissue of the lungs. The condition of urine to which I allude consists in the presence in that fluid of certain *fibrinous moulds* or *casts* of the uriniferous tubules, together with an undue quantity of renal epithelium, the latter being shed, probably, by a process somewhat analogous to the desquamation of the cuticle. These casts and epithelial-cells may be readily seen on a microscopical examination of the urine; but chemistry also affords us aid in this matter, for the usual tests of heat and nitric acid will show that the urine contains more or less albumen. Frequently, too, the urine of patients in this condition assumes more or less of a smoky hue, which either depends upon the presence of blood, or, what as commonly happens, upon that of great numbers of minute crystals of uric acid. To which of these causes the smoky appearance is due, the microscope will readily determine: in the one case, blood-corpuscles will be seen; in the other, orange-coloured, more or less rhomboidal, crystals of uric acid, or, as not unfrequently occurs, both blood-corpuscles and uric-acid-crystals may be present in considerable quantity. The urine usually continues to exhibit these appearances for some days; the amount of albumen increases; and, at length, a dropsical state of the areolar tissue generally manifests itself. This œdematous condition usually appears first in the face and eyelids, but it soon involves the lower extremities and the abdominal cavity. The amount of the dropsy in these cases will always, I believe, be found to be in inverse proportion to the amount of colouring matter in the

* Mr. Tripe, in an able statistical analysis on scarlatinal dropsy, makes out that the fourteenth day from the commencement of the fever is that on which the dropsy most frequently comes on, and that the other days on which its invasion occurs most frequently are the twenty-first, the twelfth, and the seventh, the relative frequency being in this order, and then the thirteenth, eighteenth and twentieth.—'Brit. and For. Med.-Chir. Review,' No. xxvii, p. 214, 1854.

blood, in other words, the more pallid the patient, the greater will be the dropsical effusion.

There are three conditions which, I imagine, are necessary to give rise to the production of the dropsy in these cases. These are—1st, a peculiar irritated state of the kidney; 2d, an analogous morbid state of the skin; and 3d, a certain depravity of blood, by which I mean, not only a deficiency in the amount of the red corpuscles of that fluid, as well as of the solids of the serum, but also the unnatural presence of certain poisonous matters which interfere with its proper nutrient changes. The children of the ill-fed and ill-clad poor are particularly obnoxious to the causes which tend to impoverish the blood. On this account, as well as because they are badly protected from cold, this affection is much commoner among the lower classes than among the upper; and when it is met with in the latter, it is generally in strumous children, who, though well protected from cold, have blood which is considerably *below par* in one or more of its staminal principles.

It appears to me that we do not meet with the dropsy fully developed without the concurrence of all the three conditions which I have just mentioned. If any one of them is absent, you may have a threatening of the dropsy, but the full result does not follow. Thus you may have the peculiar state of the blood, and the peculiar state of the kidney; but if the state of the skin be normal, the dropsy will be slight, or *nil*. So likewise, when the peculiar conditions of the blood and of the skin are present, but the kidneys are healthy, the dropsy does not appear; and even if the particular state of the kidney and skin both existed (and, under such circumstances, you could scarcely have a healthy state of blood), yet, if the state of that fluid did not correspond with that which is favorable to dropsy, you would have other symptoms—head affections, for instance—but there would be no dropsy.

Case CXI.—You will find a good illustration of these points and of the ordinary clinical history of this form of dropsy in the case of a little boy in Sutherland Ward: Thomas Dunn, aged five. He seems to have had a mild attack of scarlet fever, and the dropsical condition appears to have come on shortly after

the commencement of the desquamative stage. When he came into the hospital he was suffering from universal dropsy, affecting even his peritoneum. His skin was puffed out everywhere, especially over the penis, scrotum, extremities, and face, and it had that peculiar white, semi-transparent, waxy appearance, which is so characteristic of these cases. There was also either an excess of subarachnoid fluid, or, what is more likely, slight effusion into the ventricles of the brain, or, more likely than either, a poisoned state of brain from impure and poor blood; for, during the first two or three days after he came in he was very drowsy and lethargic, as if under the influence of some narcotic. His urine was deficient in quantity, smoky in colour, and it showed a great abundance of albumen on the application of the appropriate tests.

Now let us see in what way this case presented those three conditions, the concurrence of which I have just now stated to be necessary to the production of this form of dropsy.

1. The *skin* was dry, rough, and harsh, and it seemed as if in an irritated state: but this irritation was not extreme. It would have been better, I think, if it had been more irritated, as such a condition would probably have enabled the patient to have thrown off more completely by cutaneous elimination the morbid poison; for it is a known fact that the dropsy generally occurs in the mildest forms of scarlet fever, in which there has been little or no eruption; but in those cases in which the eruption has come out well, and the desquamation has consequently been excessive, it is much less likely to occur, except under the influence of a strongly exciting cause, such as prolonged exposure to cold.

2. The *kidney* presented an analogous condition to that of the skin; as there has been a desquamative state of the one, so there probably is a similar condition of the other. When we examine the kidneys in these cases (and now, thanks to recent researches, of which those of our friend, Dr. Geo. Johnson, are among the first both in time and importance, we have a very exact knowledge of its precise anatomical condition in this form of disease), we find the renal tubes filled with epithelium, and the whole organ enlarged, and in a state of hyperæmia, so far as this great filling of the uriniferous tubes would permit it to

be so. Not only is there a large quantity of blood in the organ, from the undue attraction of that fluid to it by reason of its irritated state, but also the blood is irregularly distributed in it. The principal anatomical change in the kidney results from the development of an undue quantity of epithelium in the uriniferous tubes.

The accumulation of epithelium creates an unnatural distension of the tubes, and the circulation in the small vessels which ramify upon their walls (forming the portal vessels of the kidney) becomes impeded. Thus the blood is not only strongly attracted to the Malpighian bodies, but it also finds its way out of them with difficulty; no wonder, then, that great congestion of these bodies should take place. From this over-filling of the Malpighian capillaries there results an escape of serum into the uriniferous tubes, and from the rupture of many of these minute vessels, the red particles, in greater or less quantity, pass into the urine; but little urine is secreted, and this imperfect elimination of water is negatively a further cause for the accumulation of epithelial particles in the uriniferous tubes, inasmuch as less fluid percolates the tubes to wash them out.

The congestion of the Malpighian bodies, when it exists to a certain amount, produces effusion of liquor sanguinis into the tubes; but when it occurs to a still greater degree, it leads to rupture of the Malpighian vessels, and the escape of *all* the constituents of the blood: in the former case the urine is merely albuminous; in the latter, in addition to albumen, we find in it an abundance of blood-corpuscles, and fibrinous casts of the uriniferous tubes, from the fibrin having moulded itself to their walls in the process of coagulation. This is the state of the kidney; and accompanying it we find almost always that peculiar and very characteristic smoky condition of the urine, which I have already described.

3. The *blood*.—What is the condition of the blood? Whoever looks at our patient, and, indeed, at all patients labouring under this form of dropsy, will see at a glance that there is every indication of a great want of red particles, and of a too watery condition of the blood. The best analysis of the blood in scarlet fever shows that no material change takes place in the proportions of the several staminal principles in the early stages of the

disease. But, as the disease advances, the red particles suffer a rapid diminution, and sometimes fall to as low as one third their normal amount. At the same time the specific gravity of the serum diminishes considerably, and the albumen of the serum also falls very much in quantity. Several observers notice also the presence of urea in the blood in these cases. This state of blood is clearly favorable to the transudation of its liquor sanguinis through the parietes of the blood-vessels, and unfavorable to those changes on which the maintenance of a normal state of the vital fluid depends. From the probable deficiency in the nutrient changes in the blood itself, there is doubtless more attraction from the blood *to* the tissues (*exosmose*) than from the latter to the former (*endosmose*). This excess of current out of the canal of the blood-vessels to the surrounding areolar tissue would favour greatly the production of a state of anasarca and general dropsy.

These facts will, I think, lead us to form something like a theory of the mode of formation of this dropsy. What we have got to explain is this, that to-day a child may be going on very well, and to all appearance in a favorable convalescence, and in a few days afterwards may rapidly become universally œdematous, with effusions to a greater or less extent into the various serous cavities, accompanied by scanty urine, smoky in colour, and albuminous; in fact, exhibiting all the appearances I have already described. If you look to the three conditions which I just now mentioned, you will, I think, get a satisfactory explanation of these phenomena.

First.—From some cause not easy of detection, but in some instances undoubtedly from exposure to cold or wet, there is an arrest to the proper elimination of the scarlet-fever poison through the skin, its usual emunctory, and the ordinary excretion of water through that organ is checked; not obtaining complete egress there, the poison finds for itself another channel, and is thrown on the kidneys. Its passage through these organs produces great irritation in them, the effect of which is, that water is imperfectly eliminated, and thus the escape of water from the blood is prevented through its two ordinary channels—namely, the skin, which is *an* emunctory of it, and the kidney, which is, *par excellence, the* emunctory of it.

Secondly.—As a direct consequence of this obstruction to the escape of water through its two principal channels, a watery condition of the blood is induced. It is calculated that we get rid of three pints of water in a day, by the secretion of the kidneys, and by cutaneous perspiration, and this is certainly not too high an estimate. Now this water must be got rid of in some way or other, and when its usual channel of escape is cut off, it is very apt to permeate the parietes of the blood-vessels. But why do we find it particularly in the areolar tissue, and why in that of the skin more than any other part? It finds its way into the areolar tissue of the skin, in consequence of the determination of blood to that integument, caused by its state of irritation; for in order to reach the skin the blood must pass through the subcutaneous areolar tissue. It would be wrong, however, to suppose that the effusion is always confined to the neighbourhood of the skin; we find it in the areolar tissue of the lungs, and in the serous cavities; in the former, because of the necessarily large flow of blood to the lungs; and in the latter situation, because of the extreme tenuity of the tissue of the serous membranes, which affords but the slightest obstacle to the escape of the serous part of the blood.

Thirdly.—All this is favoured by the impoverished state of the blood. If the scarlet-fever poison be not eliminated, it interferes with the proper nutrient changes which take place in the blood; and this is shown by the imperfect development of the red particles. The poison of scarlet fever appears to me to interfere with the formation of this important element of the blood, in the same way as that of rheumatism does, and other poisons likewise, inorganic as well as animal—as, for instance, lead. This impoverished state of the blood undoubtedly materially affects the proper rate and vigour of the capillary circulation. That force of attraction to which I have already referred—the capillary force, or *vis à fronte*—cannot be so vigorous when the blood is deficient in all or nearly all its solid ingredients, as when that fluid is healthy. It is not improbable, likewise, that there may be some special chemical condition of the blood in these cases. Magendie and Poiseuille found that the introduction of alkalies into the blood occasioned a great retardation of the circulation through the capillaries, and con-

sequent dropsical effusion: what the precise condition of the blood is in scarlatina has yet to be shown; at present we can only conjecture that some such abnormal state of it does exist; that is to say, that, besides containing too much water, and too little colouring matter, it contains some special chemical agent likewise, which interferes with its proper vital changes.

Such is my theory of the dropsy after scarlet fever. What may be the ultimate fate of it upon a larger induction of facts, I will not attempt to predict. I offer it to you now, as a convenient mode of connecting the various phenomena which accompany, and doubtless tend to the production of, the dropsy.

Treatment.—As you have in this disease a more or less irritated state both of the skin and kidneys, the first and chief indication for treatment is to allay that irritation. Now, for this purpose, I know no more valuable or efficient remedy than the warm bath; indeed, I would pronounce it *the* most valuable single remedy for this state of dropsy. You must use it frequently, bearing in mind, however, that both the disease and the remedy have a depressing tendency. You will be careful, therefore, not to use it oftener than your patient's strength will bear: most patients can bear it once a day for a few days: sometimes, however, you may give it twice in a day; but often you must not venture even upon the daily use of it. You must also be careful not to use it at too high a temperature. From 90° to 96° Fahr. you will find quite hot enough. In cases where you apprehend the access of dropsy, you may often succeed in arresting it by the daily use of the warm bath during the period when desquamation is or ought to be taking place. The next most important remedy is purgatives, which, by their action on the intestinal mucous membrane, open a new emunctory for the elimination of water, and with it any poisonous ingredient dissolved or suspended in it. Jalap, calomel, scammony, the compound powder of jalap, singly, or variously combined, are very admissible for this purpose, or the saline purgatives. With these you may give some of the milder diuretics, which do not exercise any direct irritative action on the kidneys; such as the liquor ammoniæ acetatis, or the bitartrate of potass. The last, in small doses, you will find extremely valuable. If, after a

treatment of this kind pursued for several days, you find that the kidneys refuse to act properly, and that the urine is very smoky, you may take a little blood from the loins by leeches or cupping, to relieve the local congestion. I do not recommend you to do this early, during the more irritative stage of the renal affection, as you will generally find bloodletting at that time much less efficacious in diminishing congestion than at a later period. Take but little at a time, and rather repeat the bloodletting in small quantities, than take much at once. It very rarely happens, indeed, that patients labouring under this disease can spare much blood.

In pursuing this treatment, I pray of you to regard it not as antiphlogistic, but as calmative and eliminatory, soothing cutaneous and renal irritation, eliminating water by the bowels, the kidneys, and the skin. Whilst using these remedies you must always support your patient by nourishing food, and often you must stimulate; indeed, you will frequently find the most signal benefit derived from giving port wine. Do not, when you feel that your patient's strength needs it, be deterred from giving stimulants by the fear of exciting fresh irritation of the kidneys. Your best guide as to the propriety of continuing stimulants or any other food, is the facility with which your patient *digests* them. If they create flatulence, gastric distress, pain, or any other symptom referable to the stomach, you must diminish the quantity or stop the supplies altogether.

Sometimes in the course of this disease you have symptoms of head affection. These arise either from dropsy of the ventricles, or from poisoning of the brain by retained urea. Free counter-irritation immediately and extensively applied to the nucha or the scalp, is the best remedy; and generally speaking, bleeding, local or general, is inadmissible. Sinapisms, succeeded immediately by blisters, are most valuable remedies in these head affections.

The boy whose case has formed the text of this lecture was treated much in the way I have described. He came in on the 15th October, and I ordered him a daily warm bath, and mild purgatives. The next day I made a slight alteration by giving him a more drastic purge, in the shape of a dose of compound powder of jalap, which carried off an abundance of watery

stools. The following day (18th) he passed a pint and a half of urine, and the dropsy had greatly diminished. The next day he passed two quarts of urine; and from this time it was wonderful with what rapidity and in what quantity the kidneys continued to secrete: this great diuresis is frequently to be noticed in this form of dropsy. On the 19th he passed 2½ quarts. Under these circumstances it is not surprising that the dropsy almost entirely disappeared. Still the urine retained a little smokiness of colour, indicating the escape of blood; and, with the view of checking this, and applying a remedy to his anæmia, I have given him a little citrate of iron, under which treatment he has been rapidly improving, and he is now nearly well.

This case affords a good example of the favorable termination of this malady, as well as of its course. Dropsy after scarlet fever is not a disease of very fatal tendency (although it often kills rapidly), more especially when the preceding fever has been treated mildly, and with due regard to preserving the powers of the patient. Furthermore, I should not, generally speaking, prognosticate any special liability of this, or other patients similarly affected, to renal disease hereafter. I know that some deservedly high authorities think otherwise; but it seems to me we need much more proof than has yet been offered, before we shall be justified in affirming that the state of kidney which follows scarlet fever predisposes either to the large fatty kidney of Bright, or to any of the other forms of chronic renal disease.

If a patient who has had scarlet fever passes over three or four weeks after the cutaneous desquamative process has fairly taken place, and if by that time there be no albumen in the urine, or other indications of an irritated state of the kidneys, you may generally regard that patient as tolerably secure from any attack of dropsy; but it is the safest practice, I think, not to pronounce a decided opinion on this point until six or even eight weeks have elapsed, during which the patient has been pretty free from all the symptoms of scarlet fever.

We have had more cases of this affection in the hospital during the last few months than for a long time previously, and I attribute this, in great measure, to the extreme coldness of the season. Among these several have been fatal. I am, there-

fore, enabled to bring before you cases in which the symptoms leading to a fatal issue have been prominent, as well as those which have run a favorable course. You will thus be the better able to form a judgment as to the cause which determines either the fatal or the more prosperous issue.

The first of these fatal cases terminated very quickly, and its recital will, I think, serve to impress upon you the principal points in the clinical history of the disease when it passes to a fatal termination.

Case CXII.—The patient was a little boy, named Edward Scales, aged 7 years (vol. xl, p. 145), who was admitted into Rose Ward on November 5th, 1853. The following report of him was made by my clinical clerk, Mr. Bird:—"He had an attack of scarlet fever about a month before his admission; the disease, however, was very slight, the eruption being ill-defined, and the febrile symptoms insignificant." Now this slight eruption is an important feature in the case, and one to which I should wish to call your special attention. Hereafter, in your own practice, you will be very likely to meet with similar cases. You will be asked to see a child, whom you will find pale, waxy, and swollen from universal dropsy. The parents will naturally be extremely anxious; they will, perhaps, tell you that the dropsy has been preceded by no illness whatever; but you will be well aware that this condition must arise from some cause, and in the majority of cases you will learn, by careful inquiry, that there has been some sore throat a short time previously, or that the child has been in the way of scarlet fever, and, possibly, may have had that disease, although it has not passed through its early stages in the usual course. In short, in consequence of the poison having been determined mainly to the kidneys, no symptoms were manifested, until the irritation of these organs had been set up, and the general dropsy established.

As a good example of the dropsy supervening, where there was little or no primary fever, I may notice the case of a little boy, named Alfred Austen, aged 8 years (vol. xl, p. 203), who was admitted into Rose Ward on December 28th, 1853. In this child the affection came on just in the way I have described, the dropsical symptoms being the first that attracted notice.

The report is this :—"He was born in the city, and has lived in London all his life; his health was good, according to his mother's account, until they removed into Windsor Court, Strand, which, she says, is very damp. His present illness commenced with soreness of the eyes, drowsiness, and swelling of the belly, legs, and face, with puffiness of the eyelids." Upon inquiring further into the case, we found that "his brother and sister, both children, are in the same state as himself, and the former is not expected to live." Here, then, was evidence that all these children had been exposed to similar morbific influences, and that the disease which had resulted had not run its usual course, but had given rise to irritation of the kidneys and dropsy.

There is a remarkable unanimity between all practical observers and authors respecting one point connected with dropsy after scarlet fever, namely, that the dropsy is most apt to come on in those cases in which the cutaneous affection has been slightly or not at all developed. Still I should tell you that though authors agree on this point, there is some difference of opinion among physicians, as to whether the tendency to dropsy is greatest when the cutaneous desquamation has been greatest, or the reverse. My own experience would lead me to adopt the latter of these propositions, and to affirm that the more abundant the cuticular desquamation, the less likelihood is there of the occurrence of dropsy. Still both propositions may be correct, and for this reason, that a large quantity of the poison of scarlet fever being imbibed, and the rash not having been produced, yet cutaneous desquamation may take place, and renal desquamation, being of a similar nature, may also occur; and these two processes, going on at about the same time, or, as is more generally the case, the renal a short time after the cutaneous, may both proceed to a considerable extent in consequence of the patient having imbibed a large dose of the poison. But it is easy to conceive that by far the greater quantity of the poison may have fallen on the skin, or on the kidneys respectively, and, consequently, that in the former case the renal, in the latter the cutaneous, affection may be almost wholly absent, or, at all events, but very slight. Although altogether an exceptional case, it is quite possible, and, indeed, I know it for a

fact, that dropsy may follow an attack of scarlet fever, in which the cutaneous desquamation has been very copious.

But to proceed with the case of Scales.—When admitted on November 4th, he had some effusion into the peritoneal cavity, but very little into the cellular tissue generally; he was passing about six ounces only of urine in the twenty-four hours, and this was highly albuminous. A warm bath was prescribed for him every night, mustard poultices were directed to be placed over the loins, a scruple of compound jalap powder was given every morning as a purgative, to carry off water by the mucous membrane of the bowels, and three-drachm doses of the liquor ammoniæ acetatis were given every fourth hour, with a view to determine more decidedly to the skin. On the 7th, three days after the commencement of the treatment, twenty-eight ounces of urine were passed in the twenty-four hours; on the 8th, the quantity fell to twelve ounces in the same period; on the 9th, also, twelve ounces; on the 14th, only eight ounces; on the 15th, ten; and on the 16th, only eight. There now came on a very important symptom—viz., vomiting without any distinct cause, and, as the quantity of the urine continued to diminish, it seems highly probable that the vomiting was due to defective action of the kidneys, with the consequent accumulation in the blood of certain of those constituents, which ought to be eliminated in the urine; due, in fact, to what is now termed *uræmic poisoning*, or, as I would prefer it, *poisoning by some of the constituents of the urine*—as, in the present state of knowledge, it cannot be decided with certainty which of the elements of the urine constitutes the poisonous matter.

On the 19th, only six ounces of urine were passed in the twenty-four hours, the dropsy had considerably increased, great dyspnœa and cough had come on, and the lungs were rapidly becoming highly œdematous, as proved by the existence of moist crepitation, heard generally over the chest; and on the 21st the patient died, in consequence of extreme difficulty of breathing.

A *post-mortem* examination of the body revealed a large amount of fluid in the pericardial sac, with several patches of recently effused lymph on the surface of the heart. The lungs were so œdematous that they almost sunk in water; and the

kidneys presented a very characteristic appearance, and one with which, if you watch many cases in which death occurs in dropsy after scarlet fever, you will almost always meet. The condition of kidney to which I allude, consists in a remarkable whiteness of the cortical substance, with a highly red and congested state of the medullary cones. The Malpighian bodies under these circumstances are pretty large, and the minute vascular system of the kidney is gorged with blood; while the uriniferous tubes, on the other hand, are crammed with epithelium, which thus gives the cortical portion of the organ its white character.

The peculiar whiteness of the cortical substance of the kidney is due to the great quantity of epithelium which is accumulated in the convoluted portions of the tubes which chiefly compose it, and which, by compressing to a certain extent the intertubular vessels, keep up the congested and gorged state of the Malpighian bodies. The great augmentation of the intra-vascular pressure in these vessels, allows not simply water to transude through them, as happens under ordinary circumstances, but water *plus* albumen, *i.e.* serum, and even fibrin; and, therefore, the amount of albumen in the urine is always proportionate to the quantity of serum transuding through the walls of the Malpighian capillaries, and proportionate, also, in great measure at least, to the degree of pressure exerted by the blood within these vessels. There is a kind of vague notion with some, that there is a sort of *secretion* of albumen, and that it is by some modification of secretion that this substance finds its way into the urine; but I can scarcely think this a correct view, for the process is, in truth, of the simplest possible nature, being merely one of *transudation* or *filtration;* the force which, under ordinary circumstances, sends the watery part of the blood out of the Malpighian vessels, being so increased as to expel more or less of albumen along with the water.

The state of the lungs, in this and other similar cases, is very remarkable. These organs are found in a highly œdematous condition, and so charged with blood and serum as nearly to sink in water. How are we to explain this? An analogous condition occurs in other diseases which arise from the influence of an animal poison, as in typhus, in measles, in hooping-cough.

It is found also in certain states of heart disease, when the returning pulmonary blood is much impeded; and we find it likewise in animals in whom the vagi nerves have been divided in the neck.

In the class of cases now before us, there is probably more than one cause which contributes to the production of this state of lungs. First, no doubt the impeded circulation through the kidneys increases the backward pressure on the left ventricle and on the left auricle, and thus a certain degree of impediment is offered to the return of venous blood from the lungs; secondly, the impure state of the blood impairs the forces of the capillary circulation in the lungs, as elsewhere—and thus we find an impeded flow and a retarded flow. Finally, the watery state of blood favours transudation, and consequent œdema of the lungs, which, in its turn, interferes with the perfect aeration of the blood, and increases the general dropsy, or at least opposes its diminution. No wonder, then, that the supervention of œdema of the lungs in dropsy after scarlet fever should always prove a formidable, and often a fatal symptom.

In the next lecture I shall call your attention to some additional cases in illustration of this form of dropsy, and make some further remarks on its clinical history and treatment.

LECTURE XXIII.

ON CERTAIN URINARY DISEASES.

DROPSY AFTER SCARLET FEVER.

In the last lecture, gentlemen, I gave you a sketch of the clinical history and pathology of that form of dropsy which often follows scarlet fever, and detailed to you an average case proceeding to a favorable termination.

I also pointed out to you some of the serious complications of the disease which tend to bring about a fatal result, and related the case of a boy (Scales—Case CXII), whose death seemed mainly due to pulmonary œdema, complicated with pericardial inflammation and effusion.

The first case to which I shall call your attention in to-day's lecture is of much the same kind, and terminated fatally with similar symptoms.

Case CXIII.—The subject of this case is a child named Samuel Andrews (vol. xlii, p. 157), lately under my care in Sutherland ward. It is of the greater interest, from the struggle which the poor boy made for life. At one time, indeed, great hopes were entertained that we might have carried this patient through, but the dropsy ultimately baffled all our efforts. We have the following history of this lad:—"He is six years of age, and was always very healthy until about three weeks before his admission into the hospital, when he became dull and did not play as usual, and had frequent shivering fits, with occasional sweating and heats." This was probably the time when he imbibed the scarlet-fever poison; and if he had then been examined by a medical man, some symptoms of his being under its influence would most probably have been detected. "Soon after this, his mother noticed that his legs were swollen, that he

had a sore throat, and that his body was very red and hot; and she took him to a 'doctor,' who said he had 'erysipelas,' and treated him for that complaint."

He was brought to the hospital on December 6th, 1853, and the following description of him was entered in the Case Book:—"He is a very pale child, his face is highly œdematous, and he has much dropsy of the legs; his throat is much inflamed, and externally the glands of the neck are considerably enlarged; his pulse is 120, and his respirations are 28 in a minute; his urine is perfectly free from albumen."

It should be here stated that the urine of this child, though it did not contain any albumen, deposited on standing a slight, cloudy sediment, which, on being subjected to microscopical examination, displayed renal epithelium and epithelial casts in considerable quantity. It was quite clear, therefore, that there existed, at this time, an irritated state of the kidneys, which, when considered in conjunction with the other facts of the case, sufficiently explained the general dropsy. Up to the 15th, the urine continued free from albumen; but on the 19th, the report is, "the urine is highly albuminous;" and from this time, this excretion contained all those materials which are usually met with in it, in the dropsy that follows scarlet fever.

This patient very soon began to suffer from congestion of the lungs, and the glands of the neck became much irritated; both very unfavorable signs in this disease. The report of the 19th runs thus:—"Much worse; the glands of the neck are greatly swollen; there is much difficulty in swallowing; breathing greatly oppressed; pulse 122; respirations 44; the urine is highly albuminous, and contains a large quantity of lithates, with *epithelial* and *small waxy casts*, and scattered renal epithelial cells in great numbers; also a few blood-corpuscles."

An interesting point in this case was the constant existence of much more œdema of the left lung than of the right. This was explained by the fact that the boy lay almost always on the left side, and that gravitation favoured the accumulation of blood and the filtration of serum in that lung. All dropsies, as you doubtless know, are much influenced by position: for example, if a person have dropsical swelling of the ankles

simply, it is always worse in the evening than in the morning, and usually disappears after lying down a short time; and again, if there be general dropsy, and one upper extremity be allowed to hang more than the other, that limb will become the more swollen of the two.

There was a great sameness of symptoms in this patient for some days, but all through the case it was apparent, from physical signs, that the left lung was far more œdematous than the right. From the 21st, the child gradually got worse; the chest symptoms increased; and on the 23d the pulse was 120, and the breathing 60 in a minute. On the 26th, the pulse was 170, and the respirations were 70; but on the 27th, a very remarkable change took place with respect to the frequency of the pulse and breathing. This I am inclined to attribute to an alteration in the treatment made the previous day, when an eighth of a grain of tartar emetic was ordered to be given every four hours. On this day the pulse had fallen to 84, and the respirations to 40. This change, however, for the better was but of short duration; on the 28th all the symptoms had returned in their former severity, the pulse was 140, and the respirations were 52. On the 29th the numbers were 132 and 64 respectively; and from this time they steadily increased, until at length the poor child died in a state of extreme exhaustion, worn out by the rapid breathing. During all this time the quantity of the urine kept at a very low point, frequently not more than five ounces being passed in the twenty-four hours; and, during the last two or three days of his life, the little patient suffered greatly from vomiting. Unfortunately, we were unable to obtain permission to examine the body in this case; but there can be very little doubt that there was a condition of kidney and lungs identical with that of the boy Scales, the cause of death in both these patients being, as we have seen, intimately connected with an œdematous state of the latter organs.

It would seem, indeed, that, in this form of disease of the kidney with acute dropsy, as well as in that which appears to proceed from cold independently of the scarlet-fever poison, the most common cause of death is the dropsical state of the lungs and the impeded state of the circulation through these organs, with the consequent imperfect respiration.

Other causes of death, however, you will find in coma, with or without impaired pulmonary circulation, and in convulsions. Both these conditions, indeed, will occur soon after the scarlet-fever poison has entered the system, before any rash has appeared, or while it is appearing. When convulsions occur, they produce great exhaustion, and with the more certainty if the convulsive paroxysms follow each other in rapid succession; the exhaustion being, without doubt, the immediate cause of death.

Although in some degree foreign to the subject of dropsy, let me here introduce to you a remarkable case to show how rapidly the scarlatinal poison may take hold of the whole frame, producing the peculiar state of kidney, and causing death by coma in a very few hours. The particulars of the case were collected by my clinical clerk, Dr. E. Liveing, whose words I shall quote.

Case CXIV.—George Dollin, aged eleven, admitted November 4, 1854 (vol. xxxviii, p. 81). This boy had never anything wrong with him until the illness for which he was brought to the hospital, which began yesterday morning (November 3d) at seven o'clock, when he was taken suddenly ill, and called out to his mother, "Mother, I am so sick, I think I am dying."

She found that the boy had vomited at least a pint and a half of yellow frothy fluid, with red spots in it like blood. She gave him a dose of tincture of jalap about eight o'clock, which operated about an hour and a half afterwards, and brought away a quantity of slimy fluid with a greenish-yellow sediment.

At seven o'clock in the evening he began to be feverish, hot, and thirsty, and drank a pint and a half of slops, and his feet and hands became extremely cold and purple. His mother then put him into a warm bath, and afterwards wrapped him up in a blanket; but he did not perspire at all. She sat up with him all night, and he was delirious the whole time, talking, dreaming, and singing. At seven o'clock this morning (November 4th) he had another attack of vomiting, but did not vomit as much as yesterday; he was also purged, a greenish-yellow biliary-looking fluid coming away, but no blood.

When he was brought in on the morning of November 4th, he appeared quite comatose, and could not be roused; he vomited a small quantity of biliary-looking fluid. The pulse at the wrist is so weak that it can scarcely be felt; the heart beats about 184 in a minute; he breathes with difficulty, and with irregularity, if you disturb him; hands and feet purple and very cold; face dusky, and lips purple; the tonsils are large, and fauces red; there is some rhonchus at every part of the chest; he has a red blush over him. Dr. Todd thinks it will turn out a case of scarlet fever, if he survives the collapse in which he is at present. The pupils are contracted. Notwithstanding the free application of stimulants, external and internal, he died in the afternoon.

On inspection of the body, the following points were noted :—

Brain — congested, grey matter very dark, looking like the brain of a person poisoned with opium.

Kidneys—had all the appearance of the scarlet-fever kidney, the peculiar whiteness of the cortical substance, and the red congested state of the medullary portion, being well marked.

Spleen—large.

Peyer's patches in the intestine were very large and distinct.

Urine contained much bile.

It was ascertained that there had been no particular illness in the house or neighbourhood from which this lad came, with the exception of a case of scarlet fever three months previously.

This lad's brother, aged twenty-seven, was in the hospital at the time of his admission, convalescent from rheumatic fever. After the boy's death, he was allowed to go out to attend his brother's funeral. A month afterwards he was re-admitted with acute renal dropsy.

It seems to me that no doubt can be entertained that this lad died under the overwhelming influence of a large dose of scarlet fever poison, and one is forcibly reminded by this case of the prostration so often caused by the choleraic poison.

Death is often caused in scarlatinal dropsy by extensive serous inflammations.

In the following case a rapid pleurisy appears to have been the cause of death.

Case CXV.—A girl, eleven years of age, had an attack of scarlet fever a month before her admission into the hospital. The symptoms of the fever were ill defined, and the eruption was imperfectly developed. The attack appeared to be a mild one. She was treated as an out-patient, and seemed to get well. Just before she was brought to the hospital, having been exposed to cold and wet while still suffering from sore-throat and a harsh dry skin, a new set of symptoms came on. The urine became scanty, smoky in colour, and highly albuminous; it contained numerous epithelial cells, and grains of uric acid. Anasarca supervened, affecting all the parts of the body. The child complained much of headache, and was deaf. She was bled and purged, had diuretics, and was blistered on the back of the neck for the head affection.

The symptoms underwent no material alteration up to the 26th of November, when she was seized with diarrhœa, which was followed by an epileptic fit, and this was repeated twice. From that time she remained in a semi-comatose state for some days, when she was seized with dyspnœa, accompanied with delirium, both of which continued till her death. During this time her urine was very scanty, and was passed almost entirely with the motions. A day or two before her death a small quantity was obtained, and this was found to be highly albuminous, and to contain epithelial particles in great abundance.

At the post-mortem examination, the brain was found to be healthy, but hyperæmic; there was no sub-arachnoid effusion; the liver was enlarged, and there was a copious effusion of serous fluid into both pleuræ, and recent lymph was poured out upon the surface of the pleural membrane. The kidneys afforded excellent examples of the scarlet-fever kidney; the cortical substance was white, and the pyramidal portion red; the tubes were with epithelium.

From these fatal cases let me turn to one or two of a more agreeable kind, the favorable termination of which, we may hope, has been in some degree aided by the care and treatment afforded here.

Case CXVI.—The general course of scarlet fever, and the visceral irritations which the poison is apt to produce, and in which are laid the foundations of dropsy, are well illustrated by the case of a little girl in Lonsdale ward, named Alice Lyons, aged ten years (vol. xliii, p. 165), who was brought to the hospital on December 19th with the scarlet fever upon her. At the time of her admission, sore-throat was the only symptom of the disease present, and on the 20th the rash made its appearance. The only peculiarity in the case at this time was, that she had almost constant vomiting, as if the fever poison had extended its influence, as that of small-pox often does, to the mucous membrane of the stomach. The rash soon began to go off, though it did not thoroughly disappear till the 27th; but still the red colour of the fauces and tongue remained for some time afterwards, and the lingual papillæ, also, continued very red and prominent. On the 28th a slight rhonchus was audible throughout both lungs, and the urine, although it remained perfectly free from albumen, exhibited under the microscope a few *epithelial* and *small waxy casts*. The chest symptoms increased, and she became oppressed in her breathing, and suffered from cough. On January 5th the urine was still albuminous, and it also contained those further indications of renal irritation, namely, scattered renal epithelium, with *epithelial* and *small waxy casts* as before, and a few blood-corpuscles. Since these symptoms showed themselves, the daily quantity of urine varied from fourteen to sixteen ounces.

Up to the time when her chest began to suffer she was treated by diffusible and alcoholic stimulants, and nitrate of silver was applied to the throat; and, although she was extremely prostrate on admission, and the fever ran high, all the primary symptoms yielded in the most satisfactory manner. For the oppressed breathing and the pulmonary symptoms, it was determined to exhibit tartar emetic in doses of one sixteenth of a grain thrice a day. Vomiting was at first excited by this remedy, but she soon became tolerant of it, the breathing and cough improved very much, and she continued to take it till the 18th. Although the quantity of urine showed no tendency to diminish, the albumen increased, and the casts and epithelium and blood-corpuscles were still found in it, and

she complained much of pain in the back. Two leeches were now applied to the loins, and bitartrate of potass was given in scruple doses every sixth hour, the antimony being omitted. A threatening of dropsy showed itself just at this time, in an increased pallor of the face, a peculiar waxy appearance of the skin, and a slightly œdematous state; but this quickly gave way, and a very free desquamation of the cuticle took place for some days, giving countenance to the view which I have expressed to you at a former part of this lecture, as to a certain degree of antagonism between free desquamation of the skin and the dropsical state. The quantity of urine now began to increase in a very decided way, but the irritated state of kidney persisted, being indicated by the continued appearance of epithelium and casts in the urine, and the addition of pus-cells and pus-casts. All fever having subsided, and the pulse having fallen to 78, she was now ordered the tincture of the sesquichloride of iron, in doses of eight minims three times daily. Under this treatment she made rapid progress, and recovered strength and colour, but was thrown back for some days by an attack of hæmorrhage from the kidney, brought on probably by some indiscretion of diet; this yielded to the use of gallic acid in a week, and she then resumed the steel, under which she improved greatly and grew fat. On the 15th of March all traces of albumen had disappeared from the urine, and on the 25th she was reported to be quite well.

The chief interest in this case consisted in the long continuance of some of the conditions which usually produce dropsy. Despite, however, of the persistence of the irritated kidney, the dropsy made no progress, although there was a decided threatening of it about the fourth week. I cannot doubt that if this child had not been most carefully protected from cold, if the action of the skin had not been promoted and soothed by the frequent use of warm baths, and if, in fine, there had not been a pretty free desquamation of the cuticle, and also, if the child had not been well and carefully fed, dropsy would have developed itself fully in this case. Of the conditions for the production of the dropsical state, we had quite sufficiently developed the irritated kidney, less perfectly the state of blood, which doubtless would have become poorer

had the child been poorly fed, and still less perfectly the state of the skin.

Case CXVII.—The case of a little boy, named Alfred Austen, aged eight (vol. xl, p. 203), to which I referred briefly in the last lecture, will serve to compare with that just detailed, it having terminated favorably.

This child was brought into the hospital in a highly dropsical condition on December 28th. His mother could give no satisfactory account of the primary symptoms of scarlet fever. She stated that the child had been healthy until she changed her residence into a court which proved to be very damp. The first signs of illness were soreness of the eyes and drowsiness; and these were very speedily followed by swelling of the belly, face, eyelids, and legs. A brother and sister were similarly affected, and this circumstance rendered it in the highest degree probable that all three must have been exposed to the same contagion.

The child exhibited, on admission, the peculiar pallor of skin, with universal dropsy. The urinary secretion was below the normal amount—about sixteen ounces in twenty-four hours; it was loaded with albumen, and contained granular and waxy casts, and some pus-cells. The sounds of the heart and lungs were natural. The pulse was 120, and the respirations were 36. He was ordered an occasional warm bath, and one sixteenth of a grain of tartar emetic every fourth hour, with beef-tea and milk diet.

The tartar emetic at first sickened him very much, but he soon became tolerant to it, and he continued its use till the 7th of January, the dropsy diminishing, the skin acting slightly, and the urine having increased in quantity up to twenty-eight ounces.

On the 7th the tincture of the sesquichloride of iron was given. On the 13th the urine had increased to fifty-four ounces, some blood-discs and casts were found in it, but there were no pus-cells. On the 16th the urine was two pints in quantity. A marked diminution had taken place in the dropsy, especially of the abdomen and limbs. The quantity of albumen in the urine was much lessened. On the 19th the albumen was very

small in amount. On the 26th the albumen was again increased, there were no blood-corpuscles nor casts, but very numerous crystals of uric acid. On the 7th of February sixty ounces of urine were passed, and no trace of albumen could be discovered. On the 14th, however, a small quantity was again found, with crystals of uric acid and casts; this had disappeared on the 18th, and from that time the urine continued free from albumen until the 2d of March, when he left the hospital apparently quite well.

Crystals of uric acid are of very common occurrence among the sedimentary matters in the urine of children suffering from dropsy after scarlet fever. They often exist in such quantity as to give a dark smoky colour to the sediment, and even to the urine itself, and to excite strong suspicion as to the presence of blood, in cases in which it will turn out not to be present. They occur chiefly, I think, in the milder cases, and in the severe cases on the decline of the irritation of the kidney. Their presence, therefore, as it appears to me, ought to be rather regarded as of favorable omen.

Let me conclude this lecture with some remarks on the treatment of this form of dropsy.

The practitioner ought chiefly to aim at relieving the irritated state of the kidneys by such means as will not deteriorate the *quality* of the blood. You must look upon the kidneys in these cases as irritated by a poison, and that in consequence of this there is an undue afflux of blood to them, just as there is to the conjunctiva when a particle of dust gets into the eye. It can be of no use to take away blood in such a case as this. You must first get rid of more or less of the poison. In the example to which I have referred, of an irritated conjunctiva, it would be of no use to, at once, apply leeches to the conjunctiva or the neighbouring integument; the rational plan would be to remove the irritating substance by some mechanical means, and then, if the congested state remained, to employ leeches.

So, in this state of kidney, it is not advisable at first to abstract blood, either generally or locally; first, because such a proceeding will not remove the *cause* of the morbid state of the kidneys; and secondly, because it tends to deteriorate the

quality of the blood, which has been already, through the influence of the poison, rendered quite poor enough.

You must, in the first instance, endeavour to bring about the elimination of the poison through some other channel, as well as the kidneys, such as through the skin or bowels; and this will relieve the kidneys far more efficiently than any other means to which you can have recourse. For this purpose, in the commencement of the disease, whether dropsy be present or not, the frequent use of the warm bath, or the vapour bath, daily if necessary, is very beneficial, provided the patient be strong enough to bear these measures; but he should never be allowed to remain in the bath sufficiently long to induce weakness, or to diminish his strength. Baths of high temperature should be avoided; a heat of from ninety to ninety-five degrees Fahr. is amply sufficient.

With the view of eliminating the poison by the bowels, such purgatives should be employed as produce watery evacuations. Now and then you may give a dose of calomel, followed by a saline aperient; or you may use, in preference to mercury, the compound jalap powder; but all purgative medicines which cause free watery discharges by the bowels are of decided service in this form of dropsy.

You may also do good by promoting the discharge of water through the kidneys by means which do not tend to increase the irritation already existing in these organs; but, as many of you are no doubt aware, in the whole list of medicines there are none so uncertain in their action, and which, for that reason, we use so tentatively, as those classed as *diuretics*. The diuretics which appear to be most useful under these circumstances are such as appear to exert a chemical action (whatever be its precise nature)—the bitartrate of potash, for example, and the benzoate of ammonia. The free use of diluents and of the alkaline carbonates or citrates is also to be recommended.

If, after an early treatment of this kind, there still remain evidence of a congested state of the kidneys, you may then have recourse to local bleeding, by leeches or cupping, over the loins. I think it is much better to adopt this measure in these cases *late* than *early*, and I have seen much more benefit result from it when so employed than when it has been resorted to at

the commencement of the attack. I explain this on the principle already referred to: the primary congestion of the kidney is due to the direct influence of an existing poison; and notwithstanding the removal or the diminution or dilution of this, a residual congestion is apt still to be present. This may be considerably relieved by the local abstraction of a small quantity of blood, just as (to have recourse to my former simile) after the removal of a particle of dust from the eye, a state of congestion often remains, which is greatly benefited by the application of cold, or of a leech or two, or it may be, in some cases, by warm fomentations. Observe, all that I ask you to do is, if you practise bloodletting at all, do it from some well-founded principle, and not in an empirical or routine way; and always bear in mind that it is a remedy of great potency, frequently for evil, sometimes for good.

There is no special remedy, that I know of, for the peculiar state of lung that so often manifests itself in these cases, and all that you can trust to is the gradual restoration of the blood to its normal condition, and the recovery of the general powers of the patient. The way to promote this is by endeavouring to keep up the heart's action, and by upholding the general strength; so that while, on the one hand, you are endeavouring to eliminate morbid matters from the system, you must, on the other, carefully feed the patient, particularly by the well-regulated exhibition of stimulants, which, properly speaking, are nothing more than a peculiar kind of food. If wine and brandy be digested badly or not at all, they should not be given; and what I would have you bear in mind is, that alcoholic stimulants should be dealt with just as beef and mutton, and that they should be regarded as merely hydrocarbonous matter in a subtle form, to be administered carefully and by system, as you would give other aliments, not in a slovenly, irregular manner, or according to the mere whim of the patient or his nurse. Try to form a fair estimate of his power of digesting these substances, and give them accordingly.

In several cases of this dropsy I have used tartar emetic with benefit, as a medicine which determines to the skin, and which exercises a certain sedative influence upon the pulmonary circulation. It is one of the best medicines for this purpose that we

possess; but if I do not have recourse to it often, it is because it is so very uncertain in its effects on different individuals. As a general rule, I think it may be laid down, that when patients quickly tolerate it, it is an excellent remedy; but when, on the other hand, it nauseates and sickens, its action is rapidly exhausting. It is for this last reason that tartar emetic has been so vaunted by the ultra-antiphlogistic practitioners, because it frequently completely knocks down the patient and, they suppose, the disease also; but be assured that you cannot be too circumspect in the employment of this drug, for very many patients have been so prostrated by it that they have never got up again. The medicine which I most prefer for acting on the skin, in these and other cases, is the *liquor ammoniæ acetatis,* in doses of from two or three to six drachms; and it likewise acts most favorably as a sedative to the circulation. Digitalis may also be used, but only under close watching; but, generally speaking, when the lungs are involved in the manner we have witnessed in the cases referred to, more good is obtained by frequent counter-irritation of the chest at different points, and by the use of diffusible stimulants, such as ammonia, chloric ether, and alcoholic fluids, than by any other means.

LECTURE XXIV.

ON CERTAIN URINARY DISEASES.

ACUTE RENAL DROPSY.

Gentlemen,—The consideration of that form of dropsy which is especially distinguished by the name of *Acute Renal Dropsy* follows naturally upon that of dropsy after scarlet fever. Both are examples of dropsy in its most acute form. In the latter we can trace the direct influence of an animal poison, which produces well-defined phenomena, as constant and specific as those which would arise from the ingestion of arsenic, prussic acid, or strychnine. In the former we have a dropsy of a precisely similar kind, and a state of kidney also the same; but we are unable to trace up these pathological conditions to the influence of any agent introduced from without.

Let me illustrate the clinical history of an ordinary case of this form of dropsy, ending favorably, by the following examples.

Case CXVIII.—James Owen, aged forty, in the Sutherland ward, has been in the hospital some time. He is by profession a chimney-sweep—a trade much exposed to privations and hardships. For the last three years he has had winter cough, with attacks of an asthmatic character, accompanied with all the symptoms of chronic bronchitis. The existence of this congestive tendency in the lungs is a powerful predisposing cause to dropsy; and this we can readily understand, from the relation of the pulmonary to the general circulation; for any obstacle in the lungs is immediately thrown back on the right ventricle, and thence through the right auricle is communicated to the whole venous system. And when we consider with what little force the venous circulation is carried on, it is not difficult to conceive

that a slight impediment can readily cause retardation of it, and consequent dropsy.

We see, then, that the previous condition of our patient was such as to predispose to dropsy. This winter he has had one or two of his old attacks of cough, and a few days before entering the hospital he was more than usually exposed to cold. This exposure to cold, I would observe, is a very common feature in the history of these cases. Its effect was first felt on the lungs, but no doubt it had also a less perceptible influence on the skin, checking the cutaneous excretion, throwing more work on the kidneys, and thus producing renal irritation. Three or four days after the exposure, the scrotum began to swell, shortly after this the legs, and finally the effusion spread to the upper extremities and face, and was accompanied with considerable dyspnœa.

On examining him, I found his chest generally resonant on percussion; the breathing was vesicular everywhere, accompanied with more or less of sibilus, which was greatest at the posterior portion of the lungs, the congestion of which was favoured by gravitation; there was universal anasarca; the urine was highly smoky, in diminished quantity, of specific gravity 1018, and on the application of heat and nitric acid it became almost solid, coagulating much in the same way as the pure serum of blood would do, and thereby showing that this fluid constituted a large portion of what he passed from his bladder.

On his first entry into the hospital, our patient was so much depressed that there was no choice of treatment, and I was obliged to prescribe the administration of ammonia and other stimulants; under the action of which he recovered himself in a great degree, and was then able to bear the ordinary plan of treatment.

I must beg your particular attention to the order in which the phenomena showed themselves in this case:—1st, there were the previous condition of chronic dyspnœa, pulmonary congestion, and the consequent tardy venous circulation (predisposing symptoms); 2d, exposure to cold; 3d, the check to the action of the skin, consequent irritation of the kidneys, and the suppression of their secretion; 4thly, the development of anasarca, which appeared not in the most dependent parts, but in those

parts which afford least resistance to the effusion: frequently, instead of the effusion seeking the most dependent part, the face is first affected, but very commonly the scrotum is its first seat: a common order is, the scrotum, eyelids, face, then the lower extremities, and finally, the upper extremities. But whether this order be followed exactly or not, the fact most worthy of your attention is, that the development of the dropsy in these cases is in a great measure independent of position; and this circumstance alone ought to lead you to the suspicion that the dropsy is of an acute character; 5thly and lastly, we have to observe the condition of the urine—its small quantity, smoky colour, and the abundance of albumen in it, showing the escape of the serum of the blood, and of its red particles.

Now, the train of phenomena afforded by this case very closely represents that which is ordinary in the generality of cases of *inflammatory* or *acute renal* dropsy, so that I may say, *ex uno disce omnes;* one case of this kind carefully studied gives you a considerable insight into the clinical history of the disease. And as the history of this patient up to this point affords a fair representation of the average cases, so will its further progress illustrate the course which this malady generally takes in its milder forms.

Its principal difference from the ordinary class of cases consists in the existence of an antecedent pulmonary irritation, which proved the more favorable to the development of dropsy. Irritation and congestion of the lungs, indicated by more or less rhonchus and sibilus, are, however, frequently present in cases of acute dropsy, being set up at the same time as the cutaneous irritation, and doubtless originating in the same cause.

On my first visit to our patient (on the 10th), I ordered him a hot-air bath: this was attended with almost immediate benefit. The next day (11th) the dropsy was much diminished, but it was still abundant, and I prescribed a purgative, choosing the compound powder of jalap, on account of its hydragogue properties, and because the bitartrate of potash which enters into its constitution favours diuresis. We found, after the frequent use of the hot-air bath, sudorifics, and the application of free counter-irritation to the chest, that in nine or ten days the dropsy had disappeared, finally leaving him on the 21st: his

urine, however, continued smoky, but was much increased in quantity. Examined by the microscope, it was found to contain blood corpuscles, fibrinous casts of tubes, epithelial cells, and crystals of uric acid; so that in this case the smokiness was kept up by the same two causes which originally produced it—namely, congestion and irritation of the kidneys; the specific gravity of the urine was 1015, and the albumen was much diminished.

I shall add, for the sake of further illustration, another example of this disease, not preceded by any chronic bronchial affection.

Case CXIX.—A woman, aged twenty-five, in the sixth month of her pregnancy, had always enjoyed good health until a month before her admission into the hospital, when, from exposure to cold, she became troubled with cough and hoarseness. A fortnight after this, she observed a swelling of the labia pudendi and of the legs; her urine became scanty, and she noticed a red sediment in it. On her admission, there was considerable œdema of the labia and of the lower extremities, accompanied with distinct signs of fluid in the abdomen. The urine was scanty, smoky, and highly acid; it was turbid and abundantly albuminous; on standing it deposited a sediment of lithate of soda, and it had a specific gravity of 1027. It was not examined microscopically. The skin was dry. In this case I endeavoured to promote the healthy action of the skin by the use of warm baths and by the administration of tartar emetic and Dover's powder. Under this treatment the urine soon became free from albumen, the dropsy entirely disappeared, and in twelve days the patient was well.

It is likely that the weight and pressure of the gravid uterus in this case determined the dropsy to the lower parts of the body.

Such, then, is the ordinary course of events in mild cases of acute renal dropsy. I shall now proceed to review, in detail, the various organic derangements which accompany and complicate this form of dropsy.

These may be arranged in two classes:—first, the *intrinsic*, and, secondly, the *extrinsic*.

The first or intrinsic derangements stand to the dropsy in the relation of cause to effect. They are those which belong specially to the peculiar anatomical condition of the kidney, which is the same as that which occurs in dropsy after scarlet fever. A large accumulation of epithelium takes place in the renal tubes, owing, probably, to its being formed more quickly than it can be expelled. In short, we find that there goes on during life, in this disease, a desquamative process, affecting the kidney, just like that of which the skin is the seat in scarlet fever. The simultaneous, or nearly simultaneous, occurrence of the analogous conditions of the skin and kidney is the ordinary course of events in the scarlatinal dropsy; but in the form now under consideration we do not find the state of desquamation affecting the skin, although great dryness and harshness of it frequently occur, showing a state of irritation of that integument.

A greater or less degree of congestion of the kidneys accompanies this accumulation of the epithelium. The Malpighian bodies are more or less enlarged, and all the small vessels dilated. The greatest accumulation of blood takes place in the Malpighian tufts, and in the minute vessels of the pyramids; and for this reason it is, that these latter parts of the organ are very red, while, owing to the accumulated epithelium in the tubes, the cortical portion is white.

This state of distension of the blood-vessels favours the occurrence of a phenomenon which often forms a very serious complication of this form of dropsy. If the primary condition of irritation or inflammation does not terminate by an early resolution, hæmorrhage is very apt to supervene. The vessels, long dilated by the pressure of an undue efflux of blood, suffer in their contractile power, and, becoming weakened, give way at various points: nor do they recover until the general nutrition of the kidney is restored by the discharge of a large quantity of the epithelial particles which surcharged its tubes, and contributed, with the increased flow of blood, to create the enlargement of the organ.*

In some cases pus is found in the tubes, affording evidence of

* See the cases in Lectures XCIV and XCV, illustrating the connexion of hæmaturia with this form of dropsy.

a suppurative process having taken place. We obtain proof of this formation of pus during life by finding the peculiar pus-corpuscles along with other matters in the urine. And when this occurs, the lungs are very apt to exhibit signs of an œdematous condition, which interferes greatly with the respiratory process, and, by impeding the heart's action, increases the dropsy and favours a fatal termination.

The second class of complications connected with this form of dropsy are—

First, inflammations of serous membranes. The pericardium and the pleura are those which are most subject to these inflammations; but sometimes the peritoneum is affected. Pericarditis, indeed, is a frequent concomitant of albuminous urine in all forms of diseased kidney, as was some years ago well proved by the late Dr. Taylor from a large induction of cases. These acute inflammations are, however, more common in the acute dropsies than in those which are associated with the various states of chronic disease of the kidneys.

Effusions are also apt to take place into the serous cavities with or without previous inflammation. The peritoneum, although the least liable to inflammation, is the most subject to these effusions.

The following is an interesting example of the simultaneous supervention of pleurisy and peritonitis, with slight pericarditis.

Case CXX.—The patient was a fine-looking young man, aged twenty-three, of regular habits, and who had never suffered from privations. He was admitted into King's College Hospital, October 12th, 1840 (vol. i, p. 262).

After exposure to cold three weeks before his admission, universal anasarca came on, beginning at the face and eyelids, and spreading quickly to the scrotum, limbs, and trunk, accompanied with much pain in the back, and a marked diminution in the quantity of the urine, which never exceeded a pint and a half in twenty-four hours, and often fell much below that amount. The urine became nearly solid under heat and nitric acid; it had a smoky hue, and its specific gravity ranged between 1015 and 1020. In addition to the external

dropsy, there was a small quantity of fluid in the peritoneal cavity.

Blood was taken freely from the loins by cupping, and small doses (one eighth of a grain) of tartar emetic were given three or four times a day. Half a grain of the extract of elaterium was administered once daily. Although he was freely purged, no material improvement followed this treatment, excepting that the kidneys began to act more freely, and as much as four and five pints of urine were passed in the day.

On the 22d he was bled from the arm to ten ounces, and, on the 27th, he was bled again to six ounces. On the 12th of November the dropsy remained much the same; there was extended dulness over the region of the heart, giving rise to the fear that some pericardial effusion had taken place. The bitartrate of potass was now exhibited, and he continued to pass four points of urine per diem.

At midnight, on the 16th, our poor patient (who was a fine, able-bodied young man, and bore his illness with great calmness, and greatly interested us all) was seized with severe rigors and vomiting of bilious matter, accompanied with severe pain in the left side, opposite the seventh and eighth ribs. Between eight and nine o'clock on the following morning (the 17th) the shivering recurred, and lasted half an hour. He was now much troubled with diarrhœa. The breathing also became quick and difficult; a very tympanitic condition of the abdomen came on, but there was no pain on pressure of that region; pulse 130. Leeches were applied to the abdomen, and opiate enemata were given.

On the 18th the abdomen was swollen and tender all over; the breathing was hurried; the abdominal pain was much increased by deep inspiration. The quantity of urine decreased considerably. On the 19th the diarrhœa was much less, but the abdomen had become much more painful; urine very scanty; much thirst. Twenty-two leeches were again applied to the abdomen, and opium was given freely. Hiccup came on, and caused much distress. On the 22d the patient sank rapidly, but retained his consciousness to the last.

On opening the body the peritoneum was found everywhere covered with flakes of recent lymph: it was very abundant on

the peritoneal surface of the bowels, and on the concave surface of the diaphragm. Recent lymph was also effused upon the pleura, but in less quantity than on the peritoneum, and serum was found in considerable quantity in both pleural sacs. There were some patches of recent lymph on the cardiac layer of the pericardium. The brain and its membranes were healthy. The kidneys were about one fourth larger than natural, and on section presented the white cortical and red tubular portions of the scarlet-fever kidney. Urea was found in the blood taken from the heart, and in the serum effused into the pleuræ.

It is worthy of note, with reference to this case, that all this acute inflammation came on notwithstanding free local and general bleeding practised before the symptoms were developed.

I made a very careful examination of the kidneys; and, so far as I know, this examination led to the first recognition of the structural difference of the diseased kidney in acute dropsy from that of the large mottled kidney (now called fatty) first described by Dr. Bright. This examination was made in October, 1840; and an extract from the notes then made was published in a clinical lecture in the 'London Medical Gazette,' in December, 1845. I quote the extract:—"On referring to my notes of the microscopic examination, I found it stated that the tubes were enlarged, and filled by an abundant epithelial formation, which seemed to distend them. On making a transverse section, the dilated state of the tubes was very conspicuous, and the epithelium could be pressed out of them, leaving only their dilated walls. Several Malpighian bodies were enlarged."

Although the functions of the brain are often disturbed, it is very rare to meet with arachnitis in acute renal dropsy. States of delirium, and even mania, often supervene, but these are due to a disturbed nutrition of the brain itself; dependent either on the watery and pale state of the blood, or on the irritation of the brain by retained urinary elements. When you examine cases of this description after death, you find no evidence of recent serous inflammation, but only a pale condition of the brain, which is of less than its normal bulk, and is surrounded with more subarachnoid fluid than is usual in the state of health. The delirium in these cases is very analogous to that which occurs in rheumatism and gout.

You may have, as a complication of this form of dropsy, an irritated state of liver, coexistent with the analogous state of kidney; and it is not improbable that this may be the reason of the greater frequency of serous effusion into, or of dropsy of the peritoneum, than of the other serous sacs. In all instances in which the liver is irritated, you may expect to find some degree of yellow discoloration of the skin, and the dropsy not confined to the areolar tissue, but also affecting the peritoneum.

Sickness and vomiting are often present in these cases, and also in the more chronic states of renal disease, and they generally indicate an irritated condition of the gastric mucous membrane due to retained urinary elements.

What conclusion may we come to as to the *pathology* of acute dropsy? We know that the kidneys are in a state of congestion, which is obviously denoted by their appearance; the cortical substance is red and congested, the Malpighian bodies being large and full. The redness of the cortical substance is in strong contrast with the whiteness of the tubular portion, which is caused by the abundance of epithelium with which the tubes are filled. If examined with a microscope in an early stage, we can see the epithelium quite filling the tubes. We thus obtain clear evidence of an irritated state of kidney, and we also know, from the previous history of the case, that there has generally been such exposure to wet or cold as would be likely to arrest the excretion by the skin. Thus we have very much the same conditions induced as in dropsy after scarlet fever—namely, an irritated state of kidney, and abnormal state of skin.

The secretion of the skin being, no doubt, checked, the organic products, which should have passed off in it, make their way out through the kidneys, there acting as a source of irritation: they produce congestion, and this congestion so interferes with the function of the organ that its secretion diminishes: the water that should be eliminated does not escape, but accumulates in the blood; and this watery blood transudes the parietes of the capillaries, while the particular portion of the areolar tissue into which it shall be poured—the subcutaneous—is determined by the irritated state of the skin, which attracts blood to it in large quantity. The pathology of

this form of dropsy is, then, in nearly all essential points the same as that of dropsy after scarlet fever.

What *prognosis* should we be led to adopt in such cases as these? To this question I answer, that, on the whole, our prognosis may be favorable: death is not common, unless the irritation of the kidney proceeds so far as to produce those extensive disorganizations to which I have referred; but, taking the ordinary class of cases, this kind of dropsy is not to be considered a formidable disease. In forming our prognosis, however, the previous condition of the patient must be taken into account. It often happens that a man has renal disease creeping gradually on without any symptom of its presence, till, on a sudden exposure to cold, active congestion of the kidney is induced, and the secretion of that organ receives thereby a greater or less check: of course in such a case the ultimate danger would be much greater than if the kidneys had previously been in a perfectly healthy state.

In the fatty disease of the kidney, the earlier changes take place slowly and insidiously, and often without a single symptom to indicate that anything is wrong with the kidney. There are many people at this moment walking about London, who would be very angry at being told that they are otherwise than healthy, yet with whom this disease is creeping on, and whom it will sooner or later kill. The deposit begins to be formed in a few sets of tubes; it gradually extends itself, producing partial congestion of the kidney. At first these congestions are not sufficient to affect the physical condition of the urine, which, if examined at this period, would not afford the slightest indication of disease. By-and-by, some of the Malpighian bodies become so filled by the retardation of the flow of blood through them, that the serum filters through them, and, passing off with the urine, renders this secretion albuminous. This escape of serum offers some relief to the congested parts of the vascular system of the kidney; and if the cutaneous secretion be fairly kept up, and the digestive organs act normally, the patient may go on for a considerable time—sometimes, perhaps—for years—without being aware that there is anything wrong with his kidneys. But let any check to the perspiratory action of the skin take place, as from exposure of cold, and immediately the

renal congestion becomes general, and dropsy quickly occurs; and these symptoms show themselves the more rapidly, and the more completely, because of the previous partially congested condition of the kidney, and the more or less impoverished state of the blood.

Cases such as these are of very frequent occurrence in the London hospitals, and many of the instances of cured or relieved Bright's disease are of this description. The patient receiving the care and comforts of an hospital, his skin quickly resumes its normal action, the kidneys cease to be congested, the urine flows freely, and the dropsy disappears.*

Now and then copious hæmorrhage takes place, as I have said. How does this influence the prognosis? Unquestionably it would do so unfavorably. From my own experience I should say that, provided you spare your patient's strength, and do not subject him to too violent antiphlogistic treatment, this copious hæmorrhage is not so much to be dreaded as might be imagined. If pus appears in the urine, I should look upon the case much more seriously. The continuous appearance of pus, derived from the kidney, in notable quantity in the urine, especially when accompanied by an œdematous state of the lungs, is one of the most unfavorable circumstances that can occur in cases of this kind, inasmuch as it indicates a tendency to that kind and degree of disorganization which does not admit of repair. But even the presence of pus need not lead you to take a very gloomy view of your patient; for many cases recover after the appearance of this product in the urine.

Treatment.—The same principles of treatment apply to the management of these cases as to those of dropsy after scarlet fever.

The first and most important object should be *to promote the action of the skin.* We know that there is that sort of mutual relation between the kidneys and the skin, that embarrassment of the one leads to embarrassment of the other: hence any relief to the irritated state of the one affords great help to the restoration of the other. In the present case, the failure of the cutaneous action has embarrassed the kidneys: hence the great thing is to promote free sweating, and this may be best done by

* See Case CVI, Lect. xix, p. 386.

the hot-air bath; or, if you fear its debilitating effects (which are to be taken into account in weakly subjects), you may substitute the warm bath.

You should, next, endeavour to obtain relief to the congestion of the kidneys. This is in part effected by restoring the function of the skin. Local bleeding by leeches and cupping will do much in some cases, but little in others. Now and then, when your patient is plethoric and robust, you may, to save time and trouble, take some blood from the arm, but these, you must remember, are exceptional cases. *As a rule,* general bleeding is better avoided. One of the symptoms which would lead me to have recourse to general bleeding in a case of this description is congestion of the lungs; but I would not adopt this practice unless the patient were young, robust, and plethoric, and had been previously in a perfectly healthy state. And even in such cases *large* venesections are much to be deprecated, as tending to debilitate the patient and to weaken those processes of nutrition to which you must look for his restoration to health.

3d. Purgatives, as indirect means of relieving the congestion of the kidneys, are of great value; they eliminate water and various morbid matters.

4th. Sudorifics may also be employed, and of these the best is the liquor ammoniæ acetatis, in at least half-ounce doses, two or three times a day. This may occasionally be combined with tartar emetic. Dover's powder is generally less suitable, on account of the tendency of the opium which it contains to check the secretion of urine.

5th. Diuretics. When the congestion of the kidney has been relieved, diuretics may be given with advantage. Of all this large class, the best, I think, is the common cream of tartar, in doses of from half a drachm to a drachm frequently during the day. If you give larger doses, it acts on the bowels, and being carried off by them, does not prove a diuretic. Benzoate of ammonia is often very efficacious. Digitalis, with due precaution, is also very useful; of digitaline I am unable to speak with any confidence, but I suspect it is a less manageable drug than digitalis.

Irritative diuretics (as cantharides, squills) must be avoided in cases of this kind, for obvious reasons, although they are very

serviceable in other forms of dropsy. Broom tea, or the compound decoction of broom of the London Pharmacopœia, is also useful; and the latter probably derives some advantages from the taraxacum which it contains. The diuretics first mentioned, however, are unquestionably the most to be relied upon, and the most efficient.

In the treatment of acute dropsy, I must caution you against the use of mercury. This mineral, so valuable in some diseases, is in these cases useless and even mischievous; at any rate, I have failed to observe any benefit from it. For these reasons it is better to abstain from its employment, unless occasionally you may have recourse to it as a purgative. But even for this purpose there is no absolute need for you to use it, as there are many other medicines which answer quite as well, if not much better. Moreover, in all cases of imperfect action of the kidneys, whether acute or chronic, patients become quickly salivated, and the salivation is very apt to prove troublesome and painful; large sloughs often forming in the mouth, and the fœtor of the breath becoming intolerable to both the patient and his attendants.

LECTURE XXV.

ON DROPSIES.

CARDIAC DROPSY.

Gentlemen,—Pursuing the clinical illustration of dropsy, I propose to-day to offer you some observations on *cardiac dropsy*, with special reference to a case of this affection at present under treatment in Fisk ward. The dropsy in this case is mainly dependent on heart-disease, but there are present various other phenomena, which partly tend to complicate, and partly to elucidate, the diagnosis, but which are all in a high degree both interesting and instructive. These are pericarditis, renal disease, a congested state of the liver, dependent on the disease of the heart, and other slighter pathological conditions, to which I shall advert as we proceed.

Case CXXI.—The patient, Alfred Baylis (vol. xxvi, xxvi,a), is a young man, aged thirty. His occupation has been that most unhealthy one, a house-painter, and he bears, in the blue line round his gums, the badge of his trade. Like many of his occupation, he has been intemperate in his habits; and to this, most probably, his present condition is mainly referable.

He was admitted on the 11th of November, suffering from palpitation, shortness of breath, and emaciation, which quickly supervened upon a profuse attack of hæmoptysis, that occurred two months previously. He had been an out-patient of one of the London hospitals; and five weeks before his admission here dropsy came on in his lower extremities, and has been steadily extending upwards ever since. Under these circumstances he applied for admission. Shortly afterwards he suffered another access of hæmoptysis, and lost a large quantity of blood. These

two attacks of hæmoptysis have produced great pallor and anæmia. The patient suffers likewise from distressing dyspnœa, amounting to *orthopnœa*. Upon any attempt to assume the recumbent posture, he is at once compelled, by the increased difficulty of breathing, to sit up, and in this position, as you have doubtless remarked, he remains day and night. This orthopnœa has been from the commencement a prominent symptom.

The great loss of blood, of which our patient has been the subject, has mainly promoted another symptom, amaurosis. I say "promoted," because I do not suppose the anæmic state to be the sole cause of the imperfect sensibility of the retina. Our patient has chronic disease of the kidneys, and the impure state of blood consequent on this disease often gives rise to more or less of an amaurotic condition.

This symptom varies with the condition of the heart. When he suffers much from palpitation, his sight is worse; when the palpitation is less, it is better; showing the dependence of the symptom on the state of the circulation. The fact is, the blood has become poor, and this impairment of quality causes any slight deficiency in the quantity sent to an organ, from inefficient action of the heart, to be felt by it more readily than if the blood were in its normal condition.

Associated with this symptom is another curious one, namely, a remarkable prominence of the eyeballs. This state of the eyeballs seems to have some curious connexion with cardiac disease. It is most frequently seen in women; and in them it is generally met with in connexion with an enlarged thyroid gland. The cause of these associations I do not attempt to explain.*

* I have seen in so many instances, especially in women, the concurrence of prominent eyeballs, rapid and vehement action of the heart, and enlarged thyroid, that I have long looked upon this aggregate of symptoms as constituting a special disease. It seems to me most probable that the primary evil is the enlarged thyroid; the altered condition of the numerous ramifications of blood-vessels through this gland gives rise to the morbid state of heart; the active nutrition going on in it drawing largely on the blood supply, and accelerating the action of the heart, while the diseased state of the blood-vessels (probably similar to that which Dr. Johnson has described in the small renal arteries, in cases of contracted kidney) creates obstruction, and consequent hypertrophy of

In looking into the previous history of our patient, we find that at the age of nineteen he suffered a slight attack of rheumatism. We can get no distinct evidence that the heart was at that time affected; but it is probable that, at the same time with the rheumatic attack, there took place some slight organic affection of the heart, either pericardial or endocardial, which his subsequent unhealthy occupation and intemperance have tended to increase.

His state on admission into the hospital was as follows:—Great exsanguineousness; breathlessness, orthopnœa. Heart's action regular; pulse 84, small. There was an unnatural extent of dulness over the cardiac region, and moreover, the pulsations of the heart were visible in the region of the scrobiculus cordis: they were likewise distinctly felt in the cardiac region. In some cases the heart's action is felt only in the scrobiculis cordis, and not beneath the left mamma; and in such there is no evidence of enlargement; for there may be, and probably is, some cause pushing the heart to the right side, so that the altered seat of the pulsations is due to change in the position of the entire organ. But when the pulsation of the heart is felt in both regions we have evidence of enlargement, and of its being, at least, the right ventricle that is enlarged; for while the left remains stationary in its normal situation, the right has extended still further to the right side. Those of you who accompany me regularly through the wards of the hospital may notice, that in all cases of dyspnœa I make it a rule to place my hand over the scrobiculus cordis, to feel if I can detect any cardiac pulsations there. My attention is particularly directed to this point in cases of long-standing asthma, and never in these cases is it altogether absent; and especially if the asthma have produced emphysema, when there is not only expansion of the right ventricle by dilatation and hypertrophy, but a pushing of the

the heart. This affection has not escaped the observation of many practised observers. It has been well described by the late Dr. Graves, by Sir H. Marsh, and recently by Dr. Stokes, in whose pages the reader will find a very complete description of the disease—'Diseases of the heart and Aorta,' p. 278.

The prominence of the eyeballs in these cases is probably due partly to actual enlargement of them, and partly, perhaps, to an enlarged or varicose condition of the orbital veins.

whole heart to the right side from the enlargement of the left lung.

We noticed also in this case a friction sound accompanying the systole and diastole, heard below and to the left of the nipple, over a space very circumscribed in extent. There was no bellows-murmur to be heard, either at base or apex. A phenomenon, connected, in its indications, with the beating of the heart in the scrobiculus cordis, was observed, namely, venous pulsation in the neck. This man has very large external jugulars, and you may see them pulsating distinctly even at some distance from the bed.

This phenomenon of venous regurgitation in the neck is due to a reflux of blood during the systole from the ventricle through the auricle into the venous system. In consequence of the obstruction in the lungs, the ventricle, already considerably dilated, cannot completely empty itself, and ere long the tricuspid valve, becoming incapable of performing its office perfectly, allows some of the blood to regurgitate, and thus to escape through the auricle into the veins. The proper source of supply to these veins is from their distal extremity. If, then, you put your finger in the course of that supply,—that is, to some distal point where the pulsation is observed,—the vein ought to become empty, because its supply of blood is thereby cut off; but in this patient it does not; it continues full, and its pulsations persist; hence the waves of blood which produce the phenomenon of pulsation must be propagated from the cardiac extremity of the vein.

There are many cases of heart-disease in which the jugular veins do not pulsate, but merely remain full below the point pressed upon. In such cases there is regurgitation, but not to such a degree as to cause pulsation. When you press on the external jugular vein of a healthy person, it will instantly become empty below the point of pressure; a slight dilatation of the right cavities is sufficient to allow of enough regurgitation to keep the vein full below the point of pressure; nay, I have no doubt that this may occur even under a dilating cause of quite a temporary nature, such as great efforts in lifting and throwing heavy weights, rowing, walking in a mountainous country, blowing wind instruments, &c. &c.

In extreme cases of venous regurgitation, when the cavities are permanently dilated, and the superficial venous system is well developed, the pulsation will be visible, not only in the jugulars, but in all the superficial veins of the neck and chest, and even in those of the arms. As regards the lungs, there was evidence, in this case, of œdema, in a certain degree of dulness on percussion over their bases posteriorly, which was most marked on the right side. Rhoncus and crepitus were also heard in these regions, but at the apices the breathing was distinct.

The liver could be felt extending slightly below the margin of the ribs; and there was ascites, dependent, no doubt, on the congested state of the liver. On examining the urine we found it acid, of specific gravity 1015, and heat and nitric acid gave evidence of the existence of albumen. When the fresh urine was allowed to stand, a scanty and flocculent deposit was formed, which, under the microscope, was found to consist of blood-corpuscles, epithelium (chiefly renal), a few pus-cells, and a great many casts, for the most part granular and transparent, some containing cells loaded with oil-globules; but these last were not very abundant. Now this is a state of things under which we should not expect our patient to survive long; yet here we see him remaining very much in *statû quo*, if anything, indeed, a little improved, until the present time (January 16th), a space of two months from his admission.

Let us now analyse these symptoms more minutely, and endeavour to determine their nature and indications, and the conclusions which can be fairly deduced from them.

The *palpitation* was no doubt an irregular rate of action. When the heart beat quickly, he had what he called palpitation; when more slowly, the palpitation ceased. When patients speak of palpitation, they often mean different things; sometimes they signify by it deranged rhythm, either complete intermission, or irregularity of action; and sometimes, as in the present case, an increased rate of action without any derangement of rhythm. Our patient has not suffered from any remarkable rapidity of the heart's action, except in the above-mentioned temporary way.

Orthopnœa is almost always connected with imperfect circulation through the lungs. This imperfection of the pulmonary

circulation may be either primary, from disturbed action of the lungs, as in asthma, or secondary, from imperfect action of the heart. In asthma no doubt there is, frequently, an anatomical change in the lungs; but let me guard you against the idea that in this lies the essence of the disease. The primary affection in asthma is in the nervous system or in the digestive organs, or both; irritation in the latter will produce the asthmatic spasm through the former, but primary irritation of the nervous system will equally give rise to it, under physical or even mental causes. But these frequent attacks of spasm may induce anatomical changes in the lungs (emphysema), which, if the attacks continue over a long period, may become permanent. When the heart is primarily affected, the embarrassment of the respiration, which is secondary, has received the name of *cardiac asthma*.

In connexion with the disturbed breathing we found *congestion* of the lungs. This no doubt was chiefly dependent on the heart, through the imperfect action of which the blood was thrown back on the lungs. To this the hæmoptysis is to be attributed; and it is most probable, if we could now examine the lungs anatomically, we should find indications of pulmonary apoplexy, and of rupture of pulmonary vessels, in the characteristic currant-jelly-like spots.

The beating of the heart in the scrobiculus cordis, and its interpretation, I have already referred to.

The *dropsy* is a result of which I shall say more by-and-by.

Diagnosis.—Now can we, from these general symptoms, make a diagnosis without reference to the physical signs? I think we can form a diagnosis which shall be sufficient for all practical purposes; and I recommend you, as a valuable clinical exercise, to endeavour to diagnose from symptoms before you have recourse to auscultation; for, by so doing, your attention will be directed to the disturbed *functions*, and you may thus receive important suggestions for treatment. Those who direct their attention exclusively to physical signs often overlook these derangements of functions, and therefore miss some valuable indications which are not only of great value for treatment, but which give a greater insight into pathology.

Were I to proceed on the data afforded by the *g enera*

symptoms alone in this case, I should diagnose, first, *dilatation of the right side of the heart:* the pulsations of the jugulars, and the beating of the heart in the scrobiculus cordis, both indicate this. The pulsation of the jugulars, if proved to be regurgitant, is conclusive evidence of dilatation of the right ventricle; no other condition of heart is capable of producing this phenomenon. But we must be careful to prove that the venous pulsation is regurgitant, as it may be due to other causes: it may depend on the impulse of the heart being transmitted through the capillaries; or it may depend on the contiguity of a large artery which communicates its pulsations to an adjacent vein; we must, therefore, be careful to determine its nature before we attach importance to its indications. Now, by making pressure on the vein, we obtain sufficiently conclusive evidence as to the nature of the pulsation. If the vein empty itself on the cardiac side of the point pressed upon, and yet the pulsation continue in the empty portion of it, we infer, and with the highest probability, that it arises from some contiguous artery; and this conclusion is rendered certain, if the pulsation ceases on stopping the circulation in the artery. If, on making pressure, the pulsation continue only on the distal side of the point of pressure, we may infer that the pulsation is due to the transmission of the pulse from the heart throughout the circulation, and especially if it be perfectly synchronous with the heart. But if the vein continues full between the point of pressure and the heart, then we know, whether there be pulsation or not, that that vein must receive blood from its cardiac side; and this it can only do by regurgitation from the right side of the heart.

You are doubtless aware that a slight regurgitation always takes place from the auricles into the veins in the normal state, and that this regurgitation is synchronous with the auricular systole. But when the ventricle and auricle are dilated, the amount of regurgitation is increased in proportion to the degree of dilatation of the cavities: blood regurgitates from the ventricle into the auricle, and from the auricle into the veins.

The beating of the heart in the scrobiculus cordis is also indicative of dilatation of the right ventricle, because by its dilatation this cavity enters into the formation of the apex of the organ, and the apex thus enlarged extends to the scrobiculus.

But before we can affirm that the beating of the heart in the scrobiculus is due to the dilatation of the ventricle, we must be careful to ascertain that there is no displacement of the heart, which may be easily determined by seeing that its impulse is felt in the normal, *as well as* in the abnormal, region.

So far, then, we may affirm that our patient suffers from dilatation of the right side of the heart. Now dilatation does not arise spontaneously. There must be some dilating force, and that force is the backward pressure of the blood. When the muscular tissue of the heart is in a weak state, we can conceive that the walls of the ventricle may yield readily, without supposing any great increase in the ordinary pressure of the blood. But, in general, dilatation arises from some obstacle to the flow of blood from the ventricle; and in proportion to the resistance which that obstacle affords will be the backward pressure of the blood on the surface of the ventricle,—in other words, the amount of obstacle will give the degree of the dilating force. In every case of dilatation of one of the heart's cavities, then, you must look for this dilating force,—this obstacle; and you will find it either at the orifice through which the cavity expels its blood, or in the capillaries of some important organ, or in some defective condition of the valves or the orifices of the heart. Dilatation of the *right* cavities of the heart rarely arises from diseases of their own orifices; for obstructive disease of these orifices is amongst the rarest of cardiac lesions. It is otherwise with the *left* cavities: experience tells us that the obstructions which the right ventricle has to encounter are either seated within the circulation through the lungs, or are due to some derangement of the circulation through the left side of the heart.

There is yet another cause which may bring about dilatation as well as hypertrophy of the right ventricle, because it may produce the same change in all the cavities of the heart; that is, extensive or universal adhesion of the opposed surfaces of the pericardium. How it happens that obliteration of the pericardial sac can produce these changes, I do not attempt to explain; but that it does so, there can be no doubt.

Let us now inquire what may be the cause of the dilatation of the right ventricle in this case. I have said that the most

common causes of this morbid condition are obstructed pulmonary circulation, or obstructed circulation through the left side of the heart. The most common causes of obstructed pulmonary circulation are the asthmatic state, or chronic bronchitis, or that which is a frequent consequence of both the asthmatic state and chronic bronchitis—emphysema of the lungs. We have no indications in this case of any of these conditions. The history of the patient shows the absence of the one, and the physical signs demonstrate the absence of the other. Is there, then, any disease of the left side of the heart sufficient to create this dilatation?

On first seeing this patient, I hazarded a guess, from the character of the pulse, that there was *mitral* disease at the foundation of his malady. The peculiarity of pulse which suggested this notion was, that it was small, produced by a small stream, apparently inadequate to the size of the artery, and not proportioned to the action of the heart. Such a state of pulse does frequently co-exist with mitral disease; but so various are the conditions capable of producing a small feeble pulse, that a diagnosis based upon that sign only must necessarily be only a guess. Now such mitral disease might be either *regurgitant* or *obstructive*,—either of the two would affect the right side of the heart. And how? By affording an obstacle to the return of the blood from the lungs. Whether the blood is unable to advance in consequence of a *contracted* mitral orifice, or whether it flows back in large quantity into the auricle, through a patent mitral valve, it is evident that an impediment is offered to its passage from auricle to ventricle, and, therefore, to its return from the lungs to the auricle; and when this has continued some time, the right ventricle, being exposed to a degree of pressure greater than customary, yields and gradually expands, until at length it assumes a permanently dilated state.

Is there mitral disease here? and if so, is it regurgitant or obstructive? The dyspnœa favours the idea that it is obstructive, because there is no form of heart-disease in which the dyspnœa is so great as in obstruction at the mitral orifice. Very often, when the disease is of this nature, sudden and terrible dyspnœa is the first evidence of its existence: the patient is suddenly awoke out of sleep by a breathlessness that seems

as if it would kill him : he starts up in bed and gasps for breath till the paroxysm gradually subsides. Such a first attack is generally the precursor of similar ones, increasing in severity and frequency, only kept off by constant watching and the upright position, till at length the dyspnœa becomes constant, and the case terminates with pulmonary apoplexy.

But, in the absence of this disease of the mitral valves, is there any other way of explaining the occurrence of the dilatation of the right cavities of the heart? The only explanation I can offer is, that there are hypertrophy and dilatation of the left cavities connected with obstruction in some part of the systemic circulation, or dependent on adherent pericardium, and that the impeded circulation through the left cavities obstructs that through the right chambers, and induces dilatation and hypertrophy of them.

So far, then, for the indications afforded by the general symptoms: let us now turn to those other symptoms which, although not more physical than the former, are technically called *physical signs.*

A careful observation of the physical signs will serve to confirm or correct our inferences from the symptoms, and help us to adopt a more decided diagnosis. The physical signs themselves were these: extensive dulness over the præcordial region, indicating enlargement of the heart; the first and second sounds were found to be essentially healthy, and unaccompanied with bellows-murmur, as well at the base as at the apex. The absence of *bruit* at the base affords strong presumptive, although not positive, evidence that there is no *obstructive aortic* disease: also its non-existence at the apex denotes that there is no *regurgitant mitral* disease; for if there were, we should hear a systolic bellows-murmur in this situation—*i. e.*, unless the heart's action were very weak. It is only in a state of great feebleness of the ventricular contraction that there would be no bellows-sound where there is mitral regurgitation; but with our patient the heart's action is not at all feeble.* Now, is there any diastolic bellows-sound to indicate obstruction at the mitral orifice? We find none. Are we, then, justified in denying

* The existence of even a well-pronounced mitral murmur (*i. e.* of a murmur heard specially at the apex and beneath the left scapula) is not conclusive evidence of mitral regurgitation. Extensive deposit on the infundibular surface of

the existence of disease of the mitral valve? Certainly we are not, for those cases of unquestionable obstructive mitral disease in which a diastolic murmur is heard are comparatively few: there must be a certain amount of force in the auricle for its production, and unless this exists the sound will not be heard. The value of this evidence, therefore, is merely negative: it does not prove that there is no obstruction at the mitral orifice, but only that the impulse given to the blood by the auricle is not sufficient to elicit a sound from the existing obstruction. So far, then, we receive really no aid in determining the question of the existence of obstructive mitral disease from examining the sounds of the heart. We have good reason, however, to believe that there is no mitral regurgitation; and as to the question of mitral obstruction, we have no further evidence than what the symptoms afford.

This evidence is undoubted as to the existence of obstruction; but it is not sufficient to determine the exact seat or nature of the obstruction. By the aid of auscultation we can determine that it is not mitral regurgitation: it may, however, arise from contraction of the mitral orifice, or from that general obstruction which the left heart has to encounter in the distal part of the systemic circulation.

Taking it for granted, then, that the circulation through the left side of the heart is obstructed, it is easy to explain all the other symptoms. The obstruction through the left side of the heart throws the blood back on the left auricle and thence on the lungs, giving rise to the state of extreme pulmonary congestion in which we find our patient, and the consequent hæmoptysis. The obstruction at the lungs is propagated backwards to the right ventricle, which, in its efforts to overcome the opposing obstacle, becomes dilated and hypertrophied; the right auricle encounters corresponding difficulties to those which oppose the action of the ventricle, and it becomes similarly dilated and hypertrophied; and in consequence of the impediment which it has to encounter, the blood is

the inner segment of the mitral valve, causing roughness of its surface, will generate a bellows-murmur, which closely resembles that caused by mitral regurgitation. Many of the long-existing mitral murmurs, which date from an attack of rheumatic endocarditis in early life, are of this kind.

thrown back upon, and delayed in, the venous system, and regurgitant venous pulsation and dropsy are the result. Thus we see all the symptoms fall in a retrograde succession, as the consequences of the obstructive force beginning at the left side of the heart, or in some part of the systemic circulation.

A feature of this case which practically is of most interest to us is the *dropsy*, because this is the symptom which generally and justly excites the greatest alarm in the minds of patients and their friends. I have already mentioned that the characteristic of cardiac dropsy is this, that it always takes place first in the most dependent parts: it does not begin, as we have seen other forms of dropsy, in the face and upper extremities. An aneurism may cause dropsy of the upper parts of the body from pressure on the descending cava or some of its large tributary veins, but pure cardiac dropsy never comes on thus: the very fact of its not existing in the upper parts of the body suggests its cardiac nature.* We find it apt to vary in amount according to position; the erect or sitting posture favours its accumulation, and hence it is that it increases in quantity towards the end of the day, and disappears or diminishes in the morning from the recumbent position during the night. Still, it will appear in the upper extremities, but always late, and often more on one side than the other, being most abundant on that side which is the most dependent. This we have seen in our patient: his left hand was the first affected; but lately we find he has been lying very much on his right side, and the consequence is that the dropsy has quitted his left hand, and accumulated in his right. We could not have a better proof than this of the purely mechanical character of the effusion.

Now what is the immediate cause of this dropsy? We have seen that the impediment to the circulation is propagated backwards from the left to the right side of the heart, and by its influence in the latter situation a considerable portion of the blood returning from the general circulation is pumped back into the veins at every systole: this obstructs the flow of venous blood throughout the body: the circulation is consequently impeded in the capillaries: these vessels become loaded, and

* See Lecture XXI.

the only relief they can obtain is by the filtration of the serum of the blood, or the liquor sanguinis, into the areolar tissue. If any part is dependent, a greater force is needed to send the blood through its capillaries, because the venous blood has to ascend against gravity: a greater accumulation is apt to take place both in the capillaries and in the veins; and, therefore, such part becomes affected with a greater amount of dropsy.

The access of dropsy is always a serious symptom, but not necessarily a fatal one. It may come on early or late. In some cases of purely cardiac disease in young persons, not complicated with either kidney or liver disease, dropsy may come on early, from which the patient may recover completely, and life may be prolonged for some time, even although the cardiac disease continue. If a patient has had rheumatic endocarditis, and such impairment of the mitral valve as gives rise to regurgitation, the effect would be to obstruct the circulation first at the left, and then at the right side of the heart. Now the right cavities feel the obstruction at first much more than they would do afterwards: the left auricle, being slow to dilate, has not yet adapted itself to the deranged condition, and the whole force of the obstruction is thrown back on the lungs and on the right side, the cavities of which dilate easily, and thus dropsy becomes rapidly developed. In the meantime the left auricle dilates, the circulation through the lungs becomes less obstructed: there is, therefore, less obstruction in the right side of the heart, which gradually recovering itself, the dropsy diminishes.

In all cases, what seems to be necessary to the production of the dropsy is the *dilatation of the right side of the heart*. I think my friend Dr. Blakiston, of St. Leonards-on-Sea, was the first to point out clearly the importance of this condition. Dr. Blakiston seems to lay it down as a rule that venous regurgitation always precedes the dropsy. But dilatation may exist without regurgitation being manifest in the external veins: at least, I think that the regurgitation need not exist to such a degree as to produce pulsation. I am now attending a case in private practice in which there is dropsy and undoubted dilatation of the right side of the heart, but no venous pulsation whatever; and I constantly witness many similar.

But there are certain other circumstances in this case, in

addition to the condition of the heart, that predispose to dropsy: these are, a state of the blood in which the water is in undue proportion, and the solid ingredients diminished, and *renal* disease. When the blood is in such a condition as to favour dropsy, the other causes will act much more speedily than when such is not the case; and, no doubt, in the present instance, the dropsy came on much earlier in consequence of the impoverished state of the blood occasioned by the repeated attacks of hæmoptysis which our patient had suffered.

Reviewing, then, the whole case, we may sum up thus: that the primary cardiac affection consisted in a rheumatic endocarditis, which narrowed the aperture of communication between the auricle and ventricle, and thus obstructed the passage of the blood into the ventricle, throwing it back upon the lungs, causing pulmonary hæmorrhage and dilatation and hypertrophy of the right cavities; or, in a rheumatic pericarditis, causing an extensive adhesion between the heart and the pericardium, and so far impeding the heart's play as to give rise to hypertrophy of its walls, with dilatation of its cavities. Or that, from some obstruction to the circulation, either in the large arteries or in some more distant part of the circulation (in the kidneys, for instance, from chronic disease in those organs), the left heart has suffered dilatation and some hypertrophy; and that, in course of time, the right heart has, in consequence of the obstructed circulation in the left side, become hypertrophied and dilated likewise. On the former supposition, the primary disease would be in the heart itself; on the latter it would be in the kidneys, or some other part of the systemic circulation.

I have already alluded to the amaurosis with which our patient is afflicted. This affection most probably results chiefly from an anæmic state of the retina. Last summer we had in the hospital a remarkable case, which illustrated how loss of blood may produce amaurosis. The patient had suffered from violent hæmatemesis: after the last attack he completely lost his sight, and he remained quite blind afterwards, notwithstanding a variety of treatment to which he was subjected. In a case of this kind the nutrition of the retina is permanently damaged by the altered quality or quantity of the blood after such severe hæmorrhage, and it bears an analogy to those cases in which

delirium or epilepsy follows excessive loss of blood, or occurs in extreme states of anæmia.

One thing I have omitted to mention, and that is the pericarditis. At the time of our patient's admission there was a very distinct rubbing sound over the region of the heart, which the next day had disappeared, and with its disappearance there supervened aggravated dyspnœa, an undulating movement in the region of the scrobiculus cordis, synchronous with the action of the heart, increased extent of præcordial dulness, and feebleness of the cardiac sounds. We set down all these signs to the development of a large quantity of fluid in the pericardium; and, with the idea of getting rid of it, I applied a blister over the region of the heart, preceding it by a mustard poultice, and purged him well, supporting him at the same time with chloric ether. The good effect of this treatment was manifested by the disappearance of the symptoms just named, and the return of the rubbing sound. Now what was the cause of the pericarditis? This is very doubtful; but this we know, that renal diseases are very apt, through the contamination of the blood by the uneliminated urinary principles, to induce serous inflammations, and that this man was, at the time of the attack of pericarditis, labouring under such renal disease.

Treatment.—In the treatment of cases of this description, your attention should be principally directed to the relief of the most urgent symptoms, and to strengthen the power of the heart. For the relief of the dropsy, a great deal may be done by attention to the position of the patient, and by the administration of diuretics. With the view of supporting his powers, we gave our patient Baylis tonics and iron; and finding the kidneys acting imperfectly, we gave him digitalis. But in such cases it is desirable to be careful in administering this medicine, and it is a good plan to combine it with some tonic. I frequently combine it with ammonia, or give the tincture of digitalis with the tincture of the sesquichloride of iron. I cannot, however, say that any great benefit has resulted in the present case, either from the digitalis or the iron: that treatment was cut short by the supervention of the pericarditis. This, however, was subdued by very simple means—counterirritation and copious discharge by the blister.

There was much difficulty in getting the kidneys to act, and the greatest benefit was derived from the bitartrate of potass, either alone or in combination with the pulvis jalapæ or the compound powder of jalap. We also gave him lemon-juice, and found it acted freely on the kidneys for some time. The dose of lemon-juice was from half an ounce to an ounce several times a day.

Elaterium was also exhibited, but without any decided benefit. The dropsy has been kept down best by means of the acupuncture: the quantity of water that has oozed away from him, and the relief that he has obtained from it, is surprising. In practising acupuncture in dropsical cases it is important that you should make the punctures at a considerable distance from each other, for each puncture is apt to become the centre of an erysipelatous inflammation, which spreads the more readily the nearer they are to each other. It must be confessed that, with whatever care you make the punctures, the difficulty which you have to encounter arises from this erysipelatous inflammation, which sooner or later almost invariably follows.

There is another method of relieving anasarcous legs, lately revived on the Continent. Instead of pricking the legs at various points with the needle, a single incision is made above the inner malleolus, or in some other convenient position (not too near a vein or artery), cutting through the skin and subcutaneous tissue down to the fascia. The length of the incision may be from half an inch to an inch. The rapid flow of water which these incisions admit of is encouraged by placing the patient in a sitting posture on the side of the bed, for a certain time during the day, with his feet placed in a tub. By this plan, the patient gains the treble advantage of increased rapidity of the escape of the fluid, the prevention of that sloppy and soaking condition which inevitably ensues when the acupunctures are allowed to ooze all day, and the ease to the breathing which results from the erect position. There is less danger of sloughing by this process, as a single incision is enough for each leg. If this man does not materially improve soon, we shall adopt this expedient, and you will have an opportunity of seeing its effects.

Unhappily we can do little to restore the impaired action of the heart, but we may often do much to ameliorate the con-

dition which that impairment entails. The principal indications are to support the patient's strength, to keep open the emunctories, and to adopt all the means in our power to get rid of the superfluous water.

LECTURE XXVI.

ON CARDIAC DROPSY, AND ON ASCITES.

ASCITES.

Gentlemen,—Before entering upon the consideration of the proper subject of this lecture, namely, that form of dropsy called Ascites, I must ask your attention to the issue of the case of cardiac dropsy which we discussed in the last lecture. Our patient, Baylis, did not live long after we last met. The dropsy, despite of the various remedies employed, increased, the difficulty of breathing also became greater, fluid was effused into the pleuræ as well as into the abdominal cavity, and at length he died exhausted.

This is the fate of all patients suffering from mechanical dropsy when the cause is irremediable. So long as the cardiac difficulty remains, the mechanical cause of the dropsy continues to exist, and you have no means of obviating the accumulation of water save by establishing drains at various points, to carry off the accumulating fluid. Hence you begin by trying to excite the kidneys and the bowels, great vascular surfaces whence large quantities of water may be carried off. The continued application of the various stimuli which the materia medica afford renders these surfaces at length insensible to their further influence; they refuse to obey the stimulus, and cease to yield a sufficient quantity of fluid. It is then that you must have recourse to more direct means of evacuating the fluid from the areolar tissue in which it has collected. You will remember that in this case, having tried the acupuncture without any great benefit, we proposed to adopt the method of a

single incision of about an inch in length in each leg.* The result of this treatment was as follows:—Immediately after the operation, the patient was made to sit up with his feet in a tub into which the fluid drained. In a couple of hours, twenty-eight ounces of serum were discharged in this way. Two days afterwards a pint of serum was obtained, and for eleven days the legs continued to discharge at the rate of about eight ounces each day. The edges of the incision then became covered with lymph and sloughy, and this diminished and ultimately stopped the discharge. The dropsy, which under the first escape of fluid had decreased, now quickly increased, and the patient became rapidly weaker, and died on the 3d of February, nearly three months after his admission into the hospital, and more than four months from the supervention of the dropsy. The practice was so far successful that it evacuated a good deal of fluid, and seemed to prolong life for some days; but it was tried rather too late, and under unfavorable circumstances, caused by the inflammation which had already been excited in the limbs by acupuncture. The case was, therefore, an unfavorable one for trying this mode of proceeding.

* This method was originally suggested by the celebrated Mead, as I am kindly reminded by my friend, Dr. Gull. The following passage, in which Mead describes his mode of procedure, deserves well to be quoted. "In the leucophlegmatia, an incision ought to be made in the inside of the leg, two fingers' breadth above the ankle, as far in as the cellular membrane and no farther; in order to serve as a drain for the water, which should run for some days. And during this time let the leg be fomented with a decoction of emollient and warm herbs, with an addition of camphorated spirits of wine; which method I have often found to be of great service, not only in this species of dropsy, but even in the ascites itself; nay, in some cases it has proved an absolute cure by drawing off an incredible quantity of water for many days together. But care must be taken, not only in this particular incision, but in all others that are made in any part of the body for drawing off the waters, not to overexhaust the patient's strength; which is as much affected by this evacuation as if the same quantity of blood were drawn. Wherefore the patient is to be supported by all possible means, lest what was intended for his cure may hasten his death; whereof I have seen two instances, one of which, indeed, happened by my own fault, in not estimating the patient's strength with sufficient caution, and the other by the rashness of a surgeon. And yet it is astonishing how great a quantity of water, drawn off in this manner, hydropicks sometimes bear to lose with ease and benefit."—'*Mead's Medical Works*,' 1767, p. 387.

The post-mortem examination showed effusion into both pleural cavities, and into the peritoneum. The cavity of the pericardium was entirely obliterated; in greatest part by means of *old* adhesions, the result of old pericarditis; but by *recent* adhesions, also, over a portion of the anterior surface of the heart, towards its left side, which you will remember was the seat of the rubbing sound, heard for some time after his admission. The former attacks of pericarditis had obliterated the greatest part of the cavity of the pericardium, leaving a small portion in front, which became the seat of the recent pericarditis.

Our diagnosis was quite correct as far as regards the condition of the walls and cavities of the heart. We found dilated and hypertrophied cavities on both sides. This diagnosis was indicated both by symptoms and by physical signs, and therefore we were not likely to be mistaken. But there were no unequivocal signs of valvular disease, and consequently we could form no positive opinion as regards their condition.

The post-mortem inspection showed that the primary disturbing cause of the action of the heart was the almost total obliteration of the pericardium by adhesions due to an attack of pericarditis eleven years ago. During all that time the heart had been bound by these adhesions, instead of moving freely in its proper serous cavity. Its contractions were doubtless, therefore, laboured, and the contents of its cavities were probably not always completely expelled; and hence, in time, all those cavities became dilated, and their walls hypertrophied.

The condition of the kidneys was what we expected to find: they were contracted, as the result of chronic disease. The cortical portion was much wasted. Many of the tubes were filled with altered epithelium and granular matter; others with fat: and others were empty and shrunk.

You noticed the immense effusion of water which existed in the pleural cavities, and how this water compressed the lungs, so as to solidify a considerable portion of these organs. This effusion must have taken place within a few hours of the fatal event, as I doubt not often occurs in other cases; for we examined the chest only three days before death, and found distinct breathing audible at every part. How remarkable the obstruct-

ing cause, which could give rise to the exosmose of all this fluid in so short a time!

Let me now call your attention to some examples of another form of dropsy—namely, ascites, or dropsy of the peritoneum, which have lately been under our observation.

Case CXXII.—The first case of which I shall speak to-day is that of a man named John Murray, aged fifty-five, who had been for several years a butler in a gentleman's family. This case is especially interesting, as affording an instance in which the dropsy was on one occasion completely removed by the aid of internal remedies; and on a second, almost so; and in which, in consequence of the subsequent death of the patient from another cause, we had an opportunity of examining the condition of organs upon which the dropsy depended.

This patient was a stout, portly man, just such a person as you would expect a gentleman's butler to be. He stated that he had always enjoyed good health, and professed himself to be strictly temperate. This, no doubt, was true; but probably he kept the key of his master's cellar, and possibly may have thought it his duty now and then to taste his wines. He drank beer, and took no great amount of exercise; and thus the seeds of disease were slowly, and gradually, and imperceptibly sown, so that he did not observe anything wrong with him, until dropsy had already developed itself in his abdomen, which began to swell by reason of the accumulating fluid. He first noticed the swollen state of the abdomen three months before his admission.

On Murray's admission into the hospital, on the 28th of August, 1848, his abdomen was quite tense with fluid, and fluctuation was most distinctly felt. His legs, too, were œdematous. No exact information could be obtained as to the state of the liver, owing to the tension of the abdomen. There was, however, an irregular and feeble action of the heart, which led us to fear that the liver was not alone at fault in the production of the dropsy.

During the three months before his admission our patient complained only of what he called a fluttering in the region of the stomach, and a gradually increasing dyspnœa. The flutter-

ing was doubtless a sensation caused by irregular action of the heart.

The action of the heart consisted in a series of short systolic contractions succeeding each other at irregular intervals, and evidently of very different degrees of strength; some being felt distinctly at the pulse, others exciting no pulsation in the artery, or only a very feeble one. The sounds of the heart were feeble, but uncomplicated, save by a slight bellows-murmur accompanying the first sound, and heard at the apex.

There was not much amiss with the urine; it was rather high-coloured, but did not deposit any sediment, nor did it contain albumen.

Having tried for a few days the bitartrate of potass without advantage, I determined to bring the patient under the influence of mercury, and to combine with it digitalis in small quantities, carefully watching the state of the heart, in consequence of the weakness of that organ. He was accordingly ordered one grain of powdered digitalis with one grain of squill, and two grains of blue pill thrice daily; and a few days afterwards ten grains of the strong mercurial ointment, and the same quantity of the compound iodine ointment, were directed to be rubbed in over the region of the liver twice a day.

In the course of three weeks there was a marked diminution in the quantity of fluid in the abdomen; the kidneys were excited to very free action, so that they secreted not less than from four to five pints of urine per diem. As the mouth had become sore in about a fortnight, the pill was ordered to be taken only once a day, and the frictions were continued. With these remedies was combined pressure on the abdomen by means of a bandage.

Under the influence of these remedies the patient rapidly lost all his dropsy, and went out quite cured *as regards it*, on the 19th of October, that is, in about seven weeks; the feeble and irregular action of the heart continuing much the same. The removal of the fluid enabled us to feel the liver enlarged and indurated, but without any irregularities upon its surface.

Unfortunately, soon after he left the hospital Murray was exposed to cold, and suffered an attack of bronchial catarrh; the dropsy returned, and he was re-admitted on the 11th of

November, only three weeks after his discharge. Upon his re-admission we had recourse to the same treatment as that previously adopted, with the same beneficial results. We again applied the iodine, mercury, and pressure; and in addition to this we gave him taraxacum, with manifest advantage. All his symptoms had improved, and the ascites had almost disappeared, when he was exposed to the contagion of erysipelas, at that time raging with great virulence in the ward. He vomited several times and shivered, and had sore-throat, which was followed by an extensive bronchitis affecting the small tubes: the respiratory mucous membrane having been, I have no doubt, the seat of an erysipelatous inflammation. Under the cough, difficulty of breathing, and excessive secretion produced by this affection, he rapidly fell into a very depressed state, and died suddenly in the night.

On examining the body, we found that the liver was considerably enlarged, and Glisson's capsule was much thickened, both around and in the substance of the organ. There was great dilatation of all the cavities of the heart, especially the right; and a good deal of deposit of fat upon and among the fibres of the heart, which seemed weak and ill-nourished, giving this organ a soft, flabby appearance. It is very possible, also, that there may have been fatty degeneration of some of the fibres; but as the heart was not examined microscopically, we cannot speak positively upon this point. The orifices of the ventricles were both dilated, and the mitral valve was shrunk and thickened at its margin, so that there can be no doubt regurgitation took place at the mitral orifice during life: this explains the bellows-sound.

In this account of symptoms and morbid appearances you have the history of a large number of cases. Either the morbid state of heart, or the chronic disease of the liver, may take precedence; or the two diseases may come on simultaneously. It is probable in this case that the liver was the first organ to go wrong; and this is indicated by the nature of the dropsy, which was mainly confined to the belly, the œdema of the lower extremities being the only dropsy referable to the heart; but this may ensue upon an ascites of long standing, even when the heart is not diseased. The heart had not yet acquired that

degree of dilatation necessary to the production of extensive cardiac dropsy. The condition of this man, then, was as follows: he had a weak fatty heart, and consequently a feeble circulation; chronic disease of the liver was established, and the course of a large portion of the blood of the body, that, namely, of the intestinal canal and the spleen, became seriously impeded. This embarrassed the heart, which, in consequence of the obstructed circulation at one point, experienced a gradual dilatation. The obstruction to the circulation in the liver determined, so to speak, the dropsy to the peritoneal cavity, and the increasing dilatation and weakness of the heart tended to render it general. In such cases, if the disease of the liver has not yet gone to the production of contraction of Glisson's capsule, diet, the antiphlogistic treatment, &c., may relieve the congestion of the liver, and diminish the obstruction to the circulation, and rest may enable the heart to recover itself somewhat; the state of dropsy may be removed, and the patient may, as in Murray's case, temporarily recover; but the occurrence of any new cause of disturbance of the circulation, as for example, the bronchial catarrh in his case, may renew the embarrassment of the heart, the dropsy may again be determined to the abdomen by the obstructed hepatic circulation, and all the old symptoms may return, to be again relieved by a similar plan of treatment to that at first adopted.

Case CXXIII.—The second case to which I shall call your attention is that of Elizabeth Whiteman; this, like the other, terminated fatally, and we can therefore speak pretty decidedly as to the exact nature of the diseased conditions. The patient was forty-five years of age, and had had eleven children. In this case there was much more evidence of the existence of intemperate habits than in that of the butler, to which I have just referred. As so frequently happens, however, she declared herself to be a very temperate person, but admitted that she was in the habit of taking a pint and a half of beer a day, and one or two glasses of gin. About twelve years ago, being then thirty-three years of age, she threw up a large quantity of florid blood. It is difficult to determine, at this distance of time, whether the blood was vomited or coughed up—whether it was a case of

hæmatemesis or hæmoptysis. The florid colour favours the latter supposition; but although in hæmatemesis the blood is generally black, it is not so always, for it may be thrown up at once, and then it may be as florid as if it came from the lungs. The colour of the blood is usually dark in hæmatemesis, because when hæmorrhage takes place into the stomach, the blood is apt to accumulate in that viscus for some time before it is cast up, and then it becomes mixed with and blackened by the secretion of the stomach. In the lungs, on the contrary, it is expelled immediately after its escape from the blood-vessels; or, if it remain in the lung, it is exposed to currents of air. Six years afterwards, this patient appears to have *vomited* a quantity of *black* blood; and the attack of vomiting was preceded by pain in the scrobiculus cordis, and followed by severe retching. It is evident that on this occasion the functions of the stomach were much impaired, either primarily, or most probably as a consequence of disease of the liver. Five years before her admission she had a severe fit of rheumatic gout, affecting nearly all her joints; and two years afterwards she had another similar seizure. About a month before her admission into the hospital she began to suffer from attacks of retching, with excessive flatulence and spasms of the stomach,—symptoms evidently denoting great derangement of the stomach, and due probably to irregularities of diet and intemperate habits. At this time she also began to pass urine of dark colour, diminished in quantity, and depositing an abundant brick-dust sediment. Her abdomen began to enlarge at its lower part, and she suffered pain in the loins. Shortly afterwards her legs showed signs of dropsy.

On her admission we found very distinct evidence of ascites in the enlarged and fluctuating abdomen. Percussion elicited a clear tympanitic sound over the region of the stomach, indicating that this organ was much distended by flatus. The patient was greatly emaciated, her skin was sallow, and there was a distinct yellow hue of the conjunctivæ. She also exhibited another symptom (and it is one of great interest, and not uncommon, but which was not so clearly marked in the last case I narrated to you), in a great enlargement of the veins coursing over the abdomen—the superficial epigastric veins. You know there is a communication at one or two points between the veins contri-

buting to form the portal vein and the systemic veins; and when the circulation through the liver becomes retarded, some of the blood of the portal system is thrown on the vena cava inferior, and through it on the superficial veins of the abdomen, which, naturally small and scarcely visible, become meandering blue lines, or even prominent vessels of considerable magnitude.

In this case, the patient being a woman, we had to make the diagnosis between ascites and ovarian dropsy,—a diagnosis which is, generally speaking, not very difficult, and for which the simple process of percussion is ordinarily sufficient. In ascites, the percussion-sound is clear in the centre of the abdomen, and dull in the flanks; in ovarian dropsy the dulness exists over the whole extent of the tumour. But in some cases of ascites the length of the mesentery is not sufficient to allow the intestine to float to the top of the accumulated fluid, and then you will have a universal dulness similar to that in ovarian dropsy. In such a case you must resort to more indirect and collateral evidence for supplying the data of your diagnosis;—the history of the case—the mode in which the dropsy commenced; if it began on one side, and gradually extended—the presence or absence of evidence of hepatic disease, or of some other abdominal lesion calculated to impede the portal circulation.

It is not very likely that the condition of pregnancy would be mistaken for ascites, or *vice versâ*. But we know that women labouring under ovarian dropsy have been accused of being pregnant, and their characters damaged; and, on the other hand, I have heard of a pregnant woman being pronounced the subject of ovarian dropsy, and prepared to be tapped.

A thin-walled uterus with a superabundant quantity of liquor amnii may give the signs of an unilocular ovarian cyst. The same signs which would distinguish such a cyst from ascites ought to prevent your mistaking such a gravid uterus for that form of dropsy. And, in examining abdominal enlargements, you must always keep in view the signs and symptoms of pregnancy. Now and then young women, trying to evade the detection of their friends, seek and obtain admission into hospitals under the pretext of suffering from abdominal dropsy, or enlargement from some other cause.

The symptoms under which our patient Whiteman laboured did not appear in any degree to yield to the treatment which was pursued. She was mercurialized, and various diuretic remedies were administered; but the urine became less and less in quantity, she was frequently sick, and she became much weaker, and sunk gradually, in a fortnight after her admission, apparently from exhaustion and insufficient nutrition.

On the examination of the body we found the peritoneal cavity filled with a yellow serous fluid. The liver was *contracted*, and slightly tuberculated on its surface. The capsule of Glisson was so much thickened as to give a peculiar distinctness to the lobulation of the liver. When you consider how intimately connected the portal veins are with the capsule of Glisson, how that structure accompanies and envelopes them throughout the greatest part of their course, you may easily understand how its thickening and contraction must interfere with the free passage of blood through these vessels. Many of the cells of the liver were loaded with fat, but others were quite devoid of it, as if starved. The heart was small: the kidneys were healthy, but their blood-vessels much congested.

Comparing this second case with the first, we find ascites existing in both, but in the first connected with *enlargement* of the liver; in the second, with *contraction* of that organ. In both, Glisson's capsule was altered and thickened, and thus there existed in both the condition most necessary for the retardation of the portal circulation, on which the ascites depends. You may have enlarged liver without ascites, as in the simple fatty liver, or in the waxy liver, or in some cancerous livers, or in those which seem to owe their enlargement to dilatation of the ducts. I suspect that the condition which determines the existence of ascites is thickening of Glisson's capsule, and that if this structure be thickened and altered, then ascites is produced whether the liver be enlarged or contracted. And I think it may be laid down that ascites is much more untractable when the liver is contracted than when it is enlarged. Some, indeed, suppose that the contracted liver is an advanced stage of the same disease which creates enlargement of that organ; but the evidence in favour of this view appears to me to rest on very slender grounds. I know of no

satisfactory evidence to prove that, in any given case, the liver has passed in succession through the stages of enlargement and contraction; although, beyond doubt, a very large liver admits of reduction, yet not in the manner and to the degree commonly described. A similar question arises as regards Bright's kidney, —Is the enlarged mottled kidney an early stage of the contracted kidney? You are doubtless aware that there are the strongest reasons for answering this question in the negative, and that there can be now no doubt that these two different states of kidney are essentially different diseases, both producing a common effect, namely, albuminous urine. So, also, it is highly probable that the enlarged liver, with thickened Glisson's capsule, is a different disease from the contracted liver, although both produce ascites: and not only so, but of the four cases of enlarged liver, all giving rise to very similar phenomena, each may exhibit a different phase of morbid change.

Case CXXIV.—The third case of which I shall speak to-day —that of Sarah Gadd, aged sixty-six—differs from those I have just related, in the absence of that condition which in them was the most significant and important, and, doubtless, the cause of the dropsy, namely, the thickening of the capsule of Glisson. On her admission, there was no doubt as to the existence of ascites; but the previous symptoms were by no means urgent, and the dropsy had come on without any of its usual premonitory signs. There was no sallowness; the liver could not be felt, nor was there any symptom to give information as to its condition. There was, however, the negative evidence of the character of the urine, which contained no undue quantity of urates; and this was so far favorable, as it denoted the probable absence of any severe organic hepatic disease.

This patient was under treatment for a long time. We gave her diuretics, especially the bitartrate of potass; and also applied the iodine and mercurial ointment, and pressure. Under this treatment the urine increased in quantity, and the size of the belly diminished, but not so much so as to bring to light any enlargement of the liver, or the existence of any other tumour. In spite of this alleviation of the most prominent symptoms, he woman became gradually weaker and weaker (still passing a

fair quantity of urine), till the night of the 9th of February, when she died suddenly. The only particular symptom that could account for her sudden sinking was a cough which she had for a day or two before her death; but this seemed quite inadequate to such a result; at any rate, the treatment could not have pulled her down, as we were giving her citrate of iron and other tonics at the time.

You will remember that I had already expressed the opinion that no hepatic disease existed; and that I stated my suspicion that some deep-seated tumour, possibly of a cancerous nature, would be found external to the liver, compressing the portal vein or some of its principal tributaries, and thus impeding the circulation through the intestinal veins, and causing peritoneal dropsy. When I heard of her sudden death, the possibility of her having some internal cancerous disease was much more strongly impressed upon me; and when we came to examine the body, we found it to exist in much greater quantity than I had at all anticipated.

The diagnosis in this case was extremely obscure and difficult, for we had no definite symptom to guide us except the dropsy. This, by its situation, denoted an impeded abdominal circulation either within or external to the liver; but we had no sign of hepatic disease. There was sufficient evidence to show that the woman's habits were not intemperate; the kidneys performed their office sufficiently, and the urine was on the whole normal, although latterly it had from time to time precipitated crystals of uric acid in the form of grains. There was no evidence of disease of any other organ, whether abdominal or otherwise. Thus, then, we had a good deal of negative evidence; all the positive evidence was the dropsy, and its persistent resistance to all remedies, denoting a persisting cause. The deposition of uric acid somewhat favoured the suspicion of malignant disease, but afforded no positive information.

On opening the abdomen a large quantity of a transparent somewhat viscid fluid escaped. The great omentum appeared shrivelled up, and converted into a solid cancerous mass, which adhered closely to the intestines and to the peritoneum, on the posterior wall of the abdomen. This tumour must have pressed upon the mesenteric veins, and probably also upon the trunk of

the portal vein itself, so as to cause engorgement of all that part of the venous circulation of the viscera which was on the distal side of the pressure, and thus mechanically to give rise to the effusion. There was a cyst, about the size of a small orange, attached to each ovary; and the interval between each of these cysts and the other pelvic viscera was filled by cancerous growths, so that all these organs were capable of being removed in a mass. The cancer was doubtless peritoneal, and probably originated in that part of the peritoneum which is connected with the ovaries. The liver itself was not diseased; the capsule was a little thickened, but only superficially, and not that portion of it which penetrates into the substance of the organ.

Now here was a very interesting cause of the dropsy, namely, the mechanical impediment to the return of blood from the abdomen to the liver, while the circulation of that organ itself was not at all impeded. The difficulty is to distinguish between this particular cause and the other to which I have before referred, namely, *intrinsic* obstruction of the portal circulation by disease of the liver itself; that, in the present case, I would call, for the sake of distinction, *extrinsic*. I know of no unequivocal sign of this condition but that of feeling the tumour. The cachectic aspect of the patient, and a decided or rapid emaciation, would doubtless excite suspicion; the deposition of uric acid, too, in a diathesis not gouty, would also increase my fears of malignant disease; but from these points we could merely guess, and, short of manual examination, I know of no sure foundation on which to build a diagnosis. Of course, the evidence of the absence of disease in other organs is valuable; and we should be greatly aided if we could detect cancer in an abdominal or pelvic viscus; and our suspicions would be confirmed if the history of the patient showed that cancer had existed in former members of the family.

The sudden death of this patient, as I said, excited my suspicions of cancer: and the reason it did so was, that I have known several cases of cancer end in a similar way; the patient suddenly sinks when perhaps you least expect it, and without anything manifest to account for it. This woman, indeed, had been suffering for some days from difficult breathing, caused by

the accumulation of fluid in the abdominal cavity; and this, no doubt, hastened her end. But we left her tolerably well the day of her death, and it was proposed to tap her; and I scarcely think that the distension was sufficient to explain the sudden change. It strikes me that, in these cases, a certain quantity of cancerous matter passing suddenly into the circulation may cause a great depression of the powers of life, and that the patient may die poisoned, just in the same manner as when the blood is contaminated by pus in cases of pyæmia. In a particular class of puerperal cases, death often occurs very suddenly in this way, the patient passing from a state of comparative health to death in the course of a very few hours; the source of the purulent infection being the surface of the uterus and the uterine veins.

That you may not conclude that ascites, although very serious, is always fatal, let me add one more case, in which the dropsy did not kill, but yielded under the treatment adopted.

Case CXXV.—William Jenkinson, aged thirty-four (vol. xxvii, p. 127), admitted May 12, 1849, about a month after Easter. He was the "hammer-man" of a smith's forge, and worked hard; he confessed that he drank hard also, rum (a highly alcoholic liquor) entering largely into his potations. Nevertheless he had been generally a healthy man, and had never been kept from work except by drunkenness. Eighteen months ago, however, he had chest symptoms somewhat resembling phthisis, and was on two occasions an out-patient at this hospital for them, and recovered to a great extent, a cough, however, remaining. On Good Friday he ate largely of hot cross buns and drank ale freely, and afterwards suffered very much from pain in his abdomen, which swelled considerably; the urine for two days was almost suppressed. This secretion became soon re-established without any medical treatment, and the swelling subsided. Some days afterwards he found the abdomen again much swollen and very painful, and on this occasion he came into the hospital.

On his admission the abdomen was found swollen and tense, and there was a distinct sense of fluctuation at every point; it

measured thirty-three inches round at the level of the umbilicus; it was likewise tympanitic; much pain was complained of in the loins and on the abdomen, which was very tender to the touch. The chest-sounds and those of the heart were natural. Pulse hard and quick, 100. Bowels much confined. There was no tenderness in the region of the liver, nor were the abdominal veins enlarged. Urine, about a pint and a half in the twenty-four hours, high-coloured, specific gravity 1025, acid, no sediment, no albumen.

In the absence of any evidence of diseased liver in this case, and with the existence of so much abdominal pain, I referred the dropsy to subacute peritonitis, and determined to treat the patient accordingly. Opium and mercury, in the form of blue pill, were at once given. I deferred interfering with the bowels for a day or two; on the third day they were opened freely by enema. Half a drachm of mercurial ointment was rubbed in over the region of the liver every night. Under this treatment the kidneys soon began to act much more freely, and their increased secretion was favoured by small doses of the bitartrate of potass. In a week (on the 24th May) the mouth became sore; still he continued to take two grains of blue pill every night, but the rubbing was discontinued. On the 26th the abdomen was softer and smaller, the mouth being still sore. On June 2d all fluctuation had vanished, and the swelling of the abdomen had disappeared.

The urine being scanty, and containing uric acid in large quantity, he was ordered to take two drachms of phosphate of soda every morning. Under this treatment the urine increased, and the quantity of uric acid diminished. He improved steadily, and left the hospital the last day in June.

I must briefly relate one more case, in which the operation of tapping was very successful.

Case CXXVI.—Mary Smith, aged forty, admitted June 21, 1852 (vol. xxxviii, p. 9), has had four miscarriages at early periods. Her last confinement was five months before her admission, when she gave birth for the first time to a living child. Six weeks after her confinement dropsy showed itself in her face, legs, and belly; the urine became scanty, and she

suffered from frequent micturition at night. On admission she had a sallow complexion, great enlargement of the abdomen, and dropsy of the lower limbs. The heart's action was faint and feeble, and there was an increased extent of dulness over the præcordia. There was distinct fluctuation in the belly, which measured forty-nine inches round. The urine was smoky, and coagulated freely with heat and nitric acid. There was no jaundice; the stools were natural in colour, and the urine free from bile. The ascites in this case seemed connected with the state of the kidneys, of which it was difficult to determine the precise condition, as we had no means of deciding how long the albumen had been present. It was in all probability an acute state supervening upon a chronic one of some duration; some epithelial and granular casts were found, and at the same time there was some impediment to the circulation through the liver. Acting on this view, diuretics and drastic purgatives were administered; calomel, squill, and digitalis were cautiously given, and also the bitartrate of potash, with occasional doses of the compound powder of jalap.

After a fortnight of this treatment, the only change observable was a slight diminution in the size of the abdomen, which was also a little softer. The urine was increased in quantity to rather more than two pints and a half, specific gravity 1007.

On the 10th July, flannels soaked in a strong infusion of digitalis were applied to the abdomen. The urine, which had reached four pints on the previous day, continued to flow at this rate, but no material reduction took place in the dropsy.

On the 15th the digitalis fomentations were left off, and elaterium was given in doses of a quarter of a grain of the extract every alternate morning, and an ounce of the infusion of digitalis thrice daily.

No improvement took place from these remedies, and, from the occurrence of great faintness, it was found necessary to diminish the dose of the digitalis four days after it was first administered. On the 8th of August the muriated tincture of iron was prescribed, instead of the other remedies.

On the 16th no improvement had taken place; the abdomen was larger and more tense, and the quantity of urine, which

had risen to five pints in the twenty-four hours, had now fallen to three pints.

A fair trial having been given to some of the best diuretics, and the patient having begun to suffer from the distension of the abdomen, it was determined to tap her. The operation was performed by Mr. Fergusson, and thirty-six pints of a yellow, slightly alkaline fluid were drawn off. This fluid had a density of 1010, and was rendered nearly solid by heat and nitric acid. A full opiate was administered immediately after the operation.

On the 18th there was much tenderness of the abdomen, and the pulse was 104; on the 19th these symptoms had very much subsided, and she passed five pints of urine, of specific gravity 1012. On the 20th, in the forenoon, she complained of severe pain in the left lumbar and hypochondriac region; and at the usual visiting hour, two o'clock, we found her in a state of collapse, with great increase of the pain, pulse very small, 100, respirations 30, skin cold with a clammy sweat; she was ordered to have more warmth applied to the legs, a large mustard poultice to the abdomen, and half an ounce of brandy every hour, with one grain of opium and one grain of calomel in a pill every two hours. In addition to this, ammonia with citric acid in effervescence was exhibited, three grains of ammonia being allowed in excess at each dose.

This treatment had the desired influence; on the 21st she had rallied considerably, the pain was subdued, the pulse had fallen to 80, respirations 20, and four pints of urine had been passed. The effervescing medicine was continued with a little opium every sixth hour, but the calomel was given up.

From this time no unfavorable symptom showed itself, with the exception of a slight diarrhœa, which was easily subdued. The pulse fell, and all fever ceased. From four to five pints of urine were secreted daily. The abdomen showed no disposition to swell, and there was not the least indication of the accumulation of fluid in it.

On the 9th of September she seemed perfectly well; a microscopical examination of the urine detected fatty casts and some large crystals of uric acid.

This woman continued in the hospital under close observation until the 9th of October, being fifty-three days from the

tapping. During all that time no sign of any return of dropsy was apparent. She was discharged apparently quite well, but still passing a small quantity of albumen in the urine.

Here, then, are various cases of ascites which I have thought worthy of your attention, as pointing out that that particular form of dropsy—peritoneal dropsy or ascites—has its origin in obstruction to the portal circulation, although not always in the same parts of that circulation. We see, also, how it may be associated with other forms of dropsy, either when, as in the butler's case, the heart is acting imperfectly; or when the kidney is diseased; or when, from the long continuance of the peritoneal effusion, pressure on the ascending cava impedes the return of the blood from the lower extremities.

Of the causes of ascites by far the most frequent is disease of Glisson's capsule, or of the liver itself. 2d. Disease of the peritoneum is not an unfrequent cause—either chronic peritonitis, or tubercular or cancerous disease of the peritoneum; but these seldom cause extreme distension of the abdomen, unless the morbid mass presses very much on the portal vein or some of its principal tributaries. 3d. Tubercular disease of the mesenteric glands, when the morbid deposit causes such enlargement of them as to form a tumour, which compresses the mesenteric veins. 4th. Enlargement of the spleen, too, is apt to produce ascites, but seldom, I think, to any great extent; as the spleen has an extraordinary power of adapting itself to changes in its circulation, and likewise because its position does not enable it, when enlarged, to exercise great pressure on the other tributary veins of the porta. 5th. I have seen, also, an instance in which an enlarged kidney appeared to give rise to ascites. 6th. Acute peritonitis will produce ascites: this is of a different nature from those previously mentioned, and falls more under the category of acute dropsy. The peritonitis induces an undue accumulation of blood in the capillaries beneath the peritoneum, and the dropsy takes place as a consequence of this congested or sub-inflammatory condition. To these causes of ascites I would add another *probable* cause, namely, temporary enlargement of the mesenteric glands. I have seen a few well-marked cases of ascites in children which

recovered perfectly under treatment. The constitution of the patients and the symptoms, which resembled those of mesenteric disease, led me to attribute the ascites to a temporary enlargement of these glands.

Lastly, in the dropsy from acute irritation of the kidneys, whether from exposure to cold or from the scarlet-fever poison, effusion into the peritoneum or ascites may occur.

Let me now call your attention to one or two conditions which are liable to be mistaken, and, indeed, have been mistaken, for ascites.

Sometimes we find in ovarian dropsy that there is a clear tympanitic sound all over the tumour; this may be caused by the development of gas in the cyst, either through a process of decomposition of its fluid contents, or by a kind of secretion, as when the bowels generate gas, but more commonly, I think, when a communication is formed between the tumour and the bowel, and the flatus from the latter gets into the ovarian cyst. A short time ago there was an interesting example of this in the hospital. A woman, having all the signs of ovarian dropsy, was tapped three or four times: there had been dulness over the whole of the tumour, and there was no doubt about the nature of the case; suddenly the tumour became perfectly tympanitic. On opening the body after death, a communication was found to exist between the ovarian cyst and the intestine, through which flatus from the latter had escaped into the former, and had thus given rise to conditions which made the case, as regards the results of percussion, resemble exactly one of ascites. If the entrance of flatus had occurred earlier in this patient, the diagnosis would have been extremely difficult.

Ascites, with very tympanitic bowels, would present similar signs to those present in this case.

There is another source of fallacy which may be mentioned, as it was one into which John Hunter fell. Sir Everard Home mentions that that great surgeon and physiologist once tapped a *distended bladder*, under the idea that it was a distended peritoneum. We know that patients are very apt to allow their urine to accumulate, especially when they are unable either to feel acutely, or to communicate their wants, as is often the case in fever. The only symptom of the commencement of this dis-

tension may be that the patient passes a restless night: the medical attendant, perhaps, does not look for the real cause, and by-and-by the bladder becomes so distended as to fill the whole abdomen. Now you can easily understand how it would be very difficult to distinguish such a state from ascites. You should always ascertain in such cases how the urine is passed; and if this be in such a manner as to leave any doubt, you should at once pass a catheter.

There is only one other condition that occurs to me as possibly mistakeable for ascites, and that is an enormously distended stomach. To what an extent this distension may take place we had an opportunity of seeing not long ago, in a woman who died of chronic gastric disease. The stomach was so much enlarged, that it filled the whole anterior part of the abdomen, all the other hollow viscera being compressed behind and below it. Such a stomach, filled with fluid, may produce fluctuations very analogous to those of ascites. The way to put the matter beyond doubt is to excite vomiting, and cause the ejection of all the contents of the stomach: if the undulating fluid had been in the stomach, the fluctuation will immediately disappear. An interesting case of this kind occurred to me a little time ago in private practice; and I ascertained the nature of the fluctuation by visiting my patient the next morning before he had taken anything, when I found that this condition had entirely vanished. Very dilated small intestines, filled with liquid, would yield the sense of fluctuation, as I have often noticed in cases of long-continued internal obstruction. The concomitant circumstances and symptoms would sufficiently distinguish such a case from one of ascites.

Treatment.—You may gather from the details already given in the history of the cases much respecting the proper course to be pursued in their treatment. If you can clearly make out that the ascites depends on disease of the liver and of its capsule, and if the liver be not permanently contracted, then the treatment should be directed to the reduction of the chronic inflammation which affects the capsule. With this view, if nothing in the habit of the patient forbids it, the use of mercury may be resorted to, and in many instances it will prove highly useful, and a free discharge of water by the kidneys

will often take place simultaneously with the production of ptyalism.

It is also desirable to increase the action of the kidneys by other means besides. In the cases which I have detailed to you, we used as diuretics, with variable degrees of success, broom, taraxacum, cantharides, nitre, bitartrate of potass, lemon-juice, digitalis, squills; and these are among the best of a very uncertain class of remedies. To them I may add a very useful diuretic, which may be prescribed in doses of from five to fifteen grains, dissolved in water; namely, the benzoate of ammonia.

I also recommend your attention to a part of the treatment which I usually pursue with great advantage, viz., *pressure* on the abdomen by means of bandages and strapping. Two of the cases derived great benefit from it, especially the man to whom I have already alluded; he expressed himself (and the feelings of the patient are in such a case very valuable) as much relieved by it.

A question often arises as to the propriety of *tapping* in cases of ascites. My opinion is, that the operation of tapping should be delayed as long as possible, to give full opportunity for the use of internal remedies; but that it should not be postponed until the distension is extreme. When the distension is allowed to become excessive, the danger from tapping is much increased; a state of collapse and of low peritonitis are, under these circumstances, more apt to ensue; therefore, if you have tried all reasonable measures, and they have failed, do not wait for extreme distension, but tap at once. This danger may be much diminished by careful treatment before and after the operation. The first point is to take care and have the bowels well emptied by mild means a day or two before the operation, such as by a full dose of castor oil, and by enema, or by both. Then, on the night preceding the tapping, give a dose of opium; repeat this just before the operation, and also an hour or two after it, and adopt measures to keep your patient moderately under opiate influence for two or three days until the great danger of peritonitis has passed by. After the operation do not meddle with the bowels; it is not desirable that they should be moved by artificial means for some

days at least. Exciting the peristaltic action of the bowels has a tendency to bring on peritonitis, or to prevent its termination by resolution or adhesion, if it has already supervened. Hence your rule should be, not to interfere, even although some days (even seven or eight) may have elapsed without an evacuation from the bowels; and then, when it may seem important to bring about their action, this should be done by means of an enema.

Still, tapping can be regarded generally only as a palliative measure, and affords very little hope of ultimate cure: nevertheless, it must not be regarded as hopeless, for some cases get quite well under it, and the dropsy never returns. An instance of this you may have often seen in a woman who frequently comes to the hospital for other complaints. She was tapped twice, and in her case I followed the practice which I am now recommending to you, but without success. I then tapped her a third time, after which she quickly recovered; and she has remained well ever since—now a period of five years. Another case was that of a man who was tapped for ascites, apparently dependent on hepatic disease; he got quite well, and several years afterwards came into the hospital for another malady, but without any recurrence of the dropsy.

Both these cases exhibited, to a remarkable extent, a sign to which I have already alluded as not unfrequently present in peritoneal dropsy—namely, a dilated and tortuous condition of the superficial epigastric veins. This venous enlargement is probably compensatory, and serves to open up a new and enlarged channel for the blood which cannot be returned through the hepatic circulation. Hence I am disposed to regard great enlargement of these veins as a favorable sign; and I should be more inclined to tap where that enlargement existed than where the veins remained small.

Tapping may be performed with better prospect of success when the liver is enlarged than when it is contracted; and it affords least prospect of benefit when the obstructing cause to the venous circulation is extrinsic to the liver, as when it consists of a tumour or of chronic peritoneal disease. Upon the whole, the conclusion at which I have arrived is, that tapping, though a remedy not lightly to be adopted, is one not lightly to

be rejected; and although in the majority of cases it does not result in an ultimate cure, yet it is not to be regarded as affording no hope, but, indeed, often tends to prolong life.

LECTURE XXVII.

ON ASCITES.

Gentlemen,—As there is now in the hospital a case of great interest for the purpose of clinical analysis in connexion with the subject of dropsy, and especially of abdominal dropsy, and as the patient has within the last few days been subjected to the operation of tapping, of the issue of which we are as yet uncertain, I will avail myself of this opportunity of calling your attention to the principal points of her case, and making some observations on ascites.

Case CXXVII.—The patient is named Eliza Richardson, and was admitted into the hospital on January 12th, 1853 (vol. xxxviii, p. 154). The first point to be noticed respecting her is her age, which is only twenty-four years. Dropsy is not common at this early period of life. At just the age which this patient has attained, dropsy is less frequently met with than during childhood; for then it often occurs in connexion with that state of kidneys which follows or accompanies scarlet fever. At this patient's age it occurs most commonly as an effect of cardiac disease, which generally dates from an attack of rheumatic carditis on a previous and, perhaps, a remote occasion. In the earlier periods of life, dropsy consequent on disease of the liver or spleen is seldom met with; and when abdominal dropsy occurs in young subjects, independently of renal disease, it is almost always associated with, and caused by, strumous peritonitis and enlarged mesenteric glands.

In the history of our patient, Richardson, we find no evi-

dence of the previous occurrence of scarlet fever. She had followed the sedentary and irregular employment of a brush-drawer. In 1847 a hard swelling formed in her right side, which was attributed to her having carried a heavy pailful of water upstairs; under local treatment this got well in a few days. As to the precise nature of this swelling, it is, of course, impossible to form anything like an accurate opinion at this distant period of time. With this exception, she never had any particular illness, nor had she ever suffered from rheumatic fever. About five months prior to her entrance into the hospital she married, and since that time the catamenia have not appeared regularly. Three months before her admission she noticed that her ankles were swollen; this œdematous state, however, got better, until seven weeks before she came in, when the swelling increased, and extended up her legs and thighs to her belly. There was no swelling of the hands or face, except a little puffiness of the right eyelid about three weeks after this. The urine was scanty and high coloured. When brought into the hospital, her abdomen was greatly distended and very tense, more so, indeed, than I ever remember to have witnessed before; there was dulness on percussion almost universally over the belly; the lower limbs were highly anasarcous, but neither the face nor the upper extremities were swollen; there was no considerable dilatation of the superficial abdominal veins; the breasts were not enlarged, and there were no areolæ, but she had been suffering some time from slight morning sickness; the sounds of the heart were quite normal; her most distressing symptom was difficulty of breathing, which appeared to be due to the upward pressure of the fluid in the peritoneal cavity. It was evident that the swelling of the abdomen depended on the presence of fluid, as fluctuation was very distinct in all directions. The urine had a density of 1023; it was thick from urates, contained a good deal of albumen, and under the microscope exhibited a few pus-cells, but no casts of tubes at this time. I should here observe, that no certain conclusions can be drawn from the presence of a few pus-cells in the urine, particularly in that of women, as they may have their origin in even a slight leucorrheal discharge.

The case, then, appeared to be, *primâ facie*, one of dropsy of

the belly and of anasarca, which had come on in three months —the abdominal dropsy, indeed, in three weeks,—and the question which we had to decide was, "What is the nature of the abdominal dropsy?"

The patient being a female, there were two views as to the cause of the dropsy which would have at once occurred to any one; either the fluid might have been contained in a large unilocular ovarian cyst, filling the belly, or it might have occupied the sac of the peritoneum. In the majority of cases the diagnosis between ovarian and peritoneal dropsy is very simple, because the amount of fluid is, generally speaking, not great; but when the effusion is considerable, the diagnosis becomes a matter of extreme difficulty, and it then usually rests upon the *history* of the case.

Let me impress upon you the following points connected with these two forms of dropsy. If the dropsy be *ovarian*, there will very often be a history of a gradual commencement of the swelling in either the right or the left side, and thence extending upwards; more or less pain likewise will usually have been complained of in one or other side, according to the primary seat of the swelling. *Ovarian* dropsy occurs most frequently at the advanced periods of life, and is much more common in persons past thirty or thirty-five years of age than before that age.

We must not look for aid in our diagnosis from the state of the uterine functions, for many women have ovarian dropsy whose catamenia are natural; whilst in many others, perhaps indeed in the greater number, the menstrual discharge is either irregular or insufficient, although not absent. In ovarian dropsy the swelling may be diffused and uniform, and distinct fluctuation may be got at all points of the abdomen, in this respect exactly resembling ascites; and when these signs exist, the fluid will generally be found to be contained in a single cyst, which is for that reason called *unilocular*. In such a case there will be dulness on percussion over the whole belly—its centre and sides, in all positions of the body; but, unless the accumulation of fluid be excessive, *deep percussion* will elicit an intestinal sound over each loin from the presence of the colon.

If, on the other hand, the case be one of *ascites*, the best test,

when the amount of fluid is not excessive, is that derived from percussion. The percussion-sound over the lateral regions of the belly is dull, while over the centre it is clear, in consequence of the intestines being there buoyed up by the fluid, so that they come into contact with the abdominal wall. Moreover, if the patient be made to turn over upon the left side, the fluid will gravitate to this side, and, consequently, the sound elicited by percussion will be dull, perhaps as high as the umbilicus, while higher up it will be clear; and *vice versâ*, when the patient is turned upon the right side.

In ovarian dropsy it often happens that there is no satisfactory history of the commencement of the swelling, the patient not having been aware of its existence until the abdomen had attained so large a size as to attract the attention of her friends, or until she had noticed that her dresses seemed much too small for her. And in ascites, if the amount of the effusion be very considerable, the facility of diagnosis is greatly diminished. In such a case the clear sound on percussion over the umbilicus is lost. The abdomen becomes so enlarged by the vast accumulation of fluid in the peritoneal sac, that the mesentery will not allow the intestine to come into contact with the abdominal wall. Or, as Dr. Watson observed, when the intestine is tied down to the spine by old adhesions, or by peritoneal disease, a similar result follows, even although the dropsy be inconsiderable. In such cases percussion is dull everywhere over the belly, and a main source of distinction being thus lost, we must trust much to the general history; and thus it was in our patient upstairs. Some aid in diagnosis is gained from an examination *per vaginam*, which will sometimes detect a solid mass, or detect the encysted character of the dropsy, if the disease be ovarian.

Here, then, was a young woman, twenty-four years of age, with this great amount of dropsy; she had no uterine disturbance, except that her catamenia had lately disappeared, and her having been recently married seemed to offer a reasonable explanation of their stoppage. She was a little too young for ovarian dropsy; but age by no means affords an absolute rule; for many cases are recorded at the age of this person, and I shall presently call your attention to the case of a girl in this

hospital,* only fifteen years of age, with ovarian dropsy, who has been tapped twice. The enlargement of the belly in Richardson came on in three weeks. This rate of accession seems to be too rapid for ovarian dropsy, for in this affection the swelling generally manifests itself much more slowly. Then, there was other dropsy combined with that of the belly, there was considerable anasarca of the lower extremities, and this had preceded the abdominal accumulation; this also was inconsistent with the diagnosis of ovarian dropsy. Ovarian dropsy is an isolated dropsy; and, where it is combined with any other forms of dropsies, these *follow* it, as where anasarca of the lower extremities results from a large ovarian tumour, which exerts so much pressure upon the ascending vena cava that a dropsical effusion takes place into the areolar tissue of the legs and thighs. Ascites, also, occurs occasionally in association with ovarian dropsy, in consequence of the pressure which the latter exerts upon the abdominal veins, combined, perhaps, with some degree of hepatic disturbance. In consequence of the anasarcous state of the pudenda, an examination of our patient *per vaginam* was not instituted.

Upon these grounds, then, we concluded that the case was one of ascites, with anasarca.

Now ascites in most instances is due to retarded circulation through the liver; it also often depends upon chronic peritonitis, tubercular peritonitis, or the presence of a large tumour of any kind in the belly, such as a tumour of the mesentery or an enlargement of the spleen. There was no unequivocal evidence of disease of any of the abdominal organs in this case, nor were there any indications of peritonitis, although the patient had suffered a little from sickness, but no more than commonly occurs in the early months of pregnancy; and she had had no pain in the belly. There was no distinct evidence of the existence of a diseased condition of the liver, and it did not appear that our patient had been addicted to intemperate habits, or had led a debauched life. Her age was almost too early for any form of diseased liver, except the scrofulous.

She had never been jaundiced, and there was no appearance

* Case CXXVIII. Mary Jane Manuel, aged fifteen (vol. xliii, p. 252).

of bile in the urine. The superficial abdominal veins, it is true, were enlarged, and they have been getting larger ever since her admission into the hospital—a condition which is of frequent occurrence when the liver is becoming contracted, and the circulation through this organ impeded. There were urates in the urine, but not in great abundance; and there were no purpurates, as often happens in cases of chronic liver disease.

Diseases of the kidney, as you are aware, are a very fertile source of dropsy; but this dropsy is anasarca, though in the advanced stages effusions frequently take place into the serous cavities; but when, with disease of the kidney, there is also a morbid condition of the liver, even although slight, fluid will often accumulate in the peritoneal cavity to a considerable extent. Oxalate of lime, urates, and epithelial cells were observed in the urine of this patient from time to time; and although there is not very satisfactory evidence, still I am inclined to believe that there is chronic disease of the kidneys, which, however, will not explain all the phenomena; and there can be but little doubt that there is, also, some derangement of the hepatic circulation from an impediment to the flow of portal blood through the liver. The real nature of the case, then, is that the dropsy is primarily dependent on a chronic renal affection, and that it has been largely determined to the peritoneum in consequence of a greater or less amount of hepatic disease. As the case progresses we shall, no doubt, get further insight into its nature.

The treatment to which this patient was at first subjected was adopted on the supposition that the dropsy was mainly attributable to the condition of the kidney; it consisted in hot-air baths, compound jalap powder, and bitartrate of potash. The compound jalap powder purged her freely, but she could not bear the hot-air baths; and as she seemed to derive but slight benefit from their employment, and, on the contrary, increased in size, and passed only two-and-a-half pints of urine daily (twenty-four hours), I changed the treatment to the exhibition of a quarter of a grain of elaterium every morning. This operated violently, griping her very much, and producing copious watery evacuations, without increasing the quantity of urine, or materially diminishing the size of the belly, and it induced a considerable

amount of sickness. This last symptom you will find in practice to be the great drawback to the use of elaterium—namely, that it acts not merely as a *drastic cathartic,* but likewise as an *emetic.*

Finding under these circumstances that there was no indication of a diminution of the dropsy, and that medicines appeared to exercise no influence upon the kidneys, I felt it important that some decided step should be taken to relieve the tension of the lower extremities and belly; for it often happens in these cases that, when the tension is relieved, the kidneys begin to act, and that diuretics then produce their usual effects. Accordingly, I adopted the plan of making a single incision, about an inch and three quarters in length, just above the inner ankle of the left leg, and a considerable quantity of fluid drained away from this opening—more than two pints in the course of a few hours. This was done on the 26th. On the 27th the patient was much relieved, and a large amount of fluid was still draining away. On the 28th I considered it important to make a similar incision in the other leg, and she was then ordered a diuretic draft containing digitalis thrice a day. The kidneys now began to act more freely, she passed from two to three pints of urine daily, and a good deal of fluid drained away from the incisions in the legs. In two or three weeks the legs became much reduced in size, still, however, remaining much swollen. The abdomen, also, continued of very great size. Under these circumstances, tapping was the only remaining resource, and it was rendered the more imperative by the fact that her breathing was becoming difficult and distressed. The operation was performed three days ago (March 12th) by the house-surgeon, Mr. George Lawson, and in this way there was drawn off a large quantity of a serous fluid, of a citron colour, slightly opalescent, of specific gravity 1012, and containing a large amount of albumen.

Now to have recourse to tapping, or indeed to any surgical operation, is not very creditable practice; for one ought to be able to effect the carrying off of the water by inducing the kidneys to act freely without resorting to this step. Still, I believe, we may leave these cases too long before calling in surgical assistance, and thus the opportunity may be lost of obtaining that

relief to the whole system which the getting rid of the fluid almost always affords.

I should have stated that we also used in this case the benzoate of ammonia, and for a short time it appeared to answer very satisfactorily; and that we likewise tried the combination of squill, calomel, and digitalis, but without any material benefit. Finding, then, that the dropsy did not diminish, we considered ourselves justified in hoping that, after the removal of the dropsical fluid, the kidneys would become more active.

The operation of tapping is by no means always successful; not uncommonly it proves fatal by inducing peritonitis; but I cannot help thinking that it is fatal in this way oftener than it ought to be, in consequence of the neglect of certain precautions which should always be carefully attended to. The precautions to which I allude are these, that the patient should be kept well under the influence of opium from the commencement of the operation, or even for a short time before it; indeed, in any operation upon the peritoneum, this point should be kept well in view. One cause of the peritonitis which is so apt to follow this operation is the increased peristaltic action of the intestines, often excited by air entering the wound, and by the altered condition of the peritoneum. You should take care not to be too anxious for the bowels to act once in three or four days, or even longer. Your best guide as to the question of relief to the bowels will be the feelings of the patient. If he feels pretty comfortable and experiences no uneasiness from the bowels not acting, you need not interfere; and unless he suffer great inconvenience, you should still adopt the non-interference policy. It is your duty to wait until all inflammation has subsided, for five or six days or even more, before you take any steps for opening the bowels. Immediately after the operation has been performed, a grain or a grain and a half of opium should be given, and the dose should be repeated every three or four hours, according to circumstances; or the dose may be larger than this if there be reason to suppose that symptoms of inflammation are about to come on. Not unfrequently this operation is successful in prolonging life, if not in curing the disease, and it is from a knowledge of this fact that I had recourse to it; on similar grounds I would recommend you not to postpone it too long,

inasmuch as the greater the tension and overstretching of the abdomen, the more apt is reaction to ensue when relief is afforded, and the longer the delay, the weaker will be the patient's condition, and the less power will he have to resist inflammation or to withstand exhaustion.

Last year we had in this hospital a case not unlike that which we have now been considering; it afforded a good instance of the successful issue of the treatment adopted in the case of Richardson. The patient remained in the hospital for nearly two months after she had been tapped, without the slightest indication of any return of the dropsy being observed during that time. She left on the 9th of October, 1852. One of the students here told me, a few days ago, that he had recently met her in the street (five months after the tapping), and that she informed him she had never been better in her life, and had remained quite well ever since she left the hospital.* The success of this case encouraged us to hope that the operation would at least succeed in prolonging Richardson's life, and that it was quite justifiable.

It will be instructive to place in juxtaposition with this case of Richardson, one of ovarian dropsy, and I shall select, as a good example of the unilocular cyst, the case to which I have already alluded as having occurred at the unusually early age of fifteen.

Case CXXVIII.—Mary J. Manuel, aged fifteen, admitted Nov. 1854, with an enormous swelling of the abdomen (vol. xlii, p. 252). A not unhealthy looking girl, very nervous and reluctant to be examined, never had robust health, but has lived comfortably at home, and has had plenty of nourishment. There were no indications of that strumous aspect which so often accompanies abdominal enlargements in young persons.

We learned from the girl that she first noticed the swelling thirteen months before her admission; she described it as having been always uniform, not having commenced at one side more

* The details of this case will be found in the preceding Lecture, Case CXXVI, p. 505.

than the other. It made but slow progress at first, but increased so rapidly within the last two or three months, as to have doubled in size. Before the swelling came on, she suffered much from sickness and vomiting, and pain after eating. Latterly she has lost flesh a good deal, but her state of emaciation is not greater, nor scarcely so great as might be expected with so much abdominal enlargement.

Menstruation commenced in this girl two or three months before her admission, and has been repeated two or three times since, but never in great quantity. This, it will be observed, was ten months after the commencement of the swelling.

After standing or sitting for some time with the legs down, an anasarcous condition of the feet and ankles would come on, which would subside on lying down.

The urine was acid, specific gravity 1016, no albumen; bowels regular; digestive functions natural.

The following description of the abdomen was noted soon after her admission by my clinical clerk, Mr. T. P. Teale:

Her belly is now of immense size, projecting very much in the antero-posterior direction, more so than laterally, and bulging out at the lower end of the sternum. The shape is not perceptibly altered by change of position, and there is not any marked bulging at the loins when she lies on her back. The swelling is nearly symmetrical: there is some bulging just above the pubes to the left of the median line, which is due to an œdematous state of the integuments there, kept up by her lying chiefly on her left side. Fluctuation is very distinct all over the abdomen. There is an absolute dulness on percussion over the whole of the belly, except for a small space in the loins, in the course of the ascending and descending colon. There is no evidence of any increase of resonance at these parts when she lies on either side, as if intestine floated to the surface.

The *primâ facie* diagnosis, in a girl at so early an age as this patient, was peritoneal dropsy. Her aspect, and the absence of all hepatic symptoms, forbade the supposition that the dropsy could arise from obstructed portal circulation in the liver. Was it due to tubercular disease of the peritoneum or of the mesenteric glands? This view was negatived by the duration of the abdominal enlargement throughout a period of thirteen months,

in which space of time tubercular disease would have terminated fatally, or have induced such a degree of emaciation as would have rendered the nature of the case unmistakeable. The great size and the duration of the swelling, the perfect fluctuation, and the supervention of menstruation, excluded pregnancy. We had, therefore, no other conclusion to adopt than that the disease was a large unilocular ovarian cyst, and in this view we had the advantage of Dr. Arthur Farre's concurrence, who, on making an examination *per vaginam*, distinctly felt a cyst containing fluid. The downward pressure of the cyst had produced prolapsus uteri of some months' duration.

The diagnosis of this case was confirmed by the subsequent history. After a fortnight's sojourn in the hospital, during which various drugs were ineffectually tried, the abdomen acquired such a size as to impede the free action of the diaphragm. It therefore became a matter of necessity to tap. This was done by Mr. Fergusson, and fifteen quarts of a viscid clear fluid, of the colour of very pale urine, were drawn off. It was of the consistence of uncoagulated white of egg, specific gravity 1007, and slightly alkaline, and contained a large quantity of albumen. After the fluid had been all discharged, no indication of any other cyst could be discovered, and the abdomen became tympanitic, there remaining immediately around the umbilicus, to a certain degree, a less clearness of sound, as if some dense structure intervened between the bowels and the abdominal wall.

The nature of the fluid evacuated in this case points strongly to ovarian disease. In ascites the fluid is either a clear straw-coloured or whey-coloured serum, or a bloody serum, and never, so far as I know, is viscid, save in some cases of chronic peritoneal disease of a malignant kind and of comparatively rare occurrence.*

It is worth bearing in mind with reference to the diagnosis

* The subject of this case remained in the hospital from early in November, 1854, until the 19th of January, 1855. She then left, the fluid having begun to re-accumulate, but only to a slight extent. In December, 1855, she returned to the hospital, the cyst having become as full as before. She was again tapped, and left the hospital in January, 1856. In June of the same year she was re-admitted the third time, her abdomen having attained about half the size it was when she was tapped the second time.

both of ovarian dropsy and of ascites, that cysts sometimes form beneath the liver, or in some other part of the peritoneum. Careful percussion, under varied positions of the patient, ought to enable you to recognise such structures in the living body.

A week after the operation in the case of the woman, Richardson (which forms the text of the lecture), vomiting and other signs of peritonitis manifested themselves, and the patient gradually sank and died on the eleventh day after the operation, and the eighth day after the delivery of this lecture.

The condition of the body, which was examined thirty-six hours after death, was as follows:

The body generally was somewhat emaciated, the lower extremities highly anasarcous, and the abdomen enlarged. The wounds upon the legs, which had been made some time before for the purpose of relieving the tension of these parts and draining off water, looked healthy and granulating, and one was partially cicatrized. The wound in the abdominal wall had, also, a healthy appearance. The peritoneal cavity contained a large quantity of a serous fluid, with some flakes of recent lymph floating in it, and also cobweb-like pieces of apparently recently effused fibrine extended from intestine to intestine, and likewise from the viscera to the abdominal walls. The lungs were healthy. The heart was rather small; its valves were quite healthy, as were also the great blood-vessels. The spleen was large and rotten, and had some rather large hæmorrhagic patches in its substance. The uterus was unimpregnated.

The liver was somewhat enlarged, and had the appearance of an early stage of cirrhosis; Glisson's capsule was thickened, and the organ contained many of those tumours which have been described by Dr. Budd under the name of "*Encysted knotty tumours of the liver.*" The kidneys were almost twice their usual size and weight, and were much congested. One kidney, with a portion of the liver and spleen, were placed in a jar of water, and sent to Dr. Johnson for examination, on March 28th (five days after death), whose report of them is as follows:—"The kidney weighed eight ounces; its surface was smooth and red, but the lobular markings were indistinct. (The

red colour probably resulted from the kidney having become stained by the bloody fluid in which it was immersed.) The colour of the whole organ was such as might have been produced by soaking it in bloody water, and the cortical and medullary substances were nearly of the same hue. The arteries were decidedly hypertrophied, and the Malpighian capillaries thickened; in the canal of one artery a collection of oil-globules was distinctly seen. There was *no* healthy epithelium in the tubes; it seemed as if the epithelium was easily detached, so as to leave the basement membrane, which appeared somewhat thickened, and the organ afforded excellent specimens of *matrix*. There were no actually denuded tubes, not yet was there a distinct non-desquamative condition (*i. e.*, the epithelium sticking to the tubes, so as to leave a free canal in the centre). In many of the tubes there was an opaque, granular condition of the epithelium, so as to leave no cellular space.

"The liver was in an early stage of cirrhosis, just beginning to be granular; besides this, there were several firm, white, circumscribed tumours, ranging in size from that of a pea to that of a small orange, some on the surface, but most collected near the entrance of the portal vein, where a large portion of the liver was of an almost cartilaginous hardness. The liver-cells were remarkably opaque and granular; but, though opaque, of a lightish colour (not yellow), with scarcely a particle of oil in them, and the nuclei concealed in great measure by the granular contents of the cells. The tumours seen by the microscope presented a finely granular appearance, with only here and there a trace of fibres in the midst of the granular matter, probably atrophied remains of the normal tissues. There were no crystals in the tumours."

The increase of size of the kidneys in this case was no doubt in part due to their excessive congestion, and it is not improbable that this was kept up first by the great weight of water pressing on them, and afterwards by the peritoneal inflammation which followed the operation of tapping. It was, nevertheless, sufficiently plain that both liver and kidneys were the seat of an analogous morbid process, tending to some more definite form of disease, such as the waxy enlargement, or

even, after the congestion had subsided, to the chronic contraction.* That these are states of perverted nutrition, rather than of inflammation, I can entertain no doubt; at the same time, it seems to me quite possible, and, indeed, it may not improbably be even of frequent occurrence, that an inflammation may supervene upon either of these states, and may develop dropsy and other complications more quickly, and even bring on the fatal result. We must not shut our eyes to the fact that we have not yet found the correct interpretation of all the morbid changes which have been recorded in this and similar cases, and that much careful and patient observation is still needed before we fully understand the true nature of the diseased action.

The diagnosis was correct so far as it attributed the dropsy to a morbid state of both kidneys and liver; but I think it ought to have done more; it ought to have made a nearer approach to a more exact indication of the precise condition of both those organs. More frequent and more careful examination of the urine and of its deposits might have enabled us to speak more definitely as regards the kidneys, and from their condition we might have inferred that of the liver. The practical result, however, would have been the same; for had we known during life what the *post-mortem* inspection revealed to us, we should have been equally led to the same course, with perhaps less hope of immediate or ultimate success.

I have the opportunity of adding yet one more case to complete this lecture. It is a good illustration of how much may be done to prolong life by tapping in ascites (especially when the liver is large), observing the precautions as to treatment to which I have already alluded.

The case occurred in the practice of my friend Mr. Lavies, with whom I frequently visited the patient, and to whom I am indebted for the notes of it. The patient was also seen by some other physicians.

Case CXXIX.—Mr. E., aged seventy, was generally healthy, with the exception of occasional attacks of bronchitis. In

* See Lectures XIX and XX.

October, 1853, he had a slight paralytic attack which affected his face and speech only, but passed off within an hour.

In March, 1854, some œdema of the legs was observed for the first time, which remained for a week, when the bowels became relaxed and black pitchy dejections were passed; this condition of the bowels continued for four or five days, accompanied by much prostration. On examining him on the 24th, in company with Mr. Lavies, it was found that the liver was much enlarged, and felt somewhat indurated, but smooth and free from any tuberculated or knotty character of its surface.

Under mercurials and diuretics the œdematous state disappeared about the 20th of May, and he continued free from dropsical symptoms till the middle of June, when the œdema re-appeared, and now a slight fluctuation could be felt in the abdomen.

Under the prolonged use of squill and digitalis, with the tincture of the sesquichloride of iron, the œdema disappeared again in September, the abdominal dropsy remaining. The patient now left town for several months.

From January till October, 1855, the dropsical symptoms gradually increased, and the abdomen became very much distended. It was now judged advisable to perform the operation of paracentesis, as no influence could be exerted on the kidneys through diuretic remedies. The abdomen measured forty-six inches in circumference.

The operation was performed by Mr. Lavies on the 23rd of October, and eighteen pints of fluid were withdrawn. Immediately before the tapping, and for some days after it, the patient was kept under the influence of opium. He recovered quickly, and was soon able to go about.

The belly gradually refilled, and in February, 1856—four months after the first tapping—it was found to measure forty-six inches in circumference. He was tapped a second time on the 22d of February, and twenty-eight pints of fluid were withdrawn. Under a similar treatment to that adopted at the first operation he recovered very speedily.

Notwithstanding the free use of various diuretics, our patient's abdomen very quickly filled again after the second operation, so that it was found necessary to tap him a *third* time after

only two months had elapsed (April 23). On this occasion thirty-three pints of fluid were evacuated. No untoward symptom followed this operation; the same treatment was adopted as on the former occasions, and in a few days he was able to go out.

On the 30th of May, 1856, about a month after the third operation, the patient was dining out; and, shortly after dinner, he was seized with giddiness and slight paralysis of the left side, which lasted only a few hours. No remedial means were adopted, except the exhibition of small doses of the tincture of the sesquichloride of iron and the tincture of squills, and rest in the horizontal posture.

In ten days the patient was well enough to take a drive in his carriage; his general health improved, and the kidneys continued for some time to secrete three pints of urine per diem. Soon after the paralytic attack, he passed some small calculi from the bladder, which were found to consist of cystic oxide.

In August, 1856, the abdomen began to increase again, and at the same time the legs became much more œdematous than usual, although the belly did not increase in a similar ratio. Mr. Lavies thought it advisable not to defer the tapping, and operated for the *fourth* time on the 28th of October, and drew off thirteen pints of fluid.

I visited the patient about three weeks after this fourth operation, and found him quite free from dropsy, and the liver reduced in size and less indurated. His general health was good, appetite excellent, and he was able to take a fair share of both foot and carriage exercise. A quart of urine was passed daily, quite free from albumen.

In concluding this lecture, let me add a few words on the operation of paracentesis itself.

All of you who have witnessed the operation in this hospital will have observed that it is invariably practised while the patient is in the horizontal position. You place him lying on his right side at the edge of the bed, with the abdomen projecting over it as far as is consistent with safety. A sheet is placed loosely around the belly, and an assistant, who stands at the patient's back, is ready, by drawing the ends of the sheet,

to exercise a gradual compression of the abdomen, if required. But this is seldom, if ever, needed; the horizontal posture completely protects the patient from exhaustion or syncope, without the necessity of the complicated, and often embarrassing, process of compression formerly used—a proceeding, by the way, which was not without its dangers as regards peritonitis. The position of the patient is, on the whole, favorable to the escape of fluid from the abdomen, and on this account is also preferable to the sitting posture formerly adopted. My experience of this mode of operating extends to a very great number of cases both of ascites and ovarian dropsy, and I have never seen any disadvantage attending it. I have, therefore, no hesitation in advising it as the best course of proceeding; it is that which I know Mr. Fergusson follows, and it is now some fifteen years since we adopted this practice in an important case in private practice in Albemarle-street. I am informed that it is also the plan of operating which is now practised by my distinguished friend, Dr. Simpson, of Edinburgh.

LECTURE XXVIII.

ON CERTAIN URINARY DISEASES.

THE GOUTY KIDNEY.

Gentlemen,—The case on which I propose to comment today is singularly illustrative of the pathology of gout, and exhibits many points of extreme practical interest, highly deserving of the attention of those who study the practice of medicine with a proper desire to ascertain the real nature of the various morbid processes which are ever coming under observation.

A knowledge of the real nature of gout, and of its kindred malady, rheumatism, is, in my opinion, at the very foundation of

all sound pathology. I am therefore glad to have an opportunity of calling your attention to a case, in which the seeds of disease have been sown most extensively, and have taken deep root in nearly every situation. The record of the case has been faithfully and carefully kept by my clinical clerk, Mr. (now Dr.) Tanner:

Case CXXX.—Eliza Rapley, aged forty, admitted June 5, 1846 (vol. xvii, p. 91): married, and has had two children, the last being now fifteen years of age. Latterly she had led a very irregular and intemperate life, and had been a prostitute. She stated that she never enjoyed good health, and had especially suffered from repeated attacks of rheumatic gout, affecting all her joints, large and small. These were, no doubt, increased in frequency and severity by her habits of intemperance.

Three months before admission she had her last attack of rheumatic gout. Shortly after this she observed her legs beginning to swell, as well as her abdomen. The swellings having increased considerably, she sought and obtained admission into the hospital.

Her appearance was strikingly indicative of that extreme disturbance of the general health and constitution which is always produced by the long continuance of gout in the system. There was an unhealthy sallow hue, with an anxious expression of countenance. Her eyelids were swollen, as also her cheeks: the facial dropsy, although sufficiently distinct, was not excessive. Her feet and legs were also œdematous, and pitted distinctly on pressure.

There was considerable swelling of both knee-joints, caused evidently by an effusion of fluid which distended their capsules. The patient complained very much of pain in both these joints, especially the right, which was the most swollen.

Most of the small joints, particularly those of the fingers and toes, were swollen from old attacks of gout, the swelling apparently being due to a thickening of the fibrous tissue;—some of them grated on moving the surfaces upon each other, indicating the absorption of their cartilages, and the probable deposition of urate of soda in their place.

You will remember that I stated more than once, at the

patient's bedside, that I thought it very likely that a similar change—namely, absorption of the articular cartilages and deposition of urate of soda, had taken place in the knee-joints; and that the articular surfaces of the femur, tibia, and patella on each side would be found thus affected.

The abdomen was not enlarged, nor dropsical. There was no evidence of enlargement of the liver. On the contrary, everything favoured the opinion that this organ was in some degree contracted: the intemperate habits of the patient, the sallow hue of her skin, a slight dilatation of the abdominal veins, and the absence of dulness on percussion over the right hypochondriac region, served to excite suspicion as to the existence of a degree of cirrhosis of the liver, and justified our expecting abdominal dropsy if it proceeded further.

The heart was likewise evidently affected. We had proof of this in the augmented impulse of the organ, and the increase of dulness in the cardiac region: and on placing the stethoscope over the heart's apex, a loud bellows-sound was heard synchronous with the systole of the ventricle. To the right of the heart's apex, and along the aorta, this sound became less distinct, and vanished altogether as the stethoscope was passed up the aorta. It was, however, very audible beneath the angle of the left scapula. The pulse was small and weak, and its frequency 100. These signs indicated some hypertrophy and dilatation of the left ventricle, with imperfection of the mitral valves.

The urine was not materially altered in quantity, being sometimes below, sometimes above, the normal amount: its specific gravity was 1012: it was clear and pale. By heat and nitric acid it yielded a slight precipitate of albumen.

What is the state of kidney which produces this condition of urine? Some would answer, an advanced stage of Bright's disease.* Such, however, is not the case, in my opinion. I do not believe that the woman ever had Bright's disease. The history of the case does not conform to the ordinary course of things in that disease. Her aspect is quite different from that in Bright's disease; the dropsy is not so general nor so much;

* The term, Bright's disease, is here used to signify the large, mottled, and *fatty* kidney.

there has been no great frequency of micturition. On the other hand, her extreme gouty state of constitution led me to think that she had a contracted and shrivelled state of kidney, in which a large portion of the organ is wasted and its structure condensed—a condition which, while it may also occur in other states of system, is peculiarly apt to be developed in the inveterate gouty diathesis.

The first case of this kind which arrested my attention (although I did not perfectly understand its nature at that time) occurred to me five years ago. The patient had been a gentleman's butler, and had had several attacks of gout. He had a sallow, unhealthy look; and the only appearance of dropsy about him on his admission was a copious effusion into one knee-joint. His urine was sufficiently copious, quite clear and pale, and contained a small quantity of albumen. This patient died suddenly, and we found a copious effusion of fluid in one pleura, which must have taken place a few hours before death; and both kidneys were much contracted, shrunk, and granulated upon their surface, presenting all the characters of what has been described as the third stage of Bright's disease.

Not many weeks ago we had a man in Sutherland ward, whom most of you may remember, the subject of chronic gout affecting the knees and ankles and the plantar fascia. This man had the same sallow unhealthy look which this woman presented. His urine was slightly albuminous, but copious, pale, and of low specific gravity. There was no dropsy in this case. I have no doubt that the same state of kidney existed here as in the case which we are now considering. The patient got better, and left the hospital.

In reviewing the case of Rapley, it would appear that she has been the subject of chronic gout, and that her joints, large and small, have all been more or less damaged by the disease; that her heart is affected, her liver probably contracted, and her kidneys likewise. How strikingly do these consequences of the long continuance of the malady comport with the humoral view of the pathology of this disease! Not only are those parts which the morbid matter of gout is most prone to affect materially damaged, but likewise the emunctories through which the poison would make its escape out of the system—the liver and

kidneys: these organs have become poisoned by the morbid matters which have escaped, or tried to make their escape from the system through them; and therefore it is natural to expect a considerable change in their nutrition.

The treatment adopted in Eliza Rapley's case, immediately after her admission, consisted in the application of a blister to the right knee, which was most swollen, mild purgatives, and a bitter tonic (inf. quassiæ) with ammonia.

On the 19th of June it was reported that she had improved considerably; the swelling had been much reduced, the urine was natural in quantity, slightly acid, specific gravity 1010, without sediment, and it contained a small quantity of albumen.

On the 20th of June there was a sudden decrease in the quantity of urine, and on that day she was seized with a fit of epileptic character, inducing loss of consciousness and convulsions: the fit lasted some minutes, and on coming out of it she continued in a stupid, drowsy state for some time. The small quantity of urine passed was not kept for examination. A mustard poultice was applied to the back of her neck, and this was succeeded by a blister.

On the 21st, at two o'clock in the afternoon, she had another fit, more severe than the last. In it she was much convulsed, and bit her tongue severely. The fit lasted a quarter of an hour, during which time she was so unconscious that on her recovery she was not aware that she had had a fit. Her water and motions were passed involuntarily.

On the 22d it was reported that she had had two severe fits since the preceding day. She complained of occasional severe lancinating pains in the abdomen. There was some dyspnœa. Respirations 20; pulse 112. She has been very delirious. Micturition very defective. On the following day she died.

This is a common mode of termination for those diseases of the kidneys which either by encroaching on the proper structure of these organs, or by any other means, materially diminish their secreting power; and the most probable explanation of this phenomenon is furnished by the fact that, as the proper constituents of the urine are not duly eliminated, they accumulate in the blood, and disturb the brain, giving rise to epilepsy, delirium, and coma.

The *post-mortem* examination presented many points of extreme interest.

The heart was somewhat increased in size, from slight hypertrophy and dilatation of both ventricles. This morbid state of it was due to the imperfection of the mitral valve caused by deposits upon its margin, which prevented the perfect closure of the orifice. It was the regurgitation through this orifice, which remained open during systole, that occasioned the bellows-murmur heard with the first sound of the heart.

The liver was hardened and condensed in structure, and somewhat reduced in size. Its secreting lobules were not materially altered, but the capsule of Glisson, on the external surface of the gland, as well as the prolongation of it into the larger portal canals, was much denser and thicker than natural. This tissue seemed to have been the seat of a morbid process, which probably was produced partly by the intemperate habits of the patient, but partly likewise by the share which the liver had in the elimination of the morbid poison of gout.

The most interesting morbid changes, however, were found in the kidneys and in the joints.

The kidneys were very much contracted in size; they retained hardly, indeed, so much as one third of their natural dimensions. They had upon their surface a shrivelled granular appearance. The capsule appeared denser and whiter than natural, and separated with great facility from the surface of the gland. On cutting into the kidney it appeared that the decrease in its size was at the expense chiefly of the cortical substance, two thirds of which must have disappeared. The cut surface presented much the same granular appearance as the external surface of the gland.

Upon examining portions of these kidneys under the microscope, I found several tubes much dilated, and furnished very scantily with epithelium; others were completely empty; and others, again, collapsed and folded into fine plaits, which gave them the appearance of fascicles of fibrous tissue. A transverse section served to display very well the dilated tubes, showing likewise how small was the quantity of contained epithelium, and how little interlobular tissue there was likewise. Here and there a tube contained at one point, at the bend of a convolu-

tion, a few epithelial cells filled with fat; these were, however, few in number, and in many parts the tubes appeared healthy. Those in the pyramids were for the most part healthy.

These appearances are distinctly indicative of a wasting or atrophy of the gland. Many of the blood-vessels are obliterated; the portions of the gland which these supply waste; the epithelium in them is formed scantily, or not at all; the tubes collapse, and are folded into plaits, giving the appearance of newly-developed fibrous tissue.

Dr. Johnson has shown that the minute arteries undergo a peculiar change in this condition of the kidney, whether it be associated with gout or not. They become much thickened, and their fibrous coats, both the transverse and the longitudinal, acquire a great increase of size. It is in this state of kidney that we often find the cysts which have long attracted the attention of anatomists, which were described by Baillie, and which were at one time supposed to be hydatids. They often occur in great numbers, and of all sizes; sometimes there is but a solitary cyst.

Such a condition of kidney as that which I have now described, one may readily conceive, may be easily produced by a tainted nutrition: the blood charged with the morbid matter or poison of gout furnishes to the glands an unhealthy pabulum, which, while it undergoes changes analogous to those which occur in healthy nutrition, experiences these changes in a very imperfect way, and insufficient to develop the healthy tissue of the gland. The contraction and shrinking of the kidney is sufficiently explained by this hypothesis. When much of the vascular system of the gland has been obliterated, partial congestions take place as a necessary consequence. Some of the Malpighian bodies would contain too much blood, while others would be imperfectly supplied with it. From those which are too full, effusion of serum would take place into the uriniferous tubes, furnishing the small amount of albumen which is found in the urine. Lastly, the small quantity of epithelium which is formed in the gland indicates a very defective elimination of the urea and uric acid, and the other elementary constituents of the urine. Hence the urine in these cases is

pale and of low specific gravity, and contains these products in very limited quantity.

To this state of kidney, when associated, as it very frequently is, with a decided gouty diathesis, I would give the name of "the gouty kidney." To what extent the changes which have taken place in it are due to inflammation, or how far simple inflammation, untainted by any morbid matter in the blood, is capable of producing similar alterations, I do not undertake at present to decide. Rayer has recognised the small and contracted kidney as the result of chronic inflammation, and it has been viewed in this country chiefly as the last stage of Bright's disease. This latter interpretation of it, I now feel convinced, must be erroneous.*

As to the diagnosis of this disease, we may gather the principal points which will assist us from the history of this and the other case to which I have referred.

The patient is evidently of gouty habit, as evinced by general signs and by his family history, by his habits of living, and by his having had, to a greater or less extent, attacks of gout in his limbs. There is more or less of dropsy, although this is by no means a necessary symptom, neither is the dropsy so general nor so great as in Bright's disease. The quantity of urine is not, in general, diminished, but, on the contrary, is either normal or increased; and it is pale, of low specific gravity, and deficient in the organic principles, whilst it contains albumen *in small quantity*. The sedimentary matters found in the urine are not, comparatively, in large quantity. Lithates are among the rarest in occurrence; indeed, when the disease is fairly established, I should say that lithates or free uric acid are not found in the sediment. Granular casts of uriniferous tubes, waxy casts of the same, generally of large size, altered epithelium, now and then fatty epithelium, and cells which are those of pus or allied thereto—these are the ingredients, of that whitish, mucoid deposit which you will always find to collect at the bottom, when the urine secreted by this kind of diseased kidney is allowed to stand in a tall conical glass for a few hours. Now and then, when an acute attack of gout threatens,

* See Lects. XIX and XX. "On those Conditions of the Kidney with which are associated Albuminous Urine and Dropsy."

or has occurred, or bronchial irritation is present, you may have lithate sediments in great abundance; but in the advanced stages blood-corpuscles and pus-cells are apt to occur.

Under these circumstances, the particles of pus, I apprehend, do not come from the kidneys, but from the mucous membrane of the infundibula and pelvis of the kidneys, and the ureters, over which the acrid gouty urine flows.

Let me conclude by directing your attention to the state of the joints, which is not unlike that of the kidneys.

During the life of our patient, the knee-joints were distended with synovia; they continued in this state till death. A large quantity of this fluid, presenting its usual viscid character, escaped when the joints were laid open. Litmus paper was strongly reddened by it, proving that it had an acid instead of its normal alkaline reaction. This I have found in several cases of the same kind. Both in gout and in rheumatic fever the synovia of the joints is apt to become acid.

The synovial membrane lining the ligaments of the joints and the synovial fringes were very red, from an undue degree of vascularity. Here and there it was sprinkled over by a fine white powder, which adhered to it. The articular cartilage had entirely disappeared, and a layer of white matter, like plaster of Paris, had usurped its place: the articular surfaces of both tibia and femur, and even of the patella, were covered with this substance. From the examination of it in similar cases, we presume this to be the well-known urate of soda, combined with phosphate and carbonate of lime. It was easy to understand from such a state of the articular textures as this, how the bones grated on each other as the joint was moved. It may be observed, that in this case there was no accumulation of urate of soda within the joint or on its exterior, or in the sheaths of tendons in its vicinity. Both knee-joints exhibited these appearances, but they were much more strongly marked in the left. All the other joints examined were similarly affected, namely, the shoulders, hips, elbows, and even the sterno-clavicular articulations, those of the left side being always worse than those of the right. Some of the phalangeal joints of the fingers and toes had suffered in a similar way.

This curious state of the articulations suggests many

reflections upon which I have not time to dilate at present. Let me, however, content myself with remarking how prone the morbid deposit has been to attack the cartilages of the joints, the nutrition of which it seems to have completely destroyed; how sparingly (comparatively) it touched the synovial membrane which lined the ligamentous surfaces, and how much it kept aloof from the more superficial textures in the vicinity of the joints. In other cases we find it in the sheaths of the tendons and under the integuments, and interfering but slightly with the articular surfaces; and in many instances it both attacks the joints, and accumulates around and outside them, and it is also occasionally seen to be deposited in the external ears, and in the coats of the arteries.

I must here introduce another case very much the counterpart of that of S. Rapley. I quote it in a condensed form from a lecture on renal epileptic coma, in which it was detailed for the purpose of illustrating the nervous phenomena which follow this derangement of the kidney.*

Case CXXXI.—M. A. Parry, aged forty-four (vol. xlii, p. 5), a married woman, mother of twelve children; her father died of gout and dropsy.

This woman had a remarkably dirty sallow complexion (very common in cases of chronic renal disease); she had been losing flesh very much for some time, and was in a state of great debility. Ever since her twenty-fourth year, she had been subject to attacks of gout. Almost every year she had an attack, which sometimes lasted only a week, sometimes five or six weeks. It affected both the large and the small joints of the extremities, and left deposits in the tendons and ligaments of the finger-joints. For some time past her breathing had been getting short, especially upon exertion.

The illness, on account of which she sought admission into the hospital, came on five weeks previously with increased dyspnœa and an attack of gout in her ankles. The gouty attack yielded, but the ankles remained swollen, and from that time her lower extremities and abdomen quickly became

* 'Clinical Lectures on Paralysis, &c.' Lect. 48.

dropsical; and the quantity of urine passed, which previously had been considerable, gradually dwindled down to only a pint in twenty-four hours. The whole of the lower extremities were œdematous, and the integuments of the abdomen and of the loins were in a similar state; and a considerable quantity of water had accumulated in the peritoneum, causing great distension of the abdomen. The urine was loaded with albumen.* Pulse very small and weak; heart's sounds feeble and distant, with a very distinct mitral systolic bellows-sound. The superficial jugular veins were large and distended, and when compressed in the neck did not empty themselves readily on the cardiac side of the point of pressure. She had cough with free mucous expectoration, and a good deal of crepitation was heard throughout both lungs.

Upon microscopical examination by Dr. Conway Evans, the urine was found to contain crystals in such abundance, that everything else which composed the sediment was almost totally obscured; here and there, however, a large and small waxy cast were seen. The crystals were chiefly thick, yellowish lozenges (uric acid), and large, irregular-shaped masses, apparently consisting of the above-mentioned lozenge-shaped crystals aggregated together. With these there were many of the masses provided with projecting needle-like processes, generally regarded as urate of soda, and also many colourless rectangular prisms; also a good deal of broad pavement-epithelium, probably from the vagina.

The case was regarded as one of gout, with gouty kidney, upon which acute inflammation had supervened. To this was added disease of the heart, consisting of imperfection of the mitral valve, probably from shrinking of the chordæ tendineæ and the fibrous basis of the valve, with dilatation of the ventricles, especially that of the right side; œdema of the lungs; congestion of the liver, consequent on the feeble action of the heart. As a distinct grating was felt on moving many of the

* The existence of so large a quantity of albumen in the urine is quite exceptional in cases of gouty kidney. Two circumstances seem, in this instance, to have contributed to the increase of the albumen; first, a state of acute renal irritation, caused by the recent attack of gout, and, secondly, the dilated condition of the right side of the heart, which favoured venous congestion.

small joints of the fingers and toes, it was inferred that their cartilages were replaced by gouty deposit, and it was conjectured that a deposit of the same nature damaged the valves of the heart.

Under the use of diuretics and sudorifics the quantity of urine increased to upwards of a quart for the first ten days, the albumen being still very abundant, and the belly diminished in size. On the 30th a diminution took place to a pint and a half; the pulse rose from 100 to 120; she had passed a bad night, and complained of being much troubled with convulsive movements of the arms and legs.

On the 31st, at six in the morning, she got out of bed and sat up for a short time before the fire, as if she had been chilly. Soon after getting into bed, she was suddenly seized with a fit, in which the whole body was convulsed; she foamed at the mouth, and the tongue was protruded between the teeth and bitten. This was followed by several other fits, each lasting about ten minutes, leaving her in a state of extreme prostration and exhaustion, and apparently insensible, until another fit was about to come on, when she would get up in bed and answer questions; and expressed herself as feeling very well, and free from pain. Between the fits the pupils remained very much contracted, but during them, they became greatly dilated, and the eyeballs were drawn upwards and inwards. In twelve hours she had eight fits, and as each lasted about ten minutes, there was an average interval of about an hour and twenty minutes between them.

The head was shaved, a large blister was placed over the occiput and down the back of the neck, and a quarter of a grain of extract of elaterium was administered every three hours. She was allowed small quantities of beef-tea and a little wine.

She was soon freely purged by the elaterium, and the fits ceased on the evening of the 31st. On the 1st of June she had quite recovered her consciousness, and from this time she gradually improved, still under the influence of elaterium. The quantity of urine increased considerably, and at the same time free watery discharges were passed five or six times a day from the bowels. The dropsy disappeared, and the heart's action

improved in force. On the 29th of June the elaterium was discontinued altogether.

On the 3d of July the urine diminished in quantity from a quart and six ounces to a pint and a half, and a slight fit occurred in the night; the elaterium was resumed on the 4th. From this time the urine increased rapidly, and the quantity of albumen in it became much diminished. Although weak, she left the hospital on the 20th, being anxious to return home.

This patient continued pretty well until the beginning of October. On the evening of the 6th of that month, she was brought to the hospital quite insensible, and suffering from a succession of epilectic fits, just of the same nature, and as violent as before, but with such rapidity that the intervals between them did not allow of anything being given, either food or medicine. They continued throughout the greater part of the night, and she died early the next morning, not a gleam of consciousness having shown itself.

The examination of the body took place on the next day. I shall quote merely the description of the kidneys.

The kidneys were small, contracted, and seemed much wasted at the expense of the cortical substance, the cones in many instances reaching almost the very surface of the organ; in some of the cones there were *opaque streaks of deposit of urate of soda,* taking the direction of the tubes, and probably occupying the canals of some of them. There was no healthy epithelium in any part of the cortical substance; in some situations the cells were filled with oil; in other places they were opaque. The walls of the minute arteries were thickened and hypertrophied. Each kidney weighed only three and a half ounces.

With regard to the immediate cause of death in both these cases (Rapley and Parry), it seems quite clear that that event was brought on by the same influence which gave rise to the paroxysms of epilepsy; and as the patient had not been epileptic previously, some new cause must have arisen to produce these fits. This was to be found in the deficiency of the urinary secretion, which had almost ceased immediately prior to the first epilectic seizure. The retention and accumulation of urea and other elements of the urine in the blood, in an already much

vitiated state of that fluid, were quite sufficient to create the irritation of the brain on which these fits depended. And in the wasted and atrophic state of the kidneys which we have described, was it to be wondered at that the urinary secretion should have failed?

How little, then, is to be done by treatment in such cases as these! In the early periods, to support the strength, and excite the secretions of the skin and bowels, as far as could be accomplished with safety, were the obvious indications. But when the kidneys failed—and such kidneys—what could we do? To open a vesicated surface on the back of the neck or on the head might relieve the cerebral circulation of some of the noxious material circulating in it, and so postpone the evil day for a very brief space. To establish any abundant discharge through some other emunctory would be obviously indicated, and would be useful where sufficient strength existed; hence we should have recourse to purging, sweating, or free dilution, whereby not only might the noxious matter be diluted, but some of the fluid, ready to escape through various outlets, might find its way to the kidneys, and re-establish a little action of those organs.*

I conclude this lecture with some other examples in illustration of the clinical history of gouty kidney, and of the form of gout with which it is associated.

* Case CXXXII.—Shortly after this lecture was delivered, I received a kidney from my friend, Mr. Robert Ceeley, of Aylesbury, taken from an extremely gouty subject. This kidney was in every respect similarly wasted and contracted like that described in the lecture; but, in addition, there *were deposits of urate of soda in the uriniferous tubes of many of the cones.* The particles of the salt were readily recognised by their peculiar processes (needless), which seem to radiate from a central mass: these are well figured in Dr. Golding Bird's book. They filled up parts of some of the tubes, and here and there appeared to be free among the particles of the epithelium, some of which contained oil in considerable quantity.

Mr. Ceeley informed me that this patient's urine was albuminous, lately scanty but not high coloured, and he had incipient œdema of the right leg. He was carried off by cholera, which was then prevalent in the neighbourhood. The articulations were loaded with chalky deposits, and the cartilages replaced by the earthy indusium, just as in the case described in the lecture. There was also a contracted state of the liver.

In the following case we were unable to obtain an inspection of the body after death, but the clinical features of the disease were so marked, and especially as regards one of its modes of elimination, that I am reluctant to withhold the case merely on account of the absence of that link in the chain of evidence, important although it be.

Case CXXXIII.—John Lovekin, aged thirty-eight, admitted Oct. 9th, 1852. Ten years before his admission this man was attacked with gout for the first time; it seized upon the great toe of his right foot first, and attacked all the other joints in succession; the attack lasted a fortnight. He attributed it to wearing a pair of tight boots, which injured his great toe by pressure.

Ever since this time he has been subject to attacks of a similar nature, varying in severity and duration, and recurring at very short intervals. He reckoned that he had had as many as a hundred of these attacks.

He stated that as the attack passed away, the cuticle of the affected joints would peel off. His urine has always been of a pale colour, and but seldom threw down any sediment.

His last attack took place nine or ten weeks before his admission, at which time his legs became swollen, and pitted on pressure. The swelling did not extend above the knees, and was most evident at night.

The ends of the fingers of both hands contain deposits of chalky-looking matter, which first appeared nine or ten months ago, and all the small joints of the fingers are damaged by similar deposits, and the articular surfaces grate against each other.

He seems to have inherited the disease from his mother, who was greatly afflicted by it.

Our patient was also suffering, at the time of his admission, from dyspnœa, with rhonchus and sibilus, all through the lungs; the heart's action was weak and very irregular. He had been subject to these asthmatic attacks from boyhood, and appeared to have inherited them from his father. The urine was very copious, of specific gravity 1009, and albuminous.

On the 13th of October, four days after his admission, we

noticed a certain wildness in the expression of his face, and he did not readily answer the questions put to him, and had been sick three times since morning.

On the 14th, at 2 p.m., we found him highly delirious, and ascertained that he had been so all night. He was noisy, calling out and speaking incessantly. In the morning he became affected with twitching of the muscles of all the limbs, more especially of the fingers. The movements of the fingers were constant, and the twitchings of the arms succeeded each other at very short intervals (half a minute or less). The pulse was very irregular and weak, 60; the respirations 20; pupils contracted. No urine had been passed since 6 a.m. During the morning the twitchings increased very much, both in frequency and violence, and his strength rapidly failed. At 3 o'clock he seemed to be speedily passing into a comatose state, and he lay on his back gasping and exhausted.

At 2·30 p.m. a drachm of blood was taken from a vein in the arm, for the purpose of testing for the presence of carbonate of ammonia. The blood was allowed to flow into a test-tube, and a glass rod was placed in the tube, and held for some minutes over the surface of the blood. Some very slight fumes appeared, but were not more distinct than if the rod had been held over water, with which these fumes were afterwards compared. An equal bulk of hydrochloric acid was afterwards added to the blood, but not the slightest effervescence occurred; not a single bubble of gas could be detected.

The patient was made to breathe for some minutes upon a moistened piece of reddened litmus paper, but no perceptible change of colour took place. A similar piece of reddened litmus was placed in the axilla without change. The mucous membrane within the lips manifested a well-marked acid reaction.

The symptoms under which this patient laboured during the last days of his life, and under which, despite of free blistering, and purging by elaterium, he succumbed, corresponded exactly with those of renal coma from the stoppage of the eliminatory action of the kidneys. The existence of albumen in the urine, and the low specific gravity of that fluid, which was devoid of uric sediment, indicated that the kidneys were of the small contracted kind. And the presence of deposits of urate of soda

about and in the joints showed a system thoroughly contaminated by that product of gouty disease, the kidneys sharing fully in the general contamination, and degenerated, no doubt, to a condition precisely similar to that described in A. Tapner's case (Case CIII, p. 374).

In the next case we have an example of the gouty paroxysm coming on in a state of constitution much damaged by the gouty poison.

Case CXXXIV.—M. Woodham, aged twenty-eight, a man of intemperate habits; his father and grandfather drank hard, and were martyrs to gout. Intemperance seems to have been a family failing, and our patient was early addicted to it.

His first invasion of gout was at the age of seventeen. It came on very suddenly in the left ankle, which did not swell much, but was extremely painful. Twelve leeches were applied, which relieved the pain, but the joint has been weak ever since. These attacks have been of very frequent recurrence, and have involved all the joints, there being often no more than three months' interval between one attack and the next.

Within the last twelve months (prior to his admission), this patient got into the habit of taking colchicum to relieve the pain of his attacks. He has found it necessary to take it in increasing doses, and the fits now, instead of lasting a week, as at first, usually remain a month or even two. He states that his urine is never high coloured, but usually pale and in good quantity, and free from sediment.

It was for one of these attacks that he was admitted into the hospital, February 11th, 1851. The left hand and wrist were the joints affected, and they were red, swollen, and painful. The phalangeal articulations of both hands were distorted, and could be felt to grate on each other. A similar grating was observed when the bones of the knee-joint were made to move on each other, and it was felt distinctly by the hand placed over the patella. On the second day after his admission the right hand and wrist became affected with gout, and the great toe of the right foot was red and swollen; the right knee was also painful, and its synovial membrane distended with fluid. He had a quick and throbbing pulse, sweated freely, and seemed very low. The

urine was abundant, pale, 1010 to 1015 in specific gravity, and slightly albuminous.

This patient was treated by moderate purging with white mixture (sulphate and carbonate of magnesia), small blisters to the joints, and occasional doses of morphia at bedtime. Brandy was given in small doses through the day.

At the end of a week the symptoms had very much abated, and on the tenth day of treatment the gouty swellings of the joints had quite disappeared. On the 3d of March a small collection of chalky matter was discharged from one of his fingers. This patient remained under observation for six weeks, and left the hospital quite recovered as regards the gouty affection; but during the whole time, and at the date of his discharge, the urine was more or less copious and albuminous.

The next case affords a good example of the clinical phenomena exhibited in the favorable instances of this affection, and is also worthy of note from the fact that the amount of dropsy was greater than is usually met with.

Case CXXXV.—William Hunt, aged fifty-six, admitted into Rose ward, November 4th, 1852 (vol. xxxix, p. 1), a man of intemperate habits, and accustomed to indulge freely in beer and spirits.

He had suffered for the last twelve or fourteen years from gout, and just on his admission was recovering from one of his gouty attacks of about two weeks' duration.

When brought to the hospital he had dropsy, which he first noticed a month previously, when he found his legs, thighs, and scrotum swollen. The swelling has since been gradually increasing.

The following is the description of this man, as entered in the Case Book by my clinical clerk:

"The patient is of a pale, pasty, unhealthy look; he is somewhat emaciated, and his skin is dry; his appetite is not defective, nor does he complain of any thirst. Pulse 84; artery hard and curved on each side. His belly is much swollen, but flaccid, and fluctuation is very marked; its superficial veins are swollen and turgid; the liver does not seem enlarged; bowels confined; chest natural; breath and heart's sounds healthy,

except a slight roughness of the first sound of the heart at the apex.

"Micturition is frequent, and in small quantity at each time; about three pints of urine are passed daily. The urine is of a somewhat smoky appearance, of very low specific gravity (1008), decidedly acid, and deposits a little albumen with heat and nitric acid.

"He complains of a dull, intermittent pain across the forehead and in the temples; it is most severe when he awakes in the morning. His legs and ankles are decidedly œdematous, and pit on pressure.

"All the joints of his feet and hands, and also his knees, are more or less enlarged; in the right knee, and in some of the finger-joints, there is a quantity of fluid; in the others there is a deposit probably of urate of soda on the articular surfaces, since they grate when moved on each other; there is a similar deposit on the articular surface of the left patella, which grates very much when moved over the joint."

Examined under the microscope, the urine was found to contain granular and large waxy casts, and a few blood-corpuscles.

Two days after admission this man had an attack of gout in his right wrist and hand, which had the effect of increasing the smokiness of the urine, owing, doubtless, to an escape into it of a greater number of blood-corpuscles.

Under the free use of alkalies, aperients, and some Dover's powder at night, and a blister to the wrist, this attack yielded completely in four days. The urine, which had diminished under the attack, now increased in quantity—two pints and a half daily; specific gravity, 1008; less smoky.

On the 12th of November the urine reached three pints and a half, specific gravity 1007, acid, light coloured, slightly turbid; the smokiness had vanished, but albumen was still present in small quantity; the abdominal swelling was diminishing, the anasarca was gone, and the articular effusions had quite disappeared. The right patella was found to be rough like the left. He was then taking ten grains of the bicarbonate of potass in an ounce and a half of water thrice a day, and five grains of the compound colocynth pill every night. On the 25th the report states, "urine from three to five pints for

the last four days; the fluid in the abdomen is still considerable."

Ten grains of compound iodine ointment were ordered to be rubbed over the right hypochondrium twice a day. On the 29th, a decided diminution was observed in the size of the belly. The quantity of urine passed was never less than three pints.

On the 13th of December the signs of fluid had disappeared, and he was now quite free from a day. All remedies were omitted, excepting one grain of quinine in a state of solution thrice daily. A large quantity of urine continued to be voided daily, sometimes as much as six pints in the twenty-four hours.

On the 20th of December this patient left the hospital free from both gout and dropsy, the articular surfaces remaining as before, roughened by the deposition of urate of soda and the absorption of their cartilages. The urine was still very abundant, of specific gravity 1008, acid, and containing albumen in small quantity.

Case CXXXVI.—In the spring of the present year I attended a gentleman, aged fifty-six, who exhibited, in a striking manner many of the clinical features accompanying this peculiar disease of the kidneys, and the form of gout in which it is apt to occur.

This gentleman had spent the earlier years of his life in Ceylon, where he had not suffered from any illness. He returned to this country at the age of forty-five, and very soon afterwards began to suffer from attacks of gout. He was not at all addicted to high living, nor to much indulgence in wine or beer, but appears to have been very moderate in his habits.

The gout at first attacked the joints of his feet and ankles, and afterwards the knees. Subsequently it affected the hands and wrists. The attacks were extremely painful. With the view of eradicating the disease, the patient gave up all alcoholic liquors, beer, wine, &c., and became a complete teetotaller. And finding no relief to his pain except under the use of colchicum, he took Blair's pills very freely, and became an enthusiastic patron of that empirical preparation, which is known to consist chiefly of the acetic extract of colchicum.

Notwithstanding his diligent use of this drug, which he was

led on to take in gradually increasing doses, the gout gained ground, and attacked all the large as well as the small joints of his extremities. Urate of soda began to be deposited in them, replacing the articular cartilages; and considerable accumulations of this substance were formed outside the joints in the subcutaneous areolar tissue. It was deposited largely beneath the skin of the thumb and fingers, and especially at their points, and disfigured them considerably.

Mr. P— was in this state on arriving in town, about the middle of March, 1856. Notwithstanding the constant use of colchicum, and abstinence from fermented liquors, he experienced a fresh attack of gout every alternate day in one or more joints; while there were also indications of its affecting the heart, and threatening the stomach. The pain was so acute that his nights were quite sleepless.

To relieve the sleeplessness, his medical attendant gave him a small dose of Dover's powder. This produced an effect so much out of proportion to the magnitude of the dose, that I was asked to see him.

I found him in a lethargic condition, very much as if he were suffering from an overdose of opium. The pupils were contracted, and, although he was easily roused and clearly apprehended the questions put to him, his eyelids dropped as he answered them, and he seemed every moment falling off to sleep.

His complexion was sallow; the subcutaneous tissue was everywhere contaminated with urate-of-soda deposits, which were accumulated in large amount at the points of the thumb and fingers, giving them a remarkably club-shaped appearance, and also about the wrist and elbows. Under the skin of the external ears, where they are not uncommonly met with, they were in larger masses than I had ever before seen in those parts. There was slight puffiness of the ankles, indicating a tendency to anasarca.

The urine had fallen in quantity considerably below the natural standard; it was pale and slightly smoky, and quite free from sediment; of acid reaction, and distinctly, but slightly, albuminous.

I had no difficulty in coming to the conclusion that this

patient was labouring under the form of renal disease described in the lecture, and that at that moment he was suffering from a semi-comatose state, due partly to the defective action of the kidneys, and partly to the influence of a very small dose of opium, of which his peculiar condition rendered him unusually susceptible.

The object of treatment was first to relieve this semi-comatose state by promoting elimination, and secondly to prevent the frequent recurrence of the attacks of gout. With the first view he was freely purged by equal parts of the compound gamboge and colocynth pills, and the citrate of ammonia was given in effervescence, allowing an excess of alkali, partly to obtain the influence of ammonia as a diffusible stimulant, and partly to promote the action of the kidneys. To ward off the attacks of gout, I insisted on the abandonment of colchicum, to which he yielded his consent with great reluctance, and, equally against his will, I prescribed for him frequent small doses of brandy and other nourishment, in regulated quantities.

Under this treatment, after the second or third day, the attacks of gout ceased, the bowels were freely acted upon, and the kidneys began to secrete abundantly; but the urine was pale, without sediment, and contained albumen in small quantity. The amount of this secretion increased to five or six pints daily. Concurrently with these changes our patient quickly lost his semi-comatose state; his nights became much better, and were greatly assisted by his being placed on a water-bed. After ten or twelve days he was out of danger.

From time to time I took the opportunity of opening with a lancet the collections of urate of soda which so much deformed his fingers, and promoted the free discharge of that substance in a semi-liquid state. As he was well and carefully supported, the wounds healed most kindly, and the fingers were restored to their natural shape.

I attended this gentleman for upwards of two months, along with Mr. Broxholm, and he left town in a very much improved state of health, and quite free from gout; but the kidneys continued to act in the way which is so characteristic of gouty disease of these organs.

He went to Brighton, and continued to improve for some

time. After an incautious exposure to the east wind, he was attacked by influenza, during the course of which malady symptoms became developed which I interpreted as due to gout attacking the heart. The action of that organ became frightfully intermittent, and the patient gasped for breath as in angina pectoris. In one of these attacks he died, apparently from severe spasm of the heart.

LECTURE XXIX.

ON CERTAIN URINARY DISEASES.

ON CASES IN WHICH PUS IS FOUND IN THE URINE, AND ON GOUTY INFLAMMATION OF THE BLADDER.

Gentlemen,—The occurrence of such a product as pus in the urine is in itself a formidable and an alarming sign, and it is most important that you should be able to determine precisely the source whence it comes. It is very essential to your success as practitioners that you should not postpone, until a case presents itself, making up your minds as to the conditions which may give rise to so serious a modification of this important secretion. I propose, therefore, to bring this subject before you in this and my next lecture.

We have at present in the hospital two very interesting cases of this nature. One is that of a man named Rickman, in Sutherland ward; the other is that of a young woman named Jenkins, in Lonsdale ward. Both these persons are passing pus in their urine, but under different circumstances. The presence of pus, however, in the urinary secretion is the prominent feature in both cases. We speak of pus in the urine generally, just as we speak of blood or of albumen in this fluid; and we find that the presence of either of these substances must be regarded not as the essence, but as a symptom, and undoubtedly a most important one, of the disease.

These two cases resemble each other only in the fact of the presence of pus in the urine; they differ in the nature of the malady under which the patients are suffering, for in each case the pus is derived from a different source, and under conditions which require a very different plan of treatment.

Before I go further, let me say a few words on the means at our command for detecting the presence of pus. Suppose a specimen of urine is brought to you in which pus is suspected to exist, how do you proceed to detect the pus? and how can you distinguish it from other deposits which we know present to the naked eye appearances not unlike those of this product? The remarks which I shall make on this point will apply to those cases in which we have a fair amount of pus present—a quantity, in fact, sufficient to form more or less of a deposit evident to the unaided eye.

Urine containing pus, then, generally exhibits a certain cloudiness or muddiness, so that when you hold it up to the light you cannot see through it; the clear, transparent appearance of the healthy secretion is absent. If you have an opportunity of inquiring into the circumstances under which this urine is passed, you will find it has been muddy from the moment when it was passed, and that it has not become so after standing for some time. This constitutes another distinctive character of urine containing pus, and enables us to distinguish it from urate of soda, which sometimes forms a deposit that exhibits much of the general appearance of pus; for the urine from which the urate of soda is deposited is almost always perfectly bright and clear when first passed, and becomes turbid only after it has cooled, the urate of soda being generally perfectly soluble in the secretion while warm. Hence you should always be particular to ask the patient if his urine is clear when passed, unless you have an opportunity of seeing and testing it yourself; but this we are frequently prevented from doing, and it becomes important, therefore, to gain as much information as possible with reference to this very important symptom. If the urine be clear when passed, and becomes muddy only after standing, we may lay it down that the turbidity depends upon urate of soda, and not upon the presence of pus. Sometimes, indeed, a notable quantity of the alkaline phosphates is preci-

pitated from urine previously clear, but this is the exception rather than the rule.

Purulent urine, besides possessing this muddiness, has the additional characteristic, that after a time a deposit from it collects at the bottom of the vessel, and forms a layer, varying in thickness (according to the quantity of pus present), of a yellowish-green material, which has a creamy consistence. This deposit leaves the supernatant fluid more or less clear, according to its greater or less completeness; but frequently the urine continues to hold a small quantity of pus in suspension, which creates a certain amount of turbidity or milkiness in the supernatant fluid. If the urine be allowed to stand for some time, this greenish layer of pus undergoes certain changes, by which the decomposition of the urine is brought about; and the fluid soon becomes alkaline, owing to the presence of carbonate of ammonia, generated by the decomposition of urea. At the same time, the alkali, thus developed, reacts in a peculiar way on the pus, which is observed to become thick, viscid, and ropy, and to lose its minutely granular appearance, becoming more or less transparent, and forming what has long been described as *glairy mucus*. It has been found that potash (and I believe the observation was first made by Dr. Babington) also reacts in this manner upon pus, and, in consequence, it becomes a valuable reagent to enable us to detect the presence of this substance.

Here is a specimen of urine exhibiting the general characters which I have just described as being distinctive of purulent urine. You see there is an abundant creamy-looking deposit; and if I pour off the supernatant fluid, and add a little of this liquor potassæ, you will perceive the change occur which I have just mentioned. As I shake the vessel, you observe the deposit becomes tenacious and glairy, so that, when I attempt to pour it into another vessel, it does not drop, but runs off in a viscid stream, very like uncoagulated white of egg; indeed, many persons might mistake this glairy mass for white of egg. Pus altered in its character in this manner by the carbonate of ammonia, set free by the decomposition of urea, was, until of late years, ordinarily considered as a deposit of glairy or *ropy mucus;* but it has since been shown that mucus never assumes

this particular form of a ropy sediment, which sinks to the bottom of the vessel, nor does it ever exist in the urine in such quantity as we frequently find this altered pus.

I have said that purulent urine is always muddy; but this is not a character by which we are enabled with certainty to say whether or not a given specimen contains pus. Urine may be muddy from other causes; for example, it may exhibit a muddy appearance, as soon as it is passed, from the presence of other deposits besides pus. Phosphatic urine is very frequently muddy, and often much resembles purulent urine; but generally it appears paler than the latter. After phosphatic urine has been standing for some time, a deposit will be found just such as occurs in purulent urine; but, in the former, the deposit usually presents a white instead of a yellowish colour, and it is flocculent and light, instead of being thick and heavy. We also observe this very important difference between these two forms of deposit, that the addition of a little acid renders the phosphatic urine perfectly clear, while it increases the turbidity of the other. This reaction will always enable us to distinguish conclusively between a specimen of urine muddy from the presence of pus and one which is rendered turbid by the precipitation of phosphates. Phosphatic deposits are all readily soluble in dilute mineral acids, and are precipitated again from the acid solution by ammonia. Purulent deposits are not dissolved by dilute acids; but the turbidity becomes greater, owing to the precipitation of a little albumen from the liquor puris. Another distinctive character of urine containing a considerable amount of phosphatic deposit is, that it is usually, though not always, alkaline; while purulent urine more frequently exhibits a slightly acid reaction, at least when quite fresh, or it may be neutral; but we do not often meet with urine of this description of a strongly acid reaction, unless the patient is taking at the time large quantities of mineral acids. Then, as I mentioned to you just now, we have in alkalies a most excellent and easily applied chemical test, which will enable us with certainty to discriminate between these deposits; and liquor potassæ has been found the most convenient alkali which we can use for the purpose, as it will keep well, and requires no great profundity of chemical knowledge in its ap-

plication, and, so far as I know, it is not open to any sources of fallacy.

There are other points distinctive of urine containing pus. If we apply heat to the clear fluid after the subsidence of the deposit, it will coagulate, and the amount of coagulation which takes place will be in direct proportion to the quantity of pus present. This will take place if the urine be acid; if it be alkaline, you must acidulate it before heating it. The albumen is derived from the liquor puris, and hence purulent urine is always albuminous, and it is albuminous because purulent.

For an additional and unequivocal test of pus, you must look to its physical constitution. Pus consists of two essential parts, *the liquor puris* and *the pus-corpuscles*, the latter being held in suspension in the former, just as the blood-corpuscles are suspended and float about in the liquor sanguinis. The *pus-corpuscles*, or *pus-cells*, are readily recognised by the microscope. If a specimen of urine contain albumen, that substance may be derived from the liquor puris, and may, therefore, be indicative of the presence of pus, or it may be due to the escape of serum only, as occurs in the various forms of chronic renal disease. This point may be at once settled, as regards the presence of pus, by examining a drop of the turbid urine under the microscope, when we shall not fail to recognise the pus-corpuscles, if this product be present in the urine. These particles are much larger than the red corpuscles of the blood, and differ from them in shape, being globular, while the latter are biconcave discs. They much resemble the colourless corpuscles of the blood, but appear darker and more highly granulated. This granular appearance seems to be owing to the presence of numerous highly refracting molecules in the pus-corpuscle, which are of a fatty nature; and to them, most probably, the change which occurs on the addition of liquor potassæ is due, the fatty matter being converted into a soap by the alkali. When treated with acetic acid, the pus-corpuscles exhibit two or three circular bodies in the centre, having much the appearance of oil-globules. Some corpuscles are found to contain three or four of these bodies, others two, and in some one only can be detected. The presence, then, of particles

like these will enable you to distinguish pus from all other deposits which occur in the urine.

We have also to distinguish the pus deposits from deposits of mucus. Mucus seldom forms a distinct stratum, like pus; if viscid, it is so when the urine is acid; but pus exhibits the glairiness which renders it liable to be mistaken for mucus, only when the urine is alkaline. If we examine mucus under the microscope, we shall not fail to detect more or less of epithelium, and the so-called mucus-particles in small numbers, which, doubtless, are incipient pus-corpuscles. Mucus, again, does not react, like pus, with solution of potash, and it is soluble, to a great extent, in acetic acid. Further than this, the liquor muci appears to be always free from albumen, a substance which, as I have already remarked, the liquor puris invariably contains in greater or less quantity.

Having considered the more prominent features of deposits of pus as they occur in the urine, let me say a few words upon the various sources from which the pus may have found its way into this excretion, and the circumstances which may give rise to its formation. The pus may be developed from the lining membrane of any of the surfaces over which the urine has to flow as it passes from the kidneys, or in contact with which it has to come. It may come from the urethra in the male; and in the female it is often derived from the mucous membrane of the vagina. When we examine the urine of a female, and find pus present in it, we should always be careful to inquire if leucorrhœa be present, and in very many cases this will be found actually to be the case. It therefore becomes important to keep this in view, for I have known many persons much puzzled from this simple circumstance. They have found pus in the urine, and have been unable, from the absence of other symptoms, to draw any conclusions with reference to its origin. Pus may likewise come from an adjoining abscess which has opened into one of the urinary passages. An inflammatory state of the mucous membrane of the bladder is one of the most common causes of the presence of pus in the urine; and inflammation of the ureter, and of the pelvis of the kidney (*pyelitis*), will also give rise to it; and, though last, not least, suppurative inflammation of the kidney itself. The presence of

a calculus in the kidney may cause pus to appear in the urine by creating irritation and inflammation around it.

When the quantity of pus discharged is considerable, I do not know of any particular feature, either of it or of the urine, by which one may determine whether the pus comes from an abscess in the kidney or from some of the surfaces over which the urine passes, as the pelvis of the kidney, the ureter, or the bladder.

There is a curious difference, and a very marked one, between the two cases which are now in the house with reference to the occurrence of triple phosphate in the urine. The woman Jenkins, in Lonsdale ward, has been passing large quantities of pus for some months, in which crystals of triple phosphate cannot be detected; and, from other signs, we should be disposed to infer that the purulent matter comes from an abscess of the kidney. On the other hand, in the urine of the man (Rickman) in Sutherland ward, we have always observed a considerable quantity of the triple phosphate, and there can be no doubt that he is labouring under an inflammatory state of the mucous membrane of the bladder, and that that membrane is the source of the large quantity of pus which is found in his urine. Whether the difference of the source of the pus has aught to do with the absence of alkaline phosphate in one case and its presence in the other, is an interesting question.

It has been stated by Prout, that an irritated portion of the urinary mucous membrane is apt to secrete phosphate of lime. May there not be a similar tendency to the formation of triple phosphate? I think this question must be answered in the negative, and confess our present inability to refer the difference to any satisfactory cause. Abscesses, formed in the neighbourhood of the urinary passages, and opening into them, may furnish pus to the urine. Thus a prostatic abscess may burst into the urethra, or a lumbar abscess may open into the ureter.

Let me now direct your attention to one of the cases which have led me to take up this subject to-day. It is that of the man named Rickman, in Sutherland ward.

I may here state, *in limine*, that Rickman appears to me to be labouring under gouty inflammation of the bladder. Gout may

occur in the bladder, as it does, undoubtedly, in the stomach; and the symptoms of gout in the hollow viscera are highly interesting, and deserve your attentive consideration.

Case CXXXVII (vol. xxxiii, p. 185).—Our patient is forty-four years of age. The history of the first part of this patient's illness, as recorded by Mr. (now Dr.) Bridgewater, is very much the same as that of all those who, in common phrase, "earn the gout for themselves." He was in early life a butler, and accustomed to drink freely of ale and gin. Having subsequently entered the police force, he was compelled to diminish his potations. He did not remain long in this line of life, and soon abandoned it for the business of keeping cows in the neighbourhood of London. In this vocation he has been engaged for the last five years; and, during that time, he has not lived so well as formerly, still, however, contriving to drink largely of porter.

Despite his bad habits, he enjoyed very good health until ten years ago, when he was seized with his first attack of gout. This came, as it so often does, first in the great toe. It was not, however, confined to that joint, but attacked subsequently the ankles, knees, and wrists, shifting its position from one to the other. In subsequent attacks, of which he had four or five in the course of each year, it would sometimes exhibit this shifting character, and sometimes attack all these joints simultaneously. The tendency to shift is an important feature in the case, for it is where the disease exhibits this proneness to wander that we most frequently find it attacking internal organs, especially hollow viscera, such as the stomach and the bladder. During the last ten years, then, this man has been subject each year to several similar attacks. About seven years ago, during a period of intermission between his attacks, he was one day seized, after a long walk, with violent pain in the bladder, accompanied with difficulty of micturition; and when he came to examine the urine which he had passed, he discovered blood in it. He immediately sought for medical assistance, fomentations were freely used, and he took some medicine, under which the attack subsided in a few days.

What, then, can we pronounce this attack to have been?

Was it caused by the sudden entrance into the bladder of a renal calculus, which had previously created no disturbance? or by a vesical calculus, which now for the first time announced its presence? The fact that the patient had evinced no previous symptom whatever of either renal or vesical calculus tells very much against both these views. The mode of access and the course of the attack comport with what we know of the clinical history of gout, as it affects acutely either joints or hollow viscera. The rapidity or suddenness of its invasion is the most striking feature, and especially as occurring in a highly gouty subject, whose gout is of the asthenic and erratic kind. Add to this, that the attack was of short duration, and yielded in a few days to medical treatment, and you have another strong point against its being due to the mechanical irritation of a foreign body, such as a calculus, of the expulsion of which from the bladder there was no evidence. Under these circumstances I am disposed to view this attack as one of gout in the bladder—that viscus being seized suddenly, just as a great toe or any other joint is, in the ordinary acute attacks of this disease. The inflammatory state of mucous membrane excited a rapid development of a high degree of congestion of that membrane, some of the vessels of which giving way, allowed the escape of some blood, and caused the hæmaturia which accompanied the attack.

Since the first attack in the bladder, our patient has been subject once a year to one in all respects similar, coming on in the same way, and yielding as speedily to treatment, but not leaving the bladder quite unscathed. Were the vesical symptoms due to a mechanical cause, there can be no doubt the attacks of pain in the bladder, &c., would have been very much more frequent.

For the last three years our patient has been disabled from work by weakness of the knees and ankles, the fibrous structures of which are a good deal thickened; and during this period he has had some severe attacks of gout.

About three months before last Christmas (not having had an attack of gout for six months previously), he was seized with pain in the left lumbar region, which for three days continued very severe, and was accompanied with discharge of bloody

urine, and with pain at the end of the penis, and itching along the whole course of the urethra after micturition. From this attack he got quite well under medical treatment; but in February he was again seized in a similar manner, the pain being referred to the loins, and blood being passed with his urine. From that time until his admission into this hospital (May 14th, 1851), he has suffered more or less from these symptoms, the blood disappearing from his urine occasionally for a whole week.

On admission we found him complaining of pain in the back, especially in the left lumbar region, slightly increased by pressure, and also of pain in the bladder more severe than that in the back. This latter pain was most distressing when the bladder contained urine. The power of retaining the urine was a good deal impaired, partly, no doubt, because of a highly irritable state of bladder, and partly in consequence of a weakened condition of its sphincter.

The urine had a decidedly smoky colour; its specific gravity was 1012, its reaction alkaline, and it contained blood, as ascertained by the microscope, and pus in considerable quantity. The man stated positively that he never observed any indication of gravel, and never passed any calculus. The quantity of urine passed in twenty-four hours was about two pints. To relieve the irritable state of the bladder, he was ordered a starch enema, with ten minims of tincture of opium every night, and was put upon dilute nitric acid, beginning with ten minims thrice a day in water.

He had only been two days in the hospital when he was attacked with gout in the thumb of the right hand, and three days afterwards (May 19th) the right knee became similarly affected, and a copious effusion took place into it. Both ankles were also attacked on the evening of the same day. When the articular affection was thus developed, the pain in the back and bladder became decidedly mitigated. The urine, however, still retained the same characters; it was of low specific gravity, alkaline, and contained blood and pus.

Now, the points which we had to determine in this case were these—first, the source of the blood and pus; secondly, the nature of the pain in the back; and, lastly, the actual state of the bladder.

Observe that all the symptoms which this patient exhibited might have been caused by the irritation of a calculus in the kidney, or of a calculus in the bladder.

I have already stated to you my reasons for believing the former attacks in the bladder to have been due to gouty inflammation of that organ, and not to the presence of a stone in it. The existence of a calculus in the bladder might easily have been ascertained by sounding; but our patient had a very contracted state of urethra at its orifice, and for some distance behind, which rendered it very difficult to introduce an instrument, even of the smallest size. We, therefore, for the present, contented ourselves with relying upon the evidence of general symptoms.

If the view which I took of the bladder affection were correct, it would indicate that the lumbar pain was probably due to a similar gouty inflammation or irritation of the ureter, and perhaps in some degree of the kidney likewise, and that it was caused by an extension of the vesical inflammation to the ureter, and perhaps also to the pelvis of the kidney.

Our three points, then, on this view of the case might be thus explained:

First, the source of the blood and pus is, and has been all along, the bladder; perhaps, also, the ureter: secondly, the pain in the back is referable to gouty irritation of the kidney and ureter: and, thirdly, the mucous membrane of the bladder is in a state of chronic congestion and inflammation, the subject of frequent hæmorrhages, and secretes pus.

But you will ask, how do I determine that the pain in the left lumbar region is not caused by a renal calculus, and that the kidney may not be the source of the pus? The relation of time which the vesical and renal symptoms bear to each other is strongly opposed to the suspicion that a renal calculus has been at the bottom of his illness. The symptoms were distinctly referable to the bladder long before any lumbar pain was manifested. The annual attacks of gouty inflammation of the bladder from which our patient suffered were, as you will remember, among his earliest symptoms. Now, these attacks are not to be explained on the supposition of the existence of a calculus in the kidney, more

especially in the absence of symptoms pointing to disturbance in that organ. The symptoms of renal calculus, *while it is yet in the kidney*, are generally sufficiently distinct: you will have more or less pain in the back; probably, also, bloody micturition to a greater or less extent; and, when the stone begins to move down, more or less pain, both in the loins and along the ureter, according to its size and the resistance which its passage encounters; sometimes, also, sickness, vomiting, and even hiccough occur, with not unfrequently retraction of the testicle. A renal calculus, however, may excite inflammation and abscess of the kidney, and so may cause discharge of pus and blood; but in such a case you will not have so complete nor so long an intermission of symptoms as in the present instance, nor will you have those symptoms referred solely to the region of the bladder.

A calculus may be impacted in the kidney or ureter, and cause no pain so long as it is stationary; when it begins to move, however, the pain and the disturbance begin. If it fix itself again, these symptoms may cease.

Case CXXXVIII.—You had a good illustration of this not very long ago, in the case of a man named Steventon, in Sutherland ward, who died of phthisis. Some weeks before his death he complained of pain in the back, and of very marked pain down the course of the ureter on the right side. For some time these symptoms disturbed him a good deal, but they subsided altogether three weeks before his death. I made the diagnosis of a calculus in the kidney or ureter on that side; and, after death, we found one, as large as an almond-shell, with a rough tuberculated surface, and composed of oxalate of lime, tightly impacted in the right ureter, about an inch from its opening into the bladder. The ureter and pelvis of the kidney were much dilated above the point of obstruction, and the kidney was beginning to become sacculated. In this patient there was a slightly irritable state of the bladder, owing, no doubt, to the stone passing down so near to that viscus; but these symptoms subsided when the stone became impacted and quiescent; and you will observe, also, that the vesical symptoms *followed*

the pain in the back and along the course of the ureter. It is otherwise with our patient, Rickman; with him the vesical symptoms were long antecedent to the lumbar affection; and, if a calculus exist (which is quite possible), it is probably in the kidney, and may have excited a separate inflammation of that organ, distinct from the bladder affection.

I attach much importance in the diagnosis of such a case as Rickman's to the attacks of gout being asthenic and erratic; for it is in these cases that gout is most disposed to fly to internal organs. Gout, even when apparently sthenic, will attack the bladder, the stomach, or the heart, when treated by depressing means. Of this the following is a good example:

Case CXXXIX.—A friend of mine, of very robust make, but overworked in laborious professional employment, had a severe attack of gout in one foot. He had previously suffered from it on many occasions. Being much in request in his profession, he was impatient to get well, and, contrary to his better judgment, allowed a number of leeches to be applied to the gouty foot. The leech-bites bled freely, and next day he found that the active gout had quitted that foot, leaving it very weak, and had settled in the other. This latter swelled up quickly, and so intense was the inflammation that a neighbouring surgeon, thinking that matter had formed, made an incision, which bled freely. On the following day both feet were free from gout, but the bladder had become irritable, micturition was painful and frequent, and the urine contained bloody mucus. The vesical irritation yielded in a few days, and now the action of the heart became frightfully irregular and intermittent; and, notwithstanding that the gout was on more than one occasion successfully invited to the extremities, it was many months before the heart's action was restored in strength and rhythm.

The view which I have taken of our patient Rickman's case is, I think, confirmed by the subsequent history. The treatment with which we began was persevered in,—the opiate enema, the dilute nitric acid,—which, after a short time, was given in decoction of Pareira brava, and the dose gradually increased to thirty or forty minims. Fomentations were frequently applied over the region of the bladder, and counter-

irritation was established in the same situation by mustard. The gouty joints were wrapped up in wool, so as to promote sweating, and small blisters were occasionally applied to them. A sedative and sudorific draught was frequently administered at bedtime.

Under this plan of treatment the general gouty state became decidedly less, the joints recovered themselves, the irritable state of bladder diminished, and the quantity of purulent discharge in the urine was much reduced; still the alkaline condition remained, and the pus, although in greatly diminished quantity, continued. This, I believe, was attributable to the impediment which existed to the free evacuation of the bladder, by the contracted state of the anterior extremity of the urethra, caused by an inflammation of that part of the canal in our patient's boyhood; and, accordingly, means were taken to diminish and remove this impediment by daily dilating the urethra. By perseverance in the dilating process the urine is improving much in quality, and I cannot doubt that, ere long, it will become quite natural.

When you find pus in the urine, and have reason to suppose that it comes from the bladder, it is a point of primary importance that it should have a free exit. Urine retained in the bladder becomes a source of irritation to that organ, by becoming decomposed, and developing carbonate of ammonia. It is in this way that we get ammoniacal and purulent urine in paralysis of the bladder from spinal disease. The retained urine becomes decomposed, inflammation of the bladder is excited, and pus is generated, which is made viscid by the alkali which is formed from the decomposed urea.

I shall continue this subject in my next lecture.

LECTURE XXX.

ON CERTAIN URINARY DISEASES.

ON GOUTY INFLAMMATION OF THE BLADDER, AND ON CASES IN WHICH PUS IS FOUND IN THE URINE.

In my last lecture, gentlemen, I began to call your attention to some of those cases in which pus makes its appearance in the urine, having first pointed out to you many of the sources whence the pus may enter this secretion. The case of Charles Rickman, in Sutherland ward, afforded us an instance of an affection of the bladder, giving rise to the formation of a large quantity of pus; and we had reason to believe that it was due to an inflammation of the vesical mucous membrane, which was of a gouty character.

From the history of this patient, we found that he had suffered from several attacks of gout, which alone might lead us to suspect that the bladder affection was of this particular nature. The mode of accession of the acute symptoms, as well of the bladder as of the joints, was peculiarly characteristic of a gouty affection; inasmuch as the bladder attack was sudden and without warning, and the articular affection exhibited an erratic character. You have had an opportunity of watching this case for some time, and most of you are, no doubt, aware that the patient suffered from a stricture at the anterior orifice of the urethra, which, by preventing the free exit of the urine, kept up the irritation of the bladder. For this reason, the disease still remains uncured; but, in the main, our treatment has been successful. The stricture, by preventing the free passage of the urine, keeps up an irritable state of the bladder, so that this viscus refuses to retain even a small quantity of urine, and hence a necessity for very frequent micturition results,

The urine is, however, now acid, having been, as you know, a considerable time, highly alkaline, — so alkaline, indeed, as to produce a characteristic reaction on the pus-globules. This, as I before explained to you, consists in the transformation of the purulent deposit into a thick and glairy jelly-like mass—the so-called glairy mucus, and is the result of the action of all alkalies on this substance. Under the long-continued use of the dilute nitric acid, gradually increased to large doses, —sometimes given with decoction of Pareira, sometimes with infusion of buchu,—and the exhibition of opium, chiefly by enema, the irritability of the bladder has greatly diminished, and the gouty symptoms have been subdued; and the patient will, I doubt not, ere long leave the hospital quite well.*

In connexion with this case, let me offer a few remarks on the occurrence of gout in the bladder, and describe to you some of the forms in which it affects this organ. I shall confine myself, in these remarks, to what I have myself observed in cases which have from time to time been brought under my notice.

Gout appears to me to affect the bladder in four different ways:

1st. It manifests itself as a distinct and very obvious inflammatory affection; and this was the form in which it occurred in our patient Rickman. I apprehend that, in these cases, the mucous membrane of the bladder is red and inflamed, presenting, indeed, the ordinary appearance of a mucous membrane in a state of inflammation. It is a condition, however, which must be distinguished from inflammation of the bladder, as excited by other causes, and unconnected with any specific poison. Gouty inflammation of the bladder is an analogous affection to gouty inflammation of the lungs, gouty bronchitis or pneumonia, and gouty inflammation of the stomach. In cases of this kind there is a great tendency to the secretion of pus by the mucous membrane of the bladder. If there be any difficulty in the free evacuation of the pus, the urine becomes alkaline, from the retention of a small quantity of the secretion, and the

* This patient remained in the hospital from the middle of May to the end of July, and was discharged quite free from all vesical irritation, and able to pass water in a good stream.

subsequent decomposition of the urea; the highly alkaline urine, in its turn, keeps up the irritability of the bladder, and promotes the secretion of more pus. In this way, either a weak or paralytic state of bladder, an enlarged prostate, or a stricture in the urethra (as likewise exemplified by the case of Rickman), may stand in the way of the complete restoration of this organ to its healthy function.*

2d. Gout occasionally attacks the bladder in a different manner to that last described, so as to produce just the opposite effect as regards the urine, namely, incontinence. A gouty man becomes troubled with great frequency of micturition, and we find that this symptom depends upon a highly sensitive state of the vesical mucous membrane, which leads ultimately to an inability of that organ to retain the urine, due, not to a paralytic condition of the sphincter vesicæ muscle, but to the bladder being impatient of its contents. In this form, the sensibility of the mucous membrane is much exalted, and the bladder becomes intolerant of the presence of the smallest quantity of urine, so that the evacuation of its contents is constantly taking place at short intervals. The prominent symptom, then, in such cases, is frequent micturition of small quantities, the urine passed being pale, acid, and devoid of mucus or pus; but not unfrequently albuminous, owing to the existence of gouty disease of the kidneys.

It is difficult to define the exact pathological condition of the mucous membrane of the bladder in this affection. It appears to be an irritable rather than an inflammatory state,—a condition in which the sensibility of the mucous membrane of the bladder is greatly exalted, owing to the influence of the gouty poison, which seems capable of irritating the bladder just as cantharidine does. Doubtless there is frequently also in these cases an irritated state of kidney with which the bladder sympathises. The cases in which it is apt to occur are generally in elderly persons, whose systems seem thoroughly imbued with gout, and in whom deposits exist in the joints, in the tendinous

* A striking example of this form of gout is referred to in the preceding lecture, p. 563. In that case the rapid transference of gout from the foot to the bladder was as obvious as it so often is in being transferred from one joint to another.

sheaths, or in the arteries. It occurs in old persons, and often accompanies enlargement of the prostate gland. Sir Benjamin Brodie describes cases which I suspect are of this nature, the primary cause of the symptoms being gout. "An elderly man," he says, "complains of frequent attacks of giddiness. Sometimes, in walking, his head turns round, so that he is in danger of falling; and this symptom, probably, arises from an altered structure of the arteries of the brain, causing an imperfect state of the cerebral circulation. This state of things is sometimes attended with an irritable condition of the bladder; and although the urine is of a healthy quality, and the bladder itself is free from disease, the patient is tormented with a constant micturition, voiding his urine without pain, but at short intervals, and in small quantity."*

3d. A third class of cases exhibits a condition the opposite of that which I have just described, in which, instead of the patient being unable to retain even a small quantity of urine in his bladder, he is suddenly or rapidly affected with an inability to pass water, and the bladder becomes distended in consequence, causing great pain and suffering. The essential difference between these two conditions consists in this, that in the former case the mucous membrane is rendered highly irritable by the gouty poison, and kept so, possibly, by some irritating quality of the urine; but in the latter case the muscular coat is the seat of the affection. There is ample evidence to show that muscles may be attacked by the rheumatic or by the gouty poison. Thus, in subjects of gouty diathesis, it is not uncommon to meet with sudden and severe affections of external muscles, accompanied with constitutional disturbance similar to that of acute gout. I am just now attending a nobleman in whom very decided constitutional disturbance, accompanied with distressing intermission of the heart's action, preceded for some time the sudden appearance of a very painful inflammatory affection of the same portion of the gastrocnemius muscle on each side, which came on in the sudden way in which gout is apt to do. This patient passes uric-acid in large quantities, not only as urate of soda, but as uric-acid gravel, and he

* 'Lecture on the Diseases of the Urinary Organs,' p. 94.

has discharged many uric-acid calculi. Lumbago is an instance of gouty affection of muscles. The intercostal muscles are often similarly attacked, giving rise to a most painful affection, which occasionally ends in pleurisy, or even pleuro-pneumonia. Just in the same way gout may attack the muscular fibres of the bladder, stomach, or colon; and in the cases of retention of urine such as I am describing, it affects the muscular coat of the bladder so as to paralyse it, in a manner probably somewhat analogous to that in which the active principle of belladonna affects the circular muscular fibres of the iris, and allows the pupil to become dilated.

Case CXL.—Let me relate to you a case in illustration of this form of gout in the bladder. A barrister of great eminence in his profession was obliged to return to town from his circuit, where he was largely employed, and, indeed, overworked. He had been seized with severe muscular pains in the thighs and loins, which I regarded as gouty. The patient was of a gouty family, generated uric acid freely, and had passed a considerable quantity of uric-acid gravel. On a former occasion, I had attended him for one of those attacks of sudden affection of the intercostal muscles (muscular gout, as I would call it), passing on to dry pleurisy. For these reasons, I was justified, I think, in regarding and treating these pains as gouty in their character. After he had been three or four days under treatment for this affection, he found one morning, on attempting to empty his bladder, that it refused to discharge its contents. A complete paralysis of the bladder had taken place, and evidently not from too great distension, as the patient did not suffer much inconvenience, and the quantity of water which had accumulated was not considerable. Under a soothing treatment, with slight counter-irritation over the region of the bladder, this paralytic state gave way within four-and-twenty hours, but it was several days before the full power and tone of the bladder were restored. Under a regimen, directed to oppose the gouty tendency, and the frequent free use of the Vichy water, this patient has continued quite free from any symptom of gout for many years.

4th. Gout attacks the bladder, in some cases, in the follow-

ing manner (and I take my remarks on this head from a case which actually came under my observation) :—A gouty man indulges more freely in the delicacies of the table than he is usually wont to do; perhaps he is guilty of some indiscretion in what he partakes of, eating cheese or some other indigestible matter which disagrees with him. Before he goes to bed, he is suddenly seized with violent pain in the region of the bladder, which in some cases lasts an hour, but in others continues to torment the patient for a much longer time, preventing him from sleeping, and often producing great distress. This condition is usually relieved by free counter-irritation, the administration of alkalies, and the cautious use of opiates.

If, then, you find a man labouring under any of the four conditions of vesical affection that I have described, and at the same time you are able to discover, from his history, symptoms characteristic of an acute or chronic gouty state, and you are convinced of the absence of calculus, you may conclude that the symptoms are dependent upon a gouty affection of the bladder, and your treatment should be influenced accordingly. It must, however, be borne in mind, that a stone will cause the development of very similar symptoms, and it will, therefore, sometimes be necessary to sound the patient carefully, in order to determine the presence or absence of stone. The sudden invasion, the existence of the gouty diathesis, and the absence of other causes to account for the symptoms present, mark the peculiar nature of the affection, and concur in making us suppose the disease to be of gouty nature.

Being decided as to the diagnosis, what means are we to adopt to relieve the symptoms? The treatment in these cases is obvious and simple. First and most important is free counter-irritation; but you must apply your counter-irritation carefully, and consider what form of counter-irritant will be best suited to the case. Blisters would be improper, because cantharidine, which is the active principle of the blistering-plaister, is a direct irritant to the mucous membrane of the bladder, and would, therefore, tend rather to increase than to diminish the distress. Neither must turpentine be employed, because it irritates the kidneys, and this irritation is liable to be propagated to the bladder. Mustard is the most effectual counter-

irritant which we can use in these cases, and has not the disadvantages of the former remedies. Strong ammonia may likewise be used as a counter-irritant. Our next consideration should be to assuage pain, which in many instances is a most urgent symptom; and this we should endeavour to do in the speediest and safest manner possible. Of whatever form the affection may be, the best plan is to give opium. This may be done by the endermic method, by rubbing in a strong opiate liniment over the region of the bladder. Or, as is much better and more certain in its action, the opium may be given in the form of an enema injected into the rectum. About half a drachm of laudanum, mixed with a small quantity of decoction of starch, of which not more than an ounce and a-half or two ounces should be employed, may be gently injected into the rectum. This you will find will act as a sort of warm opiate poultice to the bladder; in this way all kinds of irritability of this organ may be relieved. The irritable state of the bladder caused by cantharidine (strangury) is effectually relieved in the same way, and gouty inflammation is benefited in like manner. The action of cantharidine, indeed, forms a pretty illustration of the manner in which we may suppose the gouty poison to cause the vesical irritability. And both strangury and vesical gout may be treated in a similar manner. If the patient be not quite relieved after the administration of the first enema, you need not be afraid to give a second, provided you are sure he exhibits no peculiar idiosyncrasy with respect to opium. In many cases of this kind you may give opium also with advantage by the mouth, and especially in combination with sudorifics.

With reference to the treatment of all cases of gout, where the disease is apt to attack internal organs, I will give you this practical hint, and let me strongly advise you to bear it in mind, whenever you may be called upon to treat gout of this nature. It is this, that these cases are of an asthenic character, and do not bear depletory measures; so that, if you find a patient labouring under gout of the stomach, or gout affecting the bladder, you must not think of applying leeches, and employing the treatment which would be applicable to other forms of inflammation of these organs; for the abstraction even

of so small a quantity of blood as would be taken by the application of a few leeches might do the patient serious mischief, and cause prostration from which he might never rally. On this point Sir Benjamin Brodie has expressed a similar opinion; for he lays it down, that antiphlogistic treatment is inapplicable to that particular form of inflammation of the bladder which is of gouty origin. With regard to the exhibition of colchicum, I am of opinion, that, in many cases, it is inadmissible, and, in all, it should be given with great caution and circumspection; for this so-called specific is certainly very depressing in its action, and therefore unsuitable to cases which partake of the asthenic character.

The treatment which, in my experience, has been most beneficial for gout, when it attacks any of the hollow viscera, consists in employing free counter-irritation, keeping up a moderate action of the bowels, paying attention to the functions of the skin, and promoting the action of this great secreting surface by the exhibition of sudorifics. Provided the urine be not alkaline, the administration of alkalies will be found of service, and opium may be employed with great advantage for allaying that irritability of the affected organ, which is often productive of great distress to the patient.

As I have before hinted, there is much resemblance between the gouty affections of the bladder and those of the stomach. In the latter organ, gout shows itself by the sudden development of violent pain referred to the epigastric region. This is always attended with the generation of gas in large quantity, which distends the organ, and it is this great distension which chiefly, if not solely, causes the pain. Another form is that in which the stomach becomes impatient of the smallest quantity of food, as the bladder is of urine. Incessant vomiting is the characteristic symptom of this form of the complaint. Sometimes these symptoms exist together, as you have lately witnessed in the case of Pyne, in the Sutherland ward.* In other instances, the muscular coat of the stomach becomes greatly weakened, and the food is pushed on only very slowly into the bowel. It accumulates in and distends the stomach, which

* *Vide* Lect. XXXII, p. 598.

gradually becomes dilated and large, and by reason of the atonic state of the organ remains so. In all the forms of the complaint, but in none more than in this last, the tendency to the generation of a large quantity of gas is a very prominent feature.

And now, to return from this long digression, into which I have been tempted by the interest of the subject, to the cases in which pus appears in the urine.

Case CXLI.—Our second case is particularly interesting, by reason of the large quantity of pus in the urine. The patient is in Lonsdale ward; her name is Mary Anne Jenkins; she is unmarried, and only twenty-five years of age. We have good notes of the case kept by my clinical clerk, Mr. J. H. Sylvester.

This patient has been for a long time passing large quantities of pus in her urine. She was herself able to detect the pus so long ago as twelve months before her admission into the hospital, but it is probable it may have existed long before she discovered it; and she informs us that, ever since, she has passed a considerable quantity each day without intermission.

All the history that can be obtained is as follows:—For the last five years she has suffered constant pain in the loins, referred especially to the region of the left kidney. This pain varied in intensity; it was generally slight and dull, but now and then severe. It does not appear, however, that at any time she suffered so much as to oblige her to desist from her usual daily occupation—that of a household servant. There have not been any symptoms of an acute attack, nor any rigors or vomiting. She never, to her knowledge, voided blood in her urine, nor did she ever pass gravel or a calculus; nor does she seem to have ever suffered from severe pain in the direction of the ureter. We could trace no evidence of local injury, and she never remembers having had any severe blow in the loins.

Rather more than twelve months before her admission into the hospital she was suddenly attacked with retention of urine, which lasted twenty-four hours; and immediately after its cessation she first began to notice in the urine a sediment,

which presented the same characters as that which is now constantly deposited. This attack of retention of urine was preceded by slight rigors, but the constitutional disturbance was altogether of so mild a character as not to cause her to lie up at all. We could not trace the history of any inflammatory attack affecting the kidneys themselves; nor could we discover any sign denoting the existence of any peculiar diathesis.

On her admission we found that the urine deposited a large amount of pus, which was estimated daily by pouring a certain quantity of the urine into a graduated measure, and allowing the pus to subside. In this way we found as much as two, three, and even four ounces of pus deposited from the urine of twenty-four hours. It is very remarkable that all this secretion of pus produced so little constitutional disturbance, that she was, from its first appearance, never even once prevented from continuing her duties as a domestic servant. So trifling were the constitutional symptoms, she did not seek for medical assistance until she observed a large quantity of pus in the urine, when she consulted her master, a medical man, who soon afterwards sent her to see me.

Upon a careful examination, I found a very large tumour situated in the region of the left kidney, and forming a considerable projection beneath the abdominal wall. This tumour, which was three times the ordinary bulk of the kidney, was elastic and yielding to the touch, and communicated the sensation of a soft elastic swelling filled with fluid, rather than that of a solid mass. There was dulness on percussion all over the surface of the tumour; the dulness, however, did not extend up to the left hypochondriac region, neither in front nor behind in the vicinity of the spleen. The surface of the tumour was smooth and round, and free from any notches or projections.

The diagnosis of a tumour, such as I have described, is, perhaps, less complicated when it is found on the right than if it occur, as in this case, on the left side. In this latter situation it is liable to be confounded with an enlarged spleen, which is by far the most common tumour found in this situation. How, then, are we to distinguish the one tumour from the other? A splenic tumour enlarges first upwards, so as to

occupy the whole of the left hypochondrium, the posterior and lateral portions of which would yield a dull sound on percussion, the stomach being pushed by it to the right side. It then increases downwards and somewhat forwards, presenting its anterior border forwards and towards the right side, which, in thin persons and children, may be readily felt and even grasped, and in which may frequently be found one or more notches, which are very characteristic of an enlarged spleen. A kidney will not enlarge in the upward direction so as to possess itself of the left hypochondrium; it extends chiefly downwards and outwards, and presents to the hand, behind the anterior abdominal wall, a broad convex surface, causing more or less bulging of the loin posteriorly. When the subject is thin, you may seize the tumour by placing your hand on the loin, and your thumb on the anterior wall of the belly, and you may thus lift it, and form some idea of its weight.

The splenic tumour is firm and solid, smooth and convex on its outer and anterior surface, with its anterior border, as I have already said, thick, prominent, and notched. No such edge can be felt in the renal tumour; and in character it may be solid or elastic, or even fluctuating, according to the nature and cause of the enlargement.

Then it is hardly necessary to point out to you that you must call to your aid in the diagnosis certain concomitant symptoms, such as the peculiar sallow complexion, and, in all probability, some history of previous or actual ague, in splenic cases, and the presence of renal symptoms when the kidney is the organ affected. Indeed, the constitutional symptoms which often accompany suppurative disease of the kidney, the rigors, and sweats, simulate ague so nearly as to be very apt to mislead even the most vigilant observer.

The tumour, in the present case, was not painful; the patient could bear it to be handled without pain, unless hard pressure were used, when she complained of a dull pain. Her most urgent symptom was an occasional cutting pain, referred to the neck of the bladder, sometimes accompanied with slight difficulty of micturition. She stated that occasionally she had a sensation of fulness in the left side, which would go off rapidly, as if something had burst, and then there would very

soon follow an increased flow of pus in the urine. There is good reason to believe that pus itself may irritate the mucous membrane of the bladder when it passes over it; and it was probably on this account that our patient always complained of a cutting pain in the region of the bladder whenever she passed an increased quantity of pus.

The pulse, in this case, never rose above 96. Now and then our patient perspired slightly, but never profusely; and, as I said before, there has been much less constitutional disturbance than we might fairly expect to be present, when we consider the amount of lesion that must exist to account for the enormous quantity of pus that this patient passes.

The largest quantity of pus that we have found in a pint of urine is four ounces and a half, and in no instance have we obtained less than an ounce and three quarters from the same quantity; so that if the patient passed two pints of urine, the enormous amount of eight ounces of pus would have been often excreted in the twenty-four hours. Generally, however, the quantity of urine passed fell below the normal standard, and often did not exceed twelve ounces.

Such, then, is the history of this very remarkable case, which is especially interesting in a clinical point of view from the coexistence of the tumour with the daily passing of so much pus in the urine.

Assuming, for the reasons which I have already specified, that the tumour is due to an enlargement of the kidney, we must next determine the source of the pus. Did it come from the bladder, or did it result from irritation from the mucous membrane of the pelvis of the kidney or ureter, by the presence of a calculus, or from any other cause? It evidently did not come from the bladder, for so large a quantity of pus could scarcely be secreted from the mucous membrane of this viscus, without extensive disease of the bladder itself; in which case, we should have expected to find a greater disturbance of the general health, and more decided symptoms referable to the bladder. A circumstance worthy of notice, in this case, is the absence of triple phosphate. I alluded to this in my last lecture, and also to the fact of that salt existing in considerable quantity in a case of inflammation of the mucous membrane

of the bladder. In the present case, the urine has been frequently and carefully examined at short intervals, but we have never been able to detect any crystals of the triple phosphate.

Did the pus come from the ureter? I think not, because symptoms are wanting to denote irritation of that duct, and also because the ureter does not afford a sufficient extent of surface to secrete so large a quantity of pus. The diagnosis, then, becomes limited either to abscess of the kidney, or to that condition of the mucous membrane of the pelvis of the kidney, to which the term "pyelitis" has been applied by Rayer. *Primâ facie*, it seems difficult to conceive how the latter affection could create a large tumour in the region of the kidney; but I think this admits of explanation.

Let me now, by simply stating my view of the case, endeavour to account for the presence of the pus in the urine, and to explain the nature of the tumour in the side. From some cause or other—probably from inflammation of the ureter—a certain amount of contraction of that duct occurred some time ago, the seat of the constriction being, probably, very near the bladder. Let us suppose a stricture formed in this situation, and consider for a moment the phenomena to which it would give rise. Fluid would, of course, accumulate above the point of stricture; and as the quantity of urine increased, it would exert a backward pressure up the ureter towards the kidney; the effect of which would be, first, to dilate the ureter; next, the pelvis of the kidney would suffer; and, lastly, the kidney itself would gradually become expanded into a large cyst, with a thick wall, in which all the elements of the secreting structure would be retained. Of such a change we had a good instance, in its early stage, in the case of Steventon, in Sutherland ward (vol. xxxiv, p. 13), in whom we made the diagnosis, during life, of the existence of a calculus in the ureter. You will remember, that at the examination of the body a calculus of the size of a good large nut was found impacted in the lower part of the left ureter; and you saw a marked dilatation of the ureter above the impacted calculus. The pelvis of the kidney was much dilated, and the calices or infundibula which embrace the papillæ were also considerably enlarged. These papillæ themselves were

flattened and compressed, and the substance of the kidney spread out so as to present an apparent enlargement of the gland to the extent of nearly a fourth of its normal size.

Now, although, in Jenkins' case, we have no evidence of the impaction of a calculus in the ureter, there may be some obstruction in that tube; and the same series of changes which I have described in Steventon's case have been taking place probably over a much longer space of time, and to a much greater extent. The kidney has become expanded into an immense thick-walled sac, and much dilated; and ultimately it has attained the size which it now possesses, occupying the space between the last ribs and the crest of the ileum. In this way we get an immense surface, capable of generating a vast quantity of pus —in fact, a pus-secreting surface. This is the way in which I think the presence of the pus in the urine, and the existence of the large tumour in the side, may be accounted for. The stricture causes a backward pressure on the kidney, which becomes sacculated in consequence, and the healthy function of the mucous membrane becomes impaired, and at length transformed in a pus-forming surface.

Case CXLII.—Many years ago I examined, for the late Mr. Guthrie, the body of a patient who died from the long continuance of disease of the kidney of this kind. There was an enormous tumour of the right kidney, which resulted from the expansion of the organ in the manner I have described into a large sac filled chiefly with pus. Imperfect septa projected into the sac, corresponding to the dilated infundibula. The ureter was nearly as large as a portion of the small intestine, and there was a stricture of it about a couple of inches above its entrance into the bladder, caused by a thickening of the walls of the duct.

Case CXLIII.—Some eight or nine years ago, I had an opportunity of seeing another case which I have no doubt was of a similar nature to this. I was requested to see a gentleman, a young man, who was suffering from a large and painful tumour in the left side. He had seen several physicians, and there was some difference of opinion as to the nature of the tumour. Upon a careful examination, I came to the conclusion that the

tumour was due to a sacculated condition of the kidney. I was very much aided in this conclusion from having examined *post mortem* the last case I mentioned. With this view of the case, and believing that the pain arose from the pus being pent up in the huge sac of the kidney, I recommend that the patient should get up, and be kept moving about as much as his strength would permit, assuming that gravitation would favour the descent of the contents of the sac; and this plan was apparently successful, for, very soon after he commenced walking about, much glairy matter appeared in the urine, and, in a few hours more, he passed a large quantity of pus. This gentleman ultimately got quite well, and the tumour in the side gradually subsided. In this case, I was able to obtain the following explanation of the formation of the tumour:—The patient had formed a great notion of the powers of nitre in the treatment of colds, and he was in the habit of taking this substance in very large quantities. These large doses of nitre at last irritated the kidneys very much; and, inasmuch as there had been probably a stricture of the left ureter, the increased secretion of urine was prevented from flowing freely into the bladder; hence a backward pressure was exerted; distension of the ureter, and ultimately of the pelvis of the kidney, occurred, leading to the formation of pus, as I just now described.

In support of this view of the case, I may state that, although this patient got quite well from the first attack, I was informed that he was imprudent enough to have recourse to nitre again some time afterwards, and that his doing so was followed by the same train of symptoms as before, by the formation of a tumour which disappeared in the same manner as on the first occasion.

Let me add a further illustration of the mode of formation of sacculated kidney from a cause within the bladder.

Case CXLIV.—James Ash, aged forty-nine (vol. xlvi, p. 142), was admitted into the hospital in August, 1855. He was suffering from pain referred to the neck of the bladder and great frequency of micturition. For seven years he had been the subject of this latter symptom, which had very much increased of late. It was attributed by him to a severe gonorrhœa which he contracted many years ago. He would pass water with pain

and scalding. The urine was very turbid, containing a considerable quantity of pus, and often blood, which came away in clots; it was slightly alkaline, of specific gravity 1006, and contained much albumen. The bladder was examined without detecting a stone; rigors followed the passing of the catheter.

Under the influence of large doses of the tincture of the sesquichloride of iron, nitric acid, and tincture of hyoscyamus, his symptoms were mitigated for a time. One or two attempts were made to wash out the bladder, but they occasioned increased irritation and a discharge of blood.

He frequently complained of great and constant pain at the end of the penis. The irritability of the bladder now became so great as to induce incontinence. He had sleepless nights, and his powers failed more rapidly than could be explained on the supposition of irritation from calculus or by the amount of discharge.

The urine now became constantly bloody, and a considerable quantity of blood was lost in this way. On the 16th of September he sank rather suddenly.

On examining the body, the right ureter was found very much dilated; the left was also dilated, but by no means to the same degree. The right kidney had a lobulated appearance externally; on being opened it was found to contain a large quantity of a purulent fluid, all the gland-tissue was compressed into a thin wall to the large cyst which contained the pus, and imperfect septa from the inner surface projected into the cavity. The left kidney was in a similar condition, although to a much less degree, and the comparison of the two organs illustrated very strikingly the gradual expansion of the solid gland into a hollow cyst, by the backward pressure of the fluid excreted from it, but encountering an obstacle to its free discharge. The nature of this obstacle was found on opening the bladder. The walls of this organ were very much thickened; in its interior a large mass of open cancer was discovered, occupying the inferior fundus and part of the posterior wall. It involved the opening of the right ureter, which was evidently obstructed by it, and the obstruction was increased by a considerable thickening of the wall of the ureter, and a consequent narrowing of its canal. The patch of cancer was hard, thick, and considerably raised

above the surrounding wall of the bladder. The orifice of the left ureter was also narrowed, and obstructed by the thickened vesical wall.

Would not abscess of the kidney explain the phenomena occurring in our patient Jenkins? The existence of abscess implies the occurrence of suppurative inflammation of the kidney, followed by the process of sloughing, and the formation of more or less pus in the cavity left by the evacuation of the slough. Such changes as these could hardly take place to such an extent as to give rise to the secretion of so large a quantity of pus, without causing considerable constitutional disturbance, much more than has existed in this patient.

The amount of constitutional disturbance in cases of this nature varies with the extent to which the gland is affected. A very small abscess may exist without much fever; but this cannot be the case where the abscess is large, or, as frequently happens, where there are several abscesses.

Not long ago, we had in the hospital a patient who exhibited the symptoms of renal abscess in their most aggravated form. Many of you may remember the case to which I allude—that of Walter Denny, in Sutherland ward, the notes of which were kept by Dr. Edward Simpson.* This patient had been suffering, for seven years prior to his admission, from frequent attacks of severe pain, accompanied with vomiting and hiccough, and irritability of the bladder. The attacks, which he was told, and not unreasonably, were due to the irritation of a calculus, subsided on his going into the country, and he remained well for four years. At the end of this time, they came on again with great severity; he had frequent rigors, and the pain was so extreme that chloroform was administered to him on more than one occasion. Under this illness he was for eighteen weeks in a hospital in London. On leaving that hospital, he had a remission of his sufferings for some months, from May, 1849, to July, 1850; the right loin, however, remaining tender. He came into this hospital on the 9th of August, with a fresh attack, and during his stay here, he suffered from paroxysms of

* See the full details of this case at p. 368, Case CI.

pain, vomiting, and hiccough, which were always accompanied with a free discharge of pus. Then the purulent discharge would cease; and then would come a remission of the symptoms, to be followed by a fresh discharge, and renewed pain, vomiting, and hiccough.

This alternate appearance and disappearance of pus in the urine, coinciding with the development and subsidence of the symptoms, led me to think that there was an abscess in the kidney, which, on becoming full, excited great pain and constitutional disturbance, until it had freely discharged itself, or that a new process of sloughing was taking place, and a new abscess being formed. Whether or not at the root of all this mischief there was a calculus, one could not positively say. It was somewhat against this view that no blood had been passed, and that no small calculi had ever been discovered in his urine; but, on the other hand, it was greatly favoured by the excessive pain, the sickness, the vomiting, and the hiccough.

This patient died exhausted by his sufferings, and we found in the right kidney a small abscess, with an inflammatory state of the ureter, the mucous membrane of which was covered with a thick layer of lymph in its whole tract. The bladder was healthy.

Case CXLV.—The case of Sarah Furnace, aged twenty-nine (vol. xxxi, B), a married woman, who was some weeks in the hospital, will exemplify the milder form of abscess in the kidney, —that is, when the constitutional symptoms are less severe.

The symptoms, in this case, began two years before her admission, with pain in the left side, of a sharp kind, loss of appetite, and fever. Soon after this, she felt a tumour in that side, and then she observed blood and matter in her urine. From this time the tumour was found to enlarge gradually, and become painful; she then had a sensation as if it burst, and the discharge passed off in the urine.

And during her stay in the hospital this was what seemed to take place. On her admission, we could detect a decided enlargement of the left kidney: then she had the sensation of bursting, and with it relief to the pain and a free discharge of pus. Then the pus would disappear from the urine, until a fresh

accumulation took place; the kidney would again enlarge and become painful, and again empty itself as before. While the kidney was thus full and painful, the febrile symptoms would be at their height, and then they would subside as the pus flowed away.

This patient did not remain more than a fortnight in the hospital; but, even in that short time, under rest, good diet, and a little quinine, her general health considerably improved.

Let me mention another case, where the abscesses were more numerous and the symptoms more severe.

Case CXLVI.—Anne Kirton, aged thirty-seven (vols. xxxiv, p. 269, and xxxvi, p. 44). The symptoms in this case began twelve months before admission by incontinence of urine, apparently from great irritability of the bladder. This symptom continued without any fresh one (to her knowledge) from February, 1851, to November of the same year. One day in this month, after travelling in a railway carriage, she noticed that her water became very thick and red. This lasted a week, and she then began to pass urine of a yellow colour, with an abundant sediment of pus.

On examining her, a large tumour was found in the right flank. Its upper extremity appeared to touch the liver, and its lower extended down to the right iliac fossa. It was convex, elastic, almost fluctuating, and communicated very much the feeling of an hydatid cyst. Large quantities of pus were almost constantly passed in the urine, in which there was little or no triple phosphate. The tumour was painful under pressure. The size of the liver could be distinctly marked out, and isolated from the tumour, the upper portion of which it overlapped. She suffered great pain in the region of the kidney and down the ureter. This woman remained many weeks in the hospital, and went out without any change of symptoms, and died two months subsequently.

The right kidney was enormously enlarged; and several abscesses were excavated in it; the pelvis was thickened.

To return to the case of Jenkins; we tried various plans of treatment, chiefly with a view to improve her general health.

She took, for some time, bark and mineral acids. We tried, likewise, gallic acid, hoping it might affect the quantity of pus; but, although her general health improved, no material diminution of the pus took place, nor did any change in her symptoms occur.

The prognosis in the case of Jenkins need not be unfavorable; the disposition to form pus may cease, and the sacculated kidney shrivel up; the long-continued pressure on the vessels and tubes of the kidney enclosed in the wall of the sac may destroy the secreting power of the organ, while the other kidney may take on itself the work of both. The pus being evacuated, and no fresh pus secreted, both the sac and the ureter may shrink up. This, as Sir B. Brodie has suggested, is the probable explanation of the shrivelled kidney and ureter, which is sometimes met with; the other kidney being large and plump. This young woman's constitution being good, and there being a free exit for the pus, we may hope that she will yet do well, as in the case I have already related.*

But were this a case of abscess in the substance of the kidney, our prognosis would be very different. Such cases are in general fatal after a longer or shorter time, according to the severity of the symptoms and the natural power of the patient; and, in dealing with them, you must be above all things careful to uphold your patient's strength, and to caution him against everything that can weaken or exhaust it. But this must be done with due regard to the power of his digestive organs, and great care taken not to disturb or derange them. There is quite sufficient reason to suppose that a small abscess may get well, or remain quiescent for many years. I am sure I know persons now living and enjoying good health who have, or have had, a cyst in the kidney, from which pus has been secreted.

I have now directed your attention to the cases most likely

* Eighteen months after this lecture was delivered, this patient presented herself at the hospital and gave the following history. On quitting the hospital, she went to Brighton, and there improved in health very much, and the purulent discharge gradually diminished. On a careful examination of the side, there was no trace of the tumour. A few pus-globules were seen in the only specimen of the urine which was examined; but whether these came from the kidney, or merely from the vagina, could not be decided.

to come before you, in which a large quantity of pus appears in the urine. They may be classified thus:

1. Cases of affection of the bladder in which the pus is secreted by its mucous membrane, as in simple cystitis, or in that caused by retained urine, or by the presence of a stone, or by some constitutional cause, as gout.

2. Cases in which the pus is secreted by the mucous membrane of the pelvis and infundibula of the kidney,—cases of pyelitis.

3. Cases in which the pus comes from the substance of the kidney itself, in consequence of the existence of abscess. To these may be added, cases in which pus comes from the ureter; but this is an affection seldom isolated from pyelitis, or from inflammation of the mucous membrane of the bladder.

LECTURE XXXI.

ON GOUT.

Gentlemen,—We have at present two interesting cases of gout under treatment in the hospital, which will furnish material for some remarks on that disease. One is a case of gout in a very common form, and which well represents the usual course of an attack; the other exhibits the disease in a very aggravated and serious aspect; and both afford good illustrations of the most interesting points in the clinical history and pathology of the malady.

Case CXLVII.—Of the former of these cases I shall speak to-day. We have a good record of it by Mr. William Browne, my clinical clerk.* It is that of William Fountain, aged fifty-four (vol. xxxi, A, p. 142), a baker by trade, who, although not very intemperate, has been in the habit of taking his full allow-

* Now of Lichfield.

ance of fermented liquor in his time; he admitted that he lives well, and that he usually drinks two or three quarts of porter a day, to say nothing of a little gin. I refer particularly to this feature in his history, because I have no doubt that it is to this habit of drinking beer freely that he owes his gout. Most persons who indulge much in beer or porter suffer sooner or later from this disease. Malt liquor is *par excellence* the pabulum of gout, for two reasons—because its chemical composition is favorable to the formation of uric acid, or some compound very closely allied to uric acid; and also because people, who are fond of beer, generally drink it in large and unmeasured quantities at their meals, and between their meals, and by taking it freely with other food they derange the primary assimilation, and thus damage all those secondary processes concerned in nutrition. This explains how it is we meet with so much more of this disease in the English hospitals than in those of other countries.

In England malt liquors are in common use among all classes of the population, high and low; they are drunk very largely by the labouring classes, especially by those of large towns, and in particular by those whose work is laborious and trying to the constitution, and who on that account receive good wages, and therefore can afford to supply the waste caused by their great exertions by means of liberal potations of a fluid at once stimulant and nutritious like beer. Hence it is that we very commonly find coalheavers, bakers (like our patient Fountain), brewers' draymen, house-painters, and others of the working classes, inmates of the London hospitals, suffering under gout in its various forms. There is another class also who can scarcely be said to labour, who are sometimes admitted for this malady; namely, butlers or house-servants of wealthy families, and coachmen.

It is remarkable that in Ireland and Scotland gout is a disease almost entirely confined to the better classes of society, and that it is rarely, if ever, seen in hospital practice. This is because beer is but little used by the lower orders in those countries. In such towns as Dublin and Edinburgh there must be a large portion of the working classes well paid, and therefore well fed; but porridge, potatoes, fish, and bread, are the chief sources of their sustenance, meat less frequently, while

whisky is their stimulant; and these kinds of food do not generate gout unless taken in large quantities, and in habits already tainted with the disease by inheritance.

For the same reason, in France and Germany (excepting in the beer-producing Bavaria) gout is by no means common; and the meager accounts to be found of this disease in foreign works plainly show that the authors cannot have had much practical acquaintance with its characters.

I need not remind you that gout is one of those maladies which are pre-eminently hereditary; that a son will inherit it from his father or mother, and, what is more curious, from his grandfather or grandmother, the intervening generation being free, or nearly so. Such *inherited* gout is frequently most obstinate and difficult to eradicate, the gouty diathesis being more fully developed than when it has simply been acquired. It is, therefore, well to inquire into this point in the history of such patients as come before you. In the present instance, fortunately for the curability of the disease, and unfortunately for the *respectability* of our patient's gout, we cannot make out any pedigree,—it appears to owe its origin to that ignoble source, *beer*.

Fountain's first attack of gout came on ten years ago—that is to say, it did not develop itself in any paroxysm until that time, although he had often suffered from flying pains about the joints, indicating the development of the gouty diathesis,—so that he had reached the age of forty-four years before he had his first attack. This first attack came on suddenly: having gone to bed well, he was awoke in the middle of the night with violent pain in the right great toe, followed in a few hours by redness and swelling; from this he recovered in a few days, and his second attack did not occur for another year; this was more severe than the first, and affected the foot as well as the toe. His present attack came on about five days before his admission; the patient having suffered from headache, giddiness, and nausea, for a few days previously.

This history is highly deserving your attention. It is just that which we get in a large proportion of the cases of gout,—the patient being, in point of age, about or beyond the meridian of forty, and accustomed to the daily use of beer to a greater or less extent, without anything of what might be called excess

but habitually taking a regular quantum of malt liquor. Under these circumstances a man becomes dyspeptic; as he advances in years he becomes less active in his habits of exercise; he suffers, perhaps, from headache and from flatulence, and the bowels act irregularly; his complexion acquires a sallow hue, there is yellowness of the conjunctivæ, and at the same time the urine is not secreted in its usual quantity; it becomes high-coloured, and prone to precipitate uric acid or urates. With all this there may or may not be a little local uneasiness, a little stiffness or discomfort about the great toe or instep, hardly amounting to pain, which perhaps the patient may disregard at the time, but still sufficient to attract his attention to the part.

A patient suffering under these symptoms is, undoubtedly, in most instances, if he be a man of gouty habit, or if he inherit the disease, threatened with an attack of gout; yet, if you tell him so, he will not believe you,—perhaps he will laugh at the idea that *he* is to have the gout; but in the course of the night, or towards morning, he awakes up suddenly, suffering from violent pain in the great toe or some other part of the foot. At first there is no redness whatever,—merely pain, which soon becomes accompanied with a sensation of throbbing and enlargement about the joint; the pain becomes more severe, so that the patient dreads even the contact of the bed-clothes. This continues for some hours, or even for some days; then the acute pain diminishes, the redness and swelling subside, but the joint remains stiff for some time, in consequence of the thickening of the tendons and ligaments about it. This thickening is caused by a deposit in the fibrous structures about the joint, probably consisting of urate of soda, the same salt as that which enters into the composition of chalk-stones, with, perhaps, also, some phosphate and carbonate of lime. The more frequent the attacks of gout, the more the joint suffers; because each succeeding attack leaves a little deposit. The joints appear large and stiff, and the cartilage, as well as the ligaments, and of course the synovial membrane, undergo a change in structure. In certain cases, a thin layer of urate of soda, looking very much like a layer of plaster of paris smeared over the cartilage, may be observed.

An interval of nine years elapsed between our patient's first and

econd attack—a very remarkable circumstance, as in general, fter the first invasion, the attacks recur once or even twice an- ually, generally in spring or autumn. This second attack took lace only twelve months ago. Like the first, it was characterised y a sudden invasion of pain in the great toe and side of the foot, he patient having previously suffered from headache and a feel- ng of nausea for some few days. The present attack came on nly a few days before his admission.

Now you may fairly ask, why do you call these attacks gout, nd not rheumatism? Rheumatism attacks joints, and causes welling and great pain: how are we to distinguish between the heumatic and the gouty attack? The points which would lead ou in this case to the conclusion that the disease is gout are hese:—the age of the patient—his diathesis—his habitual use f malt liquor—the rapid or sudden way in which the attacks ame on—and the fact that the parts first affected were the mall joints, and especially that these were the metatarso-pha- ngeal joint of the great toe and the tarsal joints, which are re-eminently the favorite habitat of gout. Rheumatism is a isease which, generally speaking, affects early life; while gout usually met with in persons of middle or advanced age. The hances of acute rheumatic affections are very much diminished fter the age of forty; while gout most frequently occurs at and fter this age.

This patient had none of those very profuse sour-smelling veats so frequently met with in rheumatic fever, and which are ighly characteristic of that disease. You may, perhaps, have oticed a girl in Augusta ward suffering from this malady; and though only two joints are affected, and those very slightly, et she has this profuse sweating as a most prominent symptom. his tendency to sweating is never altogether absent in rheu- atic fever; it is a more essential feature of this disease than ie articular affection. It is not uncommon, indeed, to meet ith instances of fever with profuse sweating and furred tongue, st as in rheumatic fever, without any articular affection, but rhaps with inflammation of some internal organ. In this way neumonia and pleurisy, but more commonly the former, often ow themselves. The symptoms are fever, with a full bound- g pulse and a furred tongue, copious sweats, pneumonia or

pleurisy, or both: and there may or may not be articular affection; sometimes the joints do not become affected till towards the end of the fever. In cases of gout, more especially when the gout is pretty general, and affects large joints as well as small—the *rheumatic gout* of some authors, and the *synovial rheumatism* of others—you may have sweats, but rarely the very profuse sweats of the true rheumatic fever.

I have referred to the suddenness of the invasion as characteristic of gout. This is a very remarkable feature of this remarkable disease. The invasion is, no doubt, in nearly every instance, to all intents and purposes, sudden to the patient; but, on a careful analysis of many of the cases, it will be found to be more apparent than real, and to arise from an extremely rapid development of a disease which had been insidiously creeping on for a longer or shorter time before. On close inquiry, circumstances will be brought to the patient's recollection tending to show that some disturbance of his system, or of one or more of his joints, may have previously existed. Practically, however, the invasion is sudden: a man goes to bed thinking himself well; he had been walking about for a great part of the previous day; in the middle of the night, or in the early morning, he wakes up with the severe pain of an attack of gout. Now this does not occur in articular rheumatism: never, so far as I know, does the rheumatic attack come on otherwise than gradually—first in one or two joints,—those of the lower limbs generally, then in those of the upper extremities.

And it is not an unimportant diagnostic guide to observe which are the joints first affected. In gout, in the vast majority of instances, those first visited are the great toe or the instep, or some other *small* joint. In rheumatism the ankles and knees are generally the first attacked. It is certainly one of the most striking distinguishing characters between gout and rheumatism, that the former fixes upon the small joints, the latter upon the large. But no practical man would allow that either may lay claim to the one or the other class of articulations as its exclusive habitat: what is not inaptly called *rheumatic gout*—but which, I think, may be more correctly designated *general gout*—attacks all the joints, even to those of the lower jaw. And so also in rheumatism, even in rheumatic fever, all the

joints may be attacked, even to those between the articular processes of the vertebræ. Thus it is that Nature, even in her abnormalities, throws difficulties in the way of our attempts at classification and definition, just as she does with reference to the objects of study of the zoologist or botanist.

But it is to be feared that some of you will say, what a difficult matter it must be to decide what is gout and what is rheumatism! I think, however, I can promise you that you will not make this remark when you have really studied and watched many of the cases. You will then see that the mode of invasion, the age of the patient, the absence or presence of profuse sweats, the history of previous attacks, and especially of the first, will generally serve as useful guides to lead you to a satisfactory diagnosis.

Dr. Garrod has made out a positive physical character of gout, which may be regarded as surely diagnostic of that disease from rheumatism. It consists in the discovery of uric acid in the blood-serum or the blister-serum. And his process is ingenious, and so simple that any one may use the test, however little accustomed to chemical manipulation. A little serum is put into a watch-glass, and to it are added five or ten drops of acetic acid. In this acidulated serum a small skein of worsted is laid, and the watch-glass is set aside under cover to protect it from dust. After a few hours, the crystals of uric acid, if it exist, will be found adhering to the threads.

There is one feature of difference between the two diseases, of very great interest as regards both diagnosis and prognosis, upon which I have not yet touched: I allude to the liability in each to affection of the heart. This liability is greater and much more serious in rheumatism than in gout. In rheumatic fever you frequently have, as you well know, most exquisite examples of both pericarditis and endocarditis, so that a fearfully large proportion of the cases emerge from the fever with a damaged heart, in the form of an adherent pericardium or an imperfect mitral valve, or both. In gout, these acute affections are among the rarest occurrences; yet, that the heart is liable to be *slowly* damaged, both in its muscular structure and in its fibrous tissue, all experience proves; and the well-known fact of the liability to irregularities in the heart's rhythm, which is

so common with gouty patients, shows a marked proneness to cardiac complications in gout. What seems to me to be the prominent distinction between the cardiac affections of the two diseases is this—that in rheumatism there is a proneness to fibrinous concretions on the pericardium or the endocardium; in gout, the nutrition of the muscular structure suffers, and inorganic deposits (urate of soda and phosphate and carbonate of lime) take place in the chordæ tendineæ and other parts of the fibrous tissue of the heart, which stiffen them and cause them to shrink, and ultimately impair the efficiency of the valvular apparatus.

In both rheumatism and gout a tendency of the disease to shift from one place to another has long been recognised by practitioners; and hence the cardiac inflammations of rheumatic fever used to be regarded as examples of *metastasis* from external to internal parts. This tendency to shift is most signally noticed in rheumatic fever, when particular joints seem to be invaded in succession by the rheumatic state; and then, indeed, there is much the appearance of a true metastasis,—to-day, for example, the left knee is affected, to-morrow the right knee, while the left knee has become quite or nearly well. But there is no metastasis in the cardiac affection, for the external rheumatism may remain unchanged, or may even become more severe, while the heart-disease is at its height; and not unfrequently the cardiac disease comes on first, and the articular affection subsequently.

In gout there is, on the whole, less tendency than in rheumatic fever for the disease to shift *from one joint to another joint;* but in this disease we have the most remarkable examples of true metastasis from an external to an internal part, or *vice versâ*. A patient may have had at your visit yesterday well-marked gout in his instep, and at your visit to-day you find the instep nearly well, but he is suffering exquisite pain in the stomach. Or the disease may first show itself in some internal organ, and then attack one or more joints. Thus, a gouty subject gets bronchitis or irritable bladder, and you find you make no progress in your treatment until suddenly the gout appears in the great toe or instep, when, all at once, the internal affection gives way.

Nothing like this occurs, at least so far as I know, in rheumatic fever.

I will not dwell longer now upon this interesting feature of gout, because I shall have to allude to it again in connexion with the second of the cases which form the text of this lecture; but shall content myself with stating—and you will excuse me if, for the sake of brevity, I do so somewhat dogmatically—that it is when rheumatism or gout is of the low or asthenic kind that this tendency to shift is most marked. And it is also most apt to occur when the patient has become very weak under antiphlogistic treatment. Of this an interesting, and to me conclusive, proof is afforded by the results of my own practice. Formerly, when I used to bleed largely and purge freely, I found this tendency of gout to shift much more common than of late years, when I have, to a great extent, abandoned the so-called antiphlogistic treatment as worse than useless in these diseases. I would impress upon you this dogma,—an active antiphlogistic treatment creates asthenia—asthenia gives to both rheumatic fever and gout what I may call *the shifting character*, which in both diseases is most perilous, but in the latter especially so; and when you find the tendency to shift already existing in a case, depend upon it that the asthenic condition of the patient is that which demands your earliest attention.

Treatment.—I have referred you to this case of Fountain's as a good example of gout in a common form, and now let me briefly allude to the treatment to which he was subjected in illustration of the plan which you will generally find most serviceable to your patients.

But let me, *in limine*, entreat of you to bear in mind, as an important feature of the clinical history of gout (very necessary to be kept in view in our attempts to form an estimate of the value of this or that plan of treatment), that the great majority of cases such as Fountain's—cases of sthenic gout, in men of good constitution—will get well without any very specific treatment. Suppose you take a hundred cases of gout, if you confine them to bed and keep them warm, and especially keep the affected joint warm, take away their beer, and give them light, wholesome, and nourishing diet, you will find that

of these hundred cases perhaps seventy will get well, without any unfavorable symptoms, in from three or four days to a fortnight.

Well, then comes the question can we accelerate the cure by treatment? I believe that by moderate purgation, and by the use of diaphoretics, by keeping the joints warm, and, if the urine be very acid, by the administration of alkalies, we may expedite convalescence, and undoubtedly relieve pain. The best way to keep the joint warm is by enveloping it in a large quantity of carded cotton or cotton wool, and covering the whole with oiled silk: you thus place the joint in a kind of local vapour-bath, which causes free sweating of the skin around it, and likewise promotes a general diaphoresis. It has this advantage over a general vapour-bath,—that you can more exactly limit its influence, and that it does not tend, unless carried too far and kept on too long, to weaken the patient. A general vapour-bath, although highly plausible in theory, is practically very objectionable, because, whilst you may hit off with tolerable exactness the precise amount of sweating desirable in one case, you will overshoot the mark in half a dozen.

A great advantage of this local treatment is, that in nine cases out of ten it relieves pain, and that pretty quickly. As soon as the joint has become thoroughly warm, and the sweating process is fairly established, relief is generally experienced.

I have satisfied myself by repeated trials that counter-irritation over the affected joint is of decided utility in many cases both in relieving pain and removing the effusions or thickenings which remain in gouty joints. This may be effected by the local application of mustard, or turpentine, or naphtha; but what I prefer, and use most frequently, is a *small* blister. It is important that the blister should be small,—large ones increase the articular irritation; they may vary in size, according to the size of the joint, from that of a sixpence to that of a half-crown, or, at the very largest, a crown-piece. You will often find it decidedly beneficial to apply mustard or turpentine for twenty minutes or half an hour before the blister is put on, the effects of which are thereby accelerated, and the quantity of serous discharge increased. You need not be deterred from pursuing this plan of treatment, even in the earliest stages, when the joint is

most red, and looks most inflamed and excited; it succeeds admirably, provided always that you take care not to irritate or vesicate too large a surface.

The application of leeches to a gouty joint is, in my opinion, much to be deprecated. It is difficult to say why this should be so, but I have so often seen joints weakened by this practice that I have no hesitation in condemning it. There is no doubt that if you apply leeches to gouty joints you will relieve the pain pretty quickly, but you will leave a state of permanent weakness, from which the patient will be a long time in recovering. It will be for you to decide which is preferable,—to try and relieve pain quickly by a method which is at best uncertain, but which is pretty sure to leave a weakened joint; or to adopt a method less rapid as regards the relief of pain, but more sure as to its ultimate effects. I confess I prefer the slower and surer method. As regards the blistering plan, I can very confidently state that I have never seen any bad effects from it when the blisters have been confined within the limits of size which I have mentioned.

Many will say to you, you must use colchicum, and they will think you very unorthodox if you attempt the cure of either rheumatism or gout, but especially the latter, without the use of this drug. They hold, or act as if they held, that colchicum exerts a special influence over the morbid matter or other cause which gives rise to an attack of gout,—that it kills it, as it were, and bears the same relation to the gouty poison that quinine does to the paludal poison, or iodide of potassium to that of secondary syphilis. That colchicum bears some curious relation to gout, I am quite prepared to admit, but I believe that this relation is one sometimes for good, and sometimes for evil; and you will, perhaps, be startled when I tell you that it appears to me that in the majority of cases this relation is of the latter kind. I have no doubt that in sthenic cases in young subjects colchicum relieves pain, and hastens the removal of the paroxysm: but, at the same time, experience leads me to subscribe to a belief very popular among gouty patients, that if it shortens the duration of the attack, it likewise shortens the interval between the attacks. There is great danger of patients getting into the habit of taking colchicum in large and even in increasing doses,

much as they would opium. Colchicum is one of those drugs of which the system gets very tolerant; and if, in a first attack, the patient take ten minims, in the second he will require twenty, in the third more, and so on,—just as an opium-eater requires continually increasing doses of his favorite drug. Indeed, you find confirmed colchicum-drinkers just as you find confirmed opium-eaters. I once attended a lady of high rank who had gradually accustomed herself to doses of the wine of colchicum, which were measured, not by tens or twenties, but by hundreds of minims; and yet, such was the little influence of these large doses upon the essence of the disease, that her attacks became more and more frequent, her joints were horribly crippled, and her nervous system was fearfully shattered. I endeavoured to persuade her to leave off the colchicum, but without success; and she ultimately died in a state of extreme prostration,—due, as I believe, mainly to the inveterate addiction to this drug.*

For these reasons I object to the too prevalent routine practice of giving colchicum in gout, and prefer trying to cure the paroxysm without it. Those of you who constantly follow my practice know that I very rarely have recourse to it, and that my patients get well as quickly, and, I believe, more certainly, than if they had taken that drug. When I use it, it is generally in ten or fifteen-minim doses of the wine of the root, or in single grain doses of the acetic extract.

Fountain took small doses of colchicum wine immediately on his admission, but without any very encouraging result. The gout, certainly, became much less severe in his feet; but, while he was still taking the colchicum, he was attacked with gout in his right knee. This not appearing to yield, the colchicum was discontinued, alkalies were administered, a blister was applied, and an occasional aperient was given.

The remedy which seemed to act with the most marked benefit was the blistering. On the 17th, the great toe, and dorsum of the foot on both sides, were extremely painful and swollen; and on the 18th, after the blisters had been applied, the pain had greatly subsided, and the swelling was much diminished;

* See also Case CXXXVI, Lecture XXVIII, p. 548.

on the 22d he was so much better that he was able to walk along the wards; on the 26th, however, while he was still taking colchicum, the right knee became painful and swollen.

Now we observed here a point which is worth your attention in reference to prognosis—namely, that the tongue remained more or less coated so long as the general gouty condition had not materially abated. The feet had recovered, but the knee became affected, and the tongue continued foul throughout; and when the attack in the knee came on, the tongue acquired an accession of fur, nor did it become clean for several days afterwards; and we found that, as the tongue became clean, the convalescence advanced *pari passû*.

Thus, if you watch the tongue, you will find in it the best index to the increase or diminution of the constitutional disturbance which accompanies the gouty paroxysm. If it remain foul, your patient is not safe, even although the local symptoms may have wholly or in part subsided; if it continue clean, you may conclude that matters are going right.

Under a treatment by alkalies, occasional blisters, and aperients, this patient got quite well, and left the hospital on the 18th of December, one month from the date of his admission.

The next case to which I propose to call your attention is remarkable for the enormous deposits of urate of soda about the joints, and other interesting symptoms; but as I have already occupied a good deal of time with one case, I must make the other the subject of my next lecture.

LECTURE XXXII.

ON ASTHENIC GOUT.

The second case of gout, gentlemen, to which I briefly referred in my last lecture, affords many points which deserve your most attentive consideration. It is not only an excellent example of a form of disease in which the generation and deposition of urate or lithate of soda takes place in enormous quantities, but it also affords a good illustration of the symptoms which indicate that the gouty poison is attacking the stomach and the bronchial tubes. I do not know that I could select a better example of the asthenic and the erratic form of gout.

Case CXLVIII.—The patient to whom I allude is William Pyne, who has been for some weeks in Sutherland ward (vol. xxxii, 1850, p. 31). His age is forty-three, and he is a carter by trade. It does not appear that he has been at all an intemperate man in his habits, but he has been accustomed to take his daily allowance of beer, and it is not improbable that at times he indulged freely in that liquid to help him on in the labours of the day.

This man's first attack of gout occurred so long ago as nine years, when he was only thirty-four years of age; and it was followed by a second six months afterwards. Since that time he has not been free from an attack of gout for three months together. These attacks were, however, slight, and did not cause him to lie up for more than two or three days each time: possibly they were treated by colchicum: the gouty paroxysm was perhaps "knocked down" (as it is often said to be) by that medicine; however, this knocking down did not prevent the frequent recurrence of the attacks, nor did it alter a character of the disease which was conspicuous in this patient from the first, namely, a tendency to shift from one joint to another.

But during the last five years the paroxysms have been much longer in duration than they previously were, generally lasting as long as five or six weeks.

It was in one of these long attacks, four years ago, that a symptom showed itself which is characteristic of the peculiar form of gout to which this man is subject. Small deposits were observed growing beneath the skin of the ear, and about the knuckles of both hands. These deposits consisted of urate of soda. They have increased in size and number with each subsequent paroxysm.

In November, 1848, he was a patient in the hospital for another attack. On this occasion a very large collection of the chalky deposit formed on the back of the right hand; it was opened, and a great quantity of a white semi-liquid matter escaped, looking like wet plaster of Paris. This, on examination, proved to be urate of soda. In this attack other symptoms occurred of great interest, and marking this particular form of gout. He had, for instance, a severe attack of bronchitis, upon which the ordinary remedies seemed to exercise little or no influence, and which did not yield until after gout appeared in his feet. Soon after this he suffered from excruciating pain in the stomach and from vomiting, with great flatulent distension of the stomach and bowels.

All these symptoms, however, yielded to the treatment pursued, and he left the hospital much improved in health, after a sojourn in it of some seven weeks, and continued free from any serious return of the gout until the 23d of September of this year (1850), when he was admitted for an attack of the disease affecting the little finger of the right hand and the elbow of the same side. These parts were very red and much swollen, and extremely painful. The development of gout in them was preceded by a fit of shivering, and by severe pain in the stomach and vomiting. His tongue was coated with a thick fur, and his pulse was 108.

The gouty inflammation spread quickly to the other fingers and to the whole hand, and an abscess formed in the little finger, from which, when opened, a large quantity of pus mixed with urate of soda escaped. The sickness increased, so that he vomited everything almost as soon as it was taken.

Under the use of small quantities of stimulants, and ammonia in effervescence, the ammonia being slightly in excess, these symptoms subsided; the fingers ulcerated and burst, and urate of soda was discharged in considerable abundance.

On the 30th of October, however, the sickness returned; the left hand was attacked by gout, the right continuing much inflamed and discharging, and a new symptom showed itself in some difficulty of breathing, with a very general rhonchus over the whole chest. Soon afterwards both feet and knees became affected with gout, and copious effusions were formed in the knee-joints.

On the 3d of November, the hands and arms presented a most formidable appearance, of which I thought it well to preserve a record in the drawing which I now show you. Both hands were enlarged to nearly double their natural size, and the skin over them was red and tense; a deposit of urate of soda had formed over every joint, and in several places there were small collections discharging freely both pus and urate of soda. The swelling extended over the forearms, and a considerable collection of matter formed near the inner condyle of the right arm.

It was observed at this time that some albumen existed in the urine, which varied in quantity, being greatest when the constitutional disturbance was most intense, and decreasing with the diminution of the febrile excitement.

For several days abscess after abscess formed, each containing pus and urate of soda. These were opened in succession; and while this was going on, it was found necessary to administer stimulants very freely, which, by being given in small doses at short but regular intervals, he was enabled to bear, and the irritability of his stomach was thereby greatly relieved.

After all the abscesses had been freely opened, and the urate of soda discharged in large quantities, the fingers became much reduced in size, and the general constitutional disturbance quickly subsided. The catarrhal sounds disappeared, the vomiting ceased, the appetite returned, and he was enabled to eat a little solid food. The joints all improved, and he soon began to walk about the ward. The condition of his fingers became much better than before his admission, for the great masses

of urate of soda were removed; and although the fingers were stiff, they could be placed close together, and were much more serviceable than previously.

The first point that we may notice in this case is the accumulations of urate of soda. These accumulations, you observe, took place about the small joints of the upper extremities; and this is almost always the case. Although the lower extremities do not wholly escape, still the great accumulation is generally in the upper ones, and the quantity found in the lower ones is usually comparatively small.

The deposit takes place in greatest abundance in the subcutaneous tissue. I show you here a drawing of a large collection of it under the skin of the elbow. It will accumulate in the areolæ of the areolar tissue; in some instances it forms a hard dense mass intersected by the bands of fibres of that tissue, but in others these bands of fibres seem to be absorbed, and a cavity is formed filled wholly with this material, from which it may be easily dislodged when the skin is freely opened. But at the same time it is often found in joints, seeming to smear the articular surfaces of the bones, and making them rough, so that, as I have often pointed out to you in Pyne's and other cases, they grate against each other; and it will make its way into the interstices of the fibres of ligaments and tendons, and stiffen them. A very common place for these deposits is in the ear, beneath the skin covering the cartilages; and it sometimes occurs over the cartilages of the alæ nasi. Sometimes the water with which the deposit is mingled, and which gives it its soft pasty character, becomes absorbed, and a dry chalk-stone is formed which will leave a mark on a black board. Here is one of these concretions as large as a marble, removed from one of Pyne's knuckles.

I would ask you to bear in mind another curious point connected with these deposits: it is this, that they occur *early* in the disease. This man was under forty when they came on, and he had not been more than five years the subject of fits of gout before these accumulations had taken place to a considerable extent. This fact, which I have observed in several other cases, seems to me to indicate that the disease in which such

accumulations occur is a special clinical form of gout. You will see many instances of patients having had paroyxsm after paroxysm of gout for a long series of years without anything like such an accumulation: joints may be damaged, cartilages altered, ligaments and tendons stiffened, but the areolar tissue will be free from any collections of urate of soda. But in such cases as that of Pyne each fresh paroxysm is accompanied with a new deposit, or a greater or less addition to those already existing. Again, in the more ordinary forms of gout, it is the lower extremities which suffer most, and to which the greatest mischief is done; but in this the upper extremities are the seat of the most abundant deposits, while the lower extremities by no means escape unscathed.

I do not suppose that the inflammation, which in this case affected the upper extremities, reaching above the elbow, was purely of the gouty character; because we found that it ended in the formation of numerous and considerable collections of pus. The tendency of the gouty inflammation is not to form pus, but (and especially in this particular form of it) rather to generate and eliminate urate of soda. A case of erysipelas occurred in the ward shortly after this patient was admitted, and I fear he imbibed some of the erysipelatous poison. The tendency of the erysipelatous inflammation is, as you well know, in a remarkable degree to generate pus. Thus we had in the same subject two orders of inflammation, each tending to generate a different product,—the one the urate of soda, the other pus; and accordingly the collections which formed at various points were found, on being opened, to contain both these products mixed together. In the ordinary attacks of this form of gout the newly-deposited urate of soda is mixed with a thin whey-like fluid, which contains some pus-cells, and which may therefore be regarded as sero-purulent.

We had a good opportunity in this case of witnessing the clinical phenomena which accompany gout when it attacks the stomach or bronchial tubes, or when it irritates the kidneys.

The symptoms, which indicated that the stomach was attacked, were the severe and incessant vomiting, the pain in the region of the stomach, which at times was agonising, and the tympanitic distension of the organ. When the stomach is

affected with gout, its mucous membrane appears to become highly irritable, and it secretes gas with great rapidity, which distends the organ. It is this great inflation of the stomach which probably (at least in great part) causes the violent pain which patients suffer under the attack; for when they are able to expel wind in considerable quantity, the pain becomes much less, or entirely disappears. But the ability to expel wind by the efforts of the muscular coat of the stomach is impaired, the power of that coat being weakened partly by the distension and partly by the influence of the gouty poison; and this very weakening of the muscular coat allows the organ to become unduly distended. Vomiting, or the expulsion of matters from the stomach, must in such cases as these be effected mainly, if not wholly, by the abdominal muscles.

There was here no metastasis, but the stomach was attacked simultaneously with the other parts. But you may often see the selfsame symptoms, as regards the stomach, follow the sudden suppression of external gout, or precede the development of it in the foot or some other part.

It is difficult to determine what tissue of the stomach is especially the seat of the disease. It seems most probable that the mucous membrane is primarily, and the muscular coat secondarily, affected. Post-mortem examination gives us no aid in determining this question; for the changes which we may fairly suppose are caused by the attack of gout disappear at death, and no trace of previous disease remains beyond a more or less dilated and flaccid state of the organ; and this dilatation will be the greater as the attacks have been more frequent.

This case showed us likewise how the bronchial tubes become affected in gout. While the hands and forearms, and other external parts, were still suffering, the breathing became more frequent, catarrhal sounds were heard all over the chest, and a troublesome cough came on, accompanied with frothy expectoration. The very same symptoms will often be found to precede the external development of gout, or to follow its recession from some external part, just as in the stomach affection.

Lastly, we observed in this patient evidence of renal irritation, due obviously to the gouty state of the whole system. This was found in the presence of albumen in the urine, which varied

in quantity in proportion to the degree of febrile disturbance. But the albumen, although it diminished when the patient became convalescent, did not wholly disappear; whence I infer that the kidneys must have been damaged to some extent,—probably they were in an early stage of that contracted state to which, when occurring in gouty subjects, I have given the name of "the gouty kidney."*

It is worthy of your notice that much the same derangements of internal organs which are liable to occur in gout, occur also in erysipelas; and, on the supposition that this patient had imbibed some of the erysipelatous poison, these derangements may have not improbably been increased by the combined influence of the two poisons, tending to create similar disturbances in the system. Thus the poison of erysipelas, when first imbibed, will occasion severe vomiting,—a symptom often very difficult to deal with, and sometimes causing a fatal issue to the case. So also erysipelas will cause bronchitis, which sometimes precedes, sometimes follows, any external manifestation of the disease upon the skin.

Treatment.—Let me, in conclusion, briefly review the treatment to which this patient was subjected, and at the same time refer to that which seems most applicable to the generality of cases of this form of gout.

Very early in the management of this case we had to deal with the irritable state of the stomach, and the severe pain which was referred to this organ. The remedies on which I relied for the alleviation of these symptoms were opium, the application of free counter-irritation over the epigastric region by mustard and by turpentine, and the exhibition of the sesquicarbonate of ammonia in effervescence, taking care to allow three or four grains of the ammonia to remain in excess at each dose. The opium was given at night in the shape of morphia in a night draught: it was well borne, and of signal service. I preferred giving it in one dose at night rather than in repeated doses through the day, in order to obtain sleep at the natural time. For two or three nights it was combined with a couple of grains

* See Lecture XXVIII.

of the acetic extract of colchicum, but this was very soon given up, as it seemed to depress the patient. The effervescing ammonia was exhibited frequently through the day, as often as every two or three hours; and when the erysipelatous state was at its height, I was glad to add to each dose fifteen minims of the chloric ether—a very grateful and valuable stimulant.

At the same time we found it necessary to give brandy in small and frequent doses, and nothing seemed to remain upon the stomach better than this. At first he took two drachms every hour, but it was afterwards found necessary to increase it to half an ounce. You will remember that we obtained the most obvious proof of the necessity for this kind of treatment by diminishing his allowance of brandy one day when he seemed a little improved in strength, from half an ounce per hour down to two drachms. After he had been twenty-four hours on this diminished allowance, we found him very much reduced in strength, his tongue parched and dry, and his pulse quickened; but these symptoms soon disappeared on putting him again on the increased allowance; and this time we gave him two drachms every half hour instead of half an ounce every hour,—a mode of giving stimulants in maladies of a low kind, fever, erysipelas, &c., which you have many opportunities of seeing here attended with the happiest results.

After three days of this treatment the irritability and pain of the stomach had completely yielded; but, on account of the erysipelatous state and the general depression, I judged it advisable to continue it for twelve or fourteen days, and especially when the bronchitis came on. We combated this latter affection likewise by free counter-irritation, with turpentine stupes to the chest, both in front and behind, and afterwards by a blister to the sternum.

On the 21st of November, finding all these more urgent symptoms much improved, I reduced the quantity of brandy down to four ounces in the day, and gave him bark in small quantities (five minims of Battley's liquor cinchonæ), and allowed him a little animal food, still continuing an opiate at night, to relieve an irritable cough. He now improved rapidly, and on the 9th of December was able to get out of bed, and sit up for a considerable portion of the day.

It may seem to you somewhat anomalous that the treatment of a gouty state of stomach should consist in the administration of stimulants like ammonia and brandy, the intemperate use of which, according to popular belief, tends rather to generate the gouty condition. If, you would argue, there be any state of inflammation of the mucous membrane of the stomach at all like the external inflammation which we have witnessed about the joints of his upper extremities, surely the application of such hot things as ammonia and brandy must do harm. So it would seem reasonable enough, *à priori*, to assume; but in the practice of medicine our reasoning must not be *à priori*,—we must appeal to experience; and that appeal will elicit an unequivocal verdict in favour of this plan of treatment for pure gout in the stomach, and still more if there be, as in Pyne's case there seems to have been, some complication with erysipelas. Nor is this without its analogy; for in many forms of conjunctivitis—and amongst them in that which is associated with a gouty or rheumatic state—you will find great benefit from the application of tincture of opium and of a solution of the nitrate of silver to the eye.

After Pyne had become convalescent, we found his kidneys not secreting sufficiently, and, in consequence, I ordered him to take an ounce of lemon-juice three times a day. This was followed by an immediate increase in the quantity of urine secreted from a pint to more than three pints in the day; and he continued to pass water at this rate during the rest of his stay in the hospital,—not, however, free from a small quantity of albumen.

This, indeed, is the chief value of lemon-juice in rheumatic and gouty affections: it increases the quantity of the urine often in a very marked way, and it is agreeable to the taste of the patient, and more easily taken than other diuretics. It tends, therefore, to promote elimination, which is the great end of treatment in these affections; and directing that, as it does, through the kidneys, it does not pull down or depress the patient in any injurious way. But I do not hold that it possesses any specific virtue over these maladies; nor do I consider it prudent to rely on it alone in the acute forms: still I think the profession is much indebted to my friend, Dr. Owen Rees, for having

called attention to the use of this remedy in these diseases, and for having sanctioned by his high reputation its employment in opposition to prejudices which we all naturally felt against giving so much vegetable acid in maladies of which a prominent feature was the highly acid state of certain secretions.

I had been in the habit of using lemon-juice as a diuretic in dropsies of all kinds long before Dr. Rees introduced it into notice as an anti-rheumatic remedy, and in some instances with very marked benefit. I shall not readily forget one case of universal cardiac dropsy in which the use of this remedy, after a trial of most of the other diuretics, cleared off the dropsy in a few days.

You saw that we opened freely the various collections of pus immediately they were formed, and also the collections of urate of soda. There can be no second opinion as to the propriety of evacuating by mechanical means collections of pus; but as to the accumulations of urate of soda it may be a question whether it is safe or worth while to interfere with them. Now with reference to this point, you must bear this in mind,—that these collections may be small or large; the small ones generally form in positions where there is a mechanical impediment to the accumulation of the inorganic material to any very great extent; the large ones collect where the skin is more or less loosely connected with the subjacent parts. The large ones, when allowed to increase beyond a certain point, create inflammation and ulceration of the skin, and burst. In many of these cases you may, I think, save your patient a tedious process of this kind, by freely incising the skin, evacuating the collection, and using precautions afterwards—such as poulticing, fomenting—to guard against inflammation following the incision.

Very often you will succeed in completely evacuating these collections by this treatment without any untoward consequence; but sometimes the incision is followed by a good deal of troublesome inflammation, and even by an attack of gout. These consequences are, however, more apt to ensue where the urate of soda has not been completely removed, as when the accumulation is formed in the areolæ of the areolar tissue, and not in a single cavity. Hence it is more prudent not to inter-

fere with the deposit unless you can be satisfied that it is contained in a separate cavity, or in two or three large cavities, which communicate freely with each other.

By the evacuation of several accumulations in this way, Pyne's hands are now in a very much better state than before his admission, and he can use them much more freely; and it will depend on the degree of improvement which his constitution will experience under a further treatment, chiefly by regimen, whether fresh deposits will take place or not.

LECTURE XXXIII.

ON PARALYSIS.

PARALYSIS FROM LEAD POISONING—HYSTERICAL PARALYSIS.

Gentlemen,—I beg to-day to call your attention to the subject of paralysis. There are at present five cases in the hospital, which exemplify different forms of palsy; I shall, thus, be able to illustrate my observations on these diseases by reference to cases which have been under our immediate inspection.

When I use the word paralysis simply, you will understand that I mean the loss of the power of *motion*. There is a paralysis of *sensation*, as well as a paralysis of *motion*. They often occur together; but the latter generally predominates. Sometimes at the commencement of an attack they will be conjoined, but the paralysis of sensation usually disappears speedily, leaving only the paralysis of motion. Again, either may occur without the other; that which most frequently occurs alone is paralysis of motion, and that which has the readiest power of recovery is the paralysis of sensation.

Let me make some general observations on the conditions which give rise to and attend paralysis. I must ask you to

receive my statements on these points as so many *postulates*; for it would occupy too much time to enter into the proofs which could be adduced to demonstrate the correctness of my propositions.

In the first place, then, you must not look upon paralysis as a disease of itself: it is not a disease, but a symptom of a disease. Non-medical people, and sometimes even medical men, are apt to speak as if the palsy constituted the whole essence of the malady; but this is not the case. Paralysis is an effect due to a cause, which cause itself is not always the essential disease.

What are the causes which may give rise to paralysis? They are either an affection of the nerve or nerves, whose power is destroyed, in some part of their course, or a morbid state of the centre in which the nerve or nerves are implanted, or with which they may be less directly connected. The nervous trunks themselves may be impaired in their nutrition, the centre being healthy, or they may have suffered some mechanical injury from violence or pressure; thus they become either imperfect conductors of the nervous force, or altogether incapable of propagating it: or some portion of the centre of volition is the seat of a morbid process, whereby the influence of the will over certain parts is suspended; the nerves of those parts, consequently, receive no impulse from that centre, whether mental or physical; and although healthy in themselves, are incapable of taking part in voluntary acts.

Whatever interferes materially with the conducting power of nerve-fibre, or the generating power of nerve-vesicles (gray matter), will constitute a paralysing lesion. Thus, in the first place, poisoning of the nervous matter will operate in this way. Soak a portion of the nerve of a living animal in chloroform, or ether, or opium, and it will fail to propagate the nervous force as long as the influence of the poison lasts. In a similar way; the poison of lead in the living system may paralyse, by weakening the conducting or generating power of the nervous matter. Poisons formed in the living system may operate in the same way; such as retained urinary or biliary principles, or the poison of rheumatism and gout. Secondly, any morbid process which greatly impairs the natural structure of nerve-matter will

paralyse. Inflammation will do this; so also will atrophy, or wasting from want of sufficient supplies of nutrient matter, as when the flow of blood is lessened or cut off. The opposite conditions of hardening and of softening of the nervous matter become paralysing lesions for the same reason, that they greatly impair or destroy the nerve-structure. Thirdly, a solution of continuity of nerve-fibre will paralyse. Cut a nerve across, and you have immediate palsy of the parts which the nerve supplies below the section. Solution of continuity from a melting down or rupture of the fibres is, I have no doubt, the frequent cause of sudden paralysis in cases of softening, or in cases of sanguineous effusions. Fourthly, pressure on a nerve or nervous centre will paralyse. Of this we have many proofs as regards nerves; a nerve, for instance, included in a ligature, or compressed by a tumour, is paralysed thereby. A fracture of the skull with depressed bone will paralyse if the brain be sufficiently compressed; an apoplectic clot on the exterior of the brain paralyses by compression; so also a tumour in its substance. I would say that the centre of volition is of very great extent: it reaches from the corpora striata in the brain down the entire length of the anterior horns of the gray matter of the spinal cord, and includes the locus niger in the crus cerebri, and much of the vesicular matter of the mesocephale and of the medulla oblongata. Disease of any part of this centre is capable of producing paralysis; but as the intra-cranial portion of it exercises the greatest and most extended influence in the production of voluntary movements, so disease of this portion gives rise to the most extended and complete paralysis.

Another fact which I would impress upon you is one which anatomy in a great degree demonstrates, and which pathological research confirms—that the centre of volition for either side of the body is not altogether on the same side of the body. Of the centre for the left side of the body, for instance, the intra-cranial portion is on the right side, and the intra-spinal portion on the left side, and these two portions are brought into connexion with each other through certain oblique fibres from the anterior pyramidal columns of the medulla oblongata, which cross from right to left, decussating with similar fibres

proceeding from left to right, which belong to the centre of volition for the right side of the body.

Having made these introductory observations, I will now pass on to the consideration of the cases; and the first we shall take is that of a man in Sutherland ward, as affording a good example of a very serious form of paralysis, of common occurrence in the London hospitals—I mean paralysis from the poison of lead.

Case CXLIX.—The patient, J. Halliday (vol. xxi, p. 120), is thirty years of age, by occupation a house-painter, of temperate habits. It appears that he has never been obliged to desist from work on account of illness until about three years ago, when he had an attack of colic, for which he was treated in an hospital in town, and perfectly recovered. He has since had several slight attacks. Three weeks ago he first noticed that his wrists became weak, and began to drop, and that he became very nervous and irritable. About this time, or rather later, he had two paroxysms of general convulsions, fits of epilepsy, during which he suddenly fell down, lost his consciousness, and struggled violently, but he did not bite his tongue. These attacks occurred once daily on two succeeding days, came on without any warning, and each lasted about ten minutes. He has frequently had cramps in the arms and legs, but no other pain in the limbs. For some weeks past he has noticed a blue line on his gums: bowels generally confined.

I have on many occasions pointed out to you the remarkable and peculiar condition of this man's arms. When they are held out, the hands drop, from his inability to maintain them in the state of extension; nor can he, by the utmost effort, bring them into the state of extension. His power of extending the fingers is also impaired, but to a less degree. If you examine the posterior surface of the fore-arm where the extensor muscles are situated, you will find that space rendered quite concave, from the atrophy and consequent shrinking of the muscles. The fore-arm has lost its plumpness in this region, and, by pressure, you can feel the inter-osseous membrane. These are not the only muscles affected: those of the ball of the thumb are also wasted, and the movements of the

thumb are much weakened, especially those of opposition. But, in this case, the wasting of these thumb-muscles has by no means gone to so great an extent as you may often find in other severe cases. The flexor muscles of the fore-arms have suffered slightly in their nutrition, and have lost much of their firmness; their power is consequently much affected; and, although the patient can bend his wrists with sufficient power, he cannot grasp with full force. The general movements of the arm are accompanied with that kind of tremulousness which so frequently accompanies enfeebled states of nutrition of the muscles. The deltoid muscles are so paralysed that the patient has no power to raise his arm or maintain it at right angles with the trunk. The lower extremities are not paralysed, but they participate in the general weakness.

In addition to the symptoms above detailed, we find in this patient that curious sign of the presence of lead in the system first pointed out by Dr. Burton—namely, the blue line on the margin of the gums, present only where the teeth or their stumps are in the alveoli, and ceasing where a tooth is wanting. There is no indication of any special lesion of the central organs of the nervous system, although these organs cannot be regarded as sound; the digestive organs are natural, as are also those of circulation and respiration; the pulse is about 70, and feeble; and the secretions healthy.

It is not very common, in lead-palsy, to see the muscles above the elbow so much weakened as in this case; not only were the biceps and triceps thus affected, but the deltoid was so much paralysed that the man could scarcely raise his arm, much less extend it at right angles to his body. He still has, although some time under treatment, a symptom which was much more obvious at first—namely, a trembling, agitated manner, like that of an intemperate man in a state of incipient delirium tremens. It is not improbable that this, to a certain extent, arose from intemperance; for, although he did not call himself intemperate, he was fond of his glass; and intemperance is a very common vice among those of his trade. I believe, however, that it mainly depended upon a general diffusion of the lead poison through his muscular and nervous systems. Again, you will remember that he had epilepsy, and evidently in con-

nexion with the same causes which produced paralysis; the fits were slight, but still they were distinctly epileptic; they had all the essential characters of that disease: there was the sudden fall, the loss of consciousness, the convulsion. He has had, moreover, cramps in the arms and legs.

The question here arises—What is the particular tissue or organ affected in the paralysis of house-painters and others exposed to the influence of lead? I believe that the muscles and nerves are early affected, and that, at a subsequent period, the nervous centres become implicated. The muscles are contaminated, and their nerves participate in this contamination. The nervous system is therefore affected, first, at the periphery, in the nerves; and the poisoning influence continuing, the contamination gradually advances to the centres, as is sufficiently shown by the fact that the local paralysis always precedes, often for a considerable time, the epileptic convulsions or other symptoms of centric disease. In this case the epilepsy showed itself unusually early.

Another question suggests itself to us here—What is it that thus contaminates the muscular and nervous tissues, and impairs their functions? To this we answer, without hesitation, it is lead, existing in the affected tissues. If you examine the gums of patients suffering from lead-palsy, you will perceive a blue line on, or rather in, the gum, close to the neck of each tooth; and this is caused by lead existing there, perhaps as a sulphuret; you may produce precisely the same effect by the administration of acetate of lead, just as you may excite salivation by mercury. The most positive evidence, however, is afforded by the post-mortem examination of patients who have died from lead-poisoning; for from their muscles and brains chemists are able to extract lead in notable quantity.

In hospital practice, house-painters, whose employment leads them to use lead in large quantity, are those whom we have most frequently to treat for this malady. These men get the lead paint upon their skin, where it may become absorbed, or inhale it into their lungs in the form of small particles of the powder with which the paint is made, floating in the air;* or it

* I have heard house-painters say that that which does them most harm is the

may be mixed with the saliva, and so get into the stomach. From one or all of these sources the lead gets into the circulation, and during its course through the body becomes deposited in the affected organs, or combined with their constituents in some way or other.

But why, it will be asked, does it alight upon the muscular and nervous tissues chiefly? why upon the muscles of the extremities, rather than those of the trunk? and why upon the extensor muscles in preference to the flexors? The answer which appears to me most satisfactory, and which offers the best explanation of these phenomena, is this—that those tissues in which the nutrient changes are most active receive the largest proportional supply of blood, and that blood, being loaded by a poisonous material, would impregnate them with it to a greater degree than other tissues in which the circulation is less active;* that, for this reason, such highly-nourished structures as muscle and nerve become poisoned early; that, as the muscles of the upper extremities are used more, and probably on that account experience more active nutrient changes, than those of the trunk and lower extremities, the former are poisoned first. Moreover, in painters, the extensor muscles of the arm, as well as the muscles constituting the ball of the thumb, become principally paralysed, because they are most exercised during the practice of painting; and as they are

paint called *flatting*, which is largely intermixed with turpentine, which, passing off in vapour, forms a ready medium for the inhalation of lead particles.

* If we suppose that the activity of nutrition is equal in all tissues, then the supply of blood to particular tissues would be regulated by the bulk of the tissue—that is to say, each tissue would receive a supply of blood exactly adjusted to its size. We know, however, that the activity of the nutrient changes varies very much in the different tissues—those of muscle and nerve being highest in the scale. But all muscles and all nerves are not equally active in their nutrition; those whose function is most active and energetic doubtless exhibit the greatest amount of nutrient change, and draw most largely on the circulating fluid. And, in like manner, it may be said of those muscles which are symmetrical, that if those of the right side are more used than those of the left, the former would attract more blood than the latter; and any poisonous matter which may exist in the blood, and may have an affinity for the muscular or nervous tissue, will be drawn to that side in greater quantity than to the other, and will therefore affect those muscles to a greater extent than their fellows of the other side.

more exercised, are consequently more supplied with blood—poisoned blood—to repair the waste that is going on in them.

Patients who *die* of lead-poisoning are generally persons who have been exposed to its influence for a long time, or have addicted themselves to intemperate habits. The morbid appearances in the brain and spinal cord denote imperfect or depraved nutrition of those centres, and are frequently associated with marks of chronic irritation of the membranes, such as accompany intemperance; these changes may be doubtless also due to the presence of lead. The brain especially presents the appearance of an ill-nourished organ: pale, soft; its convolutions wasted; the sulci between them wide; and sometimes patches of white softening are found in the hemispheres. I have seen this condition in patients who have experienced several paroxysms of epilepsy before death, or who have died in one, and in whose brains lead has been detected.

Treatment.—In the treatment of lead-palsy, the great object is, if possible, to eliminate the poison from the body, and to prevent the introduction of further supplies of it.

The patient should be kept clean, should wash much, and use such means as friction, exercise, &c., to stimulate the excreting power of the skin.

It has been thought that sulphur, when introduced into the system, has the power of neutralizing the effects of lead, by forming some innocuous compound with it; whether or not any such compound is formed I cannot say, but I have certainly found sulphur a very useful remedy in the form of a sulphur-bath. The bath which I usually employ consists of from two to four ounces of sulphuret of potassium, mixed with from twenty to thirty gallons of water. I use this empirically; and I am quite sure the patients derive much benefit from its employment.

Iodide of potassium, as we learn from the experiments of Melsens, promotes the elimination of lead, and may be used with advantage in these cases, and in large doses. I often give along with it the citrate of iron. Frequently patients suffering from lead contamination are much benefited by the use of iron in some form or other. Sandras recommends the use of the hydrated per-sulphuret of iron, made by the

addition of an alkaline sulphuret to a solution of a persalt of iron.*

Galvanism, as a local stimulant to the nerves, should not be neglected: I am certain it is of service. Our patient Halliday was much improved by it; and to its use I mainly attribute the recovery of the power of moving his deltoid muscle, which he has now attained.

In the use of galvanism, you must take care not to continue its application too long each time. Half-an-hour each day, or still better, ten minutes or fifteen minutes at three different periods of the day, will be found quite sufficient.

Added to this, the subject of lead-palsy should breathe pure air, and have good sustaining food.

Case CL (vol. ii, p. 111).—The next case, gentlemen, is one of paralysis of the arm produced by a bandage improperly applied to a man who had suffered fracture of the clavicle; and I hope that from it you will not only learn an important lesson in pathology—namely, that pressure on a nerve is capable of producing paralysis of the parts supplied by it, and likewise the particular treatment which paralysis so produced requires; but that you will also deduce a moral from it—that a surgeon cannot be too careful in watching cases that are under his care, and in noticing every symptom which may indicate that his patient is not progressing favorably; for had that been done in the present instance, this man would not have come to our hospital with paralysis of his arm.

The patient who is the subject of this case has been in the hospital before under my care, for some pectoral complaint, when a full report of his history, his former health, and habits, was taken; the notes made at his admission for his present illness are therefore brief, although sufficient for the purpose: I will read them to you.

"Timothy Sullivan, admitted into Rose ward, November 18th. This patient is twenty-three years of age; has lived in

* Bouchardat, 'Annuaire de Thérapeutique,' 1844, and Sandras, 'Maladies Nerveuses,' 1851. For the formula of the syrup, see Bouchardat's 'Nouveau Formulaire,' p. 282. Paris, 1851.

London for about a year; by occupation, a labourer. Last June he was admitted into this hospital, suffering from pain in the side, and cough; both these symptoms were relieved, and he went out. Shortly after leaving the house, his right clavicle was broken by an old wall falling upon him. He went to an hospital, and the ordinary figure-of-eight bandage was applied. After a time, the patient found that his right hand and arm were numb; and soon after this, he noticed a great loss of power of the extensors of the hand. Notwithstanding these symptoms, the bandage was allowed to remain, and both the paralysis of sensation and muscular motion have continued up to the present time.

"Nov. 19th.—At present there are numbness of the hand and arm, and entire loss of power in the extensors of the hand, which is completely flexed when the arm is raised. All the muscles of the arm have less power than natural."

This case was treated with galvanism, and the patient left the hospital better, having gained some power of the extensors, and that of the flexors being nearly restored to its healthy state. It was some time, however, before the improvement became manifest. In the reports of the 22d and 26th of November, it is stated that no change had taken place;—he first began to mend on the 28th.

Paralysis produced by pressure on the axillary plexus of nerves is not of uncommon occurrence. I have seen some cases in which it was produced in the following way:—A man gets intoxicated, and falls asleep with his arm over the back of a chair; his sleep under the influence of his potations is so heavy, that he is not roused by any feelings of pain or uneasiness, and when at length he awakes, perhaps at the expiration of some hours, he finds the arm benumbed and paralysed. It generally happens that the sensibility is restored after a short time, but the palsy of motion continues: cases of this kind sometimes derive benefit from galvanism; but if the pressure, which caused the paralysis, has been very long continued, they seldom come to a favorable termination. Nerve-tissue is one which never regenerates quickly, and seldom completely, so that any great or long-continued lesion of its structure is not likely to be removed.

I shall next call your attention to a case illustrative of another form of paralysis—namely, hysterical paralysis.

Case CLI (vol. xxi, p. 170).—The following is the report of the case:—Mary Leigh, aged forty-two years, was admitted into Lonsdale ward on October 28th, 1847: states that she is a native of, and has resided all her lifetime in, London; her occupation was that of a housemaid for twelve years, when she was married; has been a widow for seven years; had an attack of rheumatic fever when she was fifteen years of age, and a second about nine years ago; three years since she had erysipelas in the left ankle; and twelve months back she suffered from typhus fever.

About six weeks ago she worked hard for several days in succession, during which time she suffered from headache; and going to bed tired on a Saturday night, fell asleep almost immediately. About five o'clock on the following morning, she woke up with pain and loss of power in the right arm. For this she applied to a druggist, who purged her and gave her a liniment for the arm. About three weeks after this, she became an out-patient at this hospital: took mineral and saline purgatives for a fortnight, when the leg also became affected like the arm, with pain and loss of power; she also suffered from pain in the head, and dimness of sight.

In this case the most important points to be remembered are these:—In the first place, the invasion was sudden, and occurred after hard work; and it was not accompanied by any loss of sensibility, nor impairment of intellect. The face is quite free from paralysis; and this, considering the extent of the paralysis elsewhere, is a remarkable circumstance. I was, at first, however, disposed to think that there was a small amount of facial paralysis; but I am now quite sure that such is not the case, and that what I took for palsy is nothing more than that want of symmetry on the opposite sides of her countenance, which the majority of people present. Examine the faces of a number of persons collected together, as I, with a numerous class before me, have now an opportunity of doing, and I will venture to say that, without any disparagement to the good expression of the countenance, you will find but few which exhibit

perfect symmetry. The tongue too, at first sight, appeared to be paralysed; but we soon discovered that the obliquity in the direction in which the tongue was protruded was due to a cause which will be very apt to mislead you if you are not prepared for it,—namely, an undue projection of two or more of the teeth in the lower jaw, which gave an oblique course to the movement of the tongue.

Our patient exhibits no certain sign of brain disease; all the symptoms under which she labours may have occurred independently of disease of that organ. There was no injury, no impairment of intellect; the function of deglutition was unimpaired; there was no tongue or face paralysis; pain of the head there was, but this was not fixed in its position. All this militates against the idea that these symptoms were caused by any lesion of the brain. What, then, it may be asked, did cause them? We know that there are certain conditions of the system—*hysterical*—in which organic diseases are simulated by mere functional disturbance, and that even the most grave affections are occasionally imitated with great accuracy, and among these paralysis. Hysterical paralysis, however, generally affects only one limb, or a portion of one limb, as a joint or a finger.

The case of Mary Leigh, which we have just been considering, I believe to be one of hysterical paralysis in its least common form, being far more general than usual, and nearly amounting to hemiplegia. Added to the negative evidence which I have already adduced, there is much positive evidence to show that the malady is an hysterical affection; the patient's physiognomy is hysterical, as well as her general constitution; the catamenia have been irregular; she has had decided globus hystericus, and is in the habit of voiding large quantities of very pale urine of low specific gravity. Again, the great extent of the paralysis in the limbs, and the total absence of it in the face and tongue, are certainly evidence in favour of its hysterical character; for although hysterical paralysis occurs in all parts of the trunk and extremities, it very rarely, if ever, attacks the face. But I would particularly call your attention to the peculiar character of the movement of the paralysed leg when the patient walks, which, in my opinion, is characteristic

at all impaired, indicating that the *portio dura* of the seventh pair of nerves is untouched. In general, in cases of hemiplegic paralysis, the tongue deviates to the paralysed side. This case was an apparent, though not a real, exception to this rule; for the tongue deviated to the sound side. On careful examination, however, it was found that certain projecting teeth in the lower jaw diverted the course of the tongue from its ordinary channel into a deviation to the right side; and the fact illustrates the remarks which I made on this subject in my last lecture.

Now the points, in this case, which served for the foundation of a diagnosis were, first, the existence of pain; next, the occurrence of paralysis on the opposite side to the pain; and, lastly, the irregular movements of the eyeballs, and the double vision.

The existence of fixed pain in the head in general indicates intracranial irritation. Pain in the head may be situate in the course of some of the nerves of the scalp, over the brow, or across the forehead, or in the temple, or spreading upon the parietal or occipital bone, or at the vertex. Pain in any of these situations may shift, or intermit, or it may give the sensation of a nail being driven into the head—*the clavus hystericus*. When pain exhibits such characters as these, it is not, in general, indicative of any mischief going on within the skull, but is rather symptomatic of deranged digestion, or of some constitutional disturbance, or of a hysterical or hypochondriacal state, or it is the result of debility or exhaustion; but where the pain, whether sharp and burning, or dull and heavy, is fixed in its situation, as in this case, and varies only in intensity, and not in locality, it may generally be referred to intracranial irritation, such as probably would arise from disease of the membranes, or of some superficial parts of the brain. Disease of the corpus striatum, or of the optic thalamus, does not usually produce pain, which is distinctly referable to a particular spot. When disease of these parts occurs, it either causes no pain at all, or a dull, heavy pain, more or less diffused; unless, indeed, the pia mater in connexion with them be extensively diseased. If the dura mater, or the arachnoid, or the pia mater, become the seat of the disease, then pain is produced, and the patient refers it to a position which very nearly corresponds to the site of the morbid lesion; hence such pain as our patient suffers may be looked

upon as indicating rather a superficial than a deep-seated lesion.

Another important symptom under which this man laboured was dimness of vision, which also assumed the form of double vision. This symptom, although it sometimes occurs independently of cerebral lesion, ought, nevertheless, always to excite the suspicion of such lesion, and more especially if there be at the same time any affection of the muscles of the eyeball.

The paralysis in this case is of that kind which generally depends on cerebral lesion, its one-sided character denoting a cerebral rather than a spinal origin; at the same time, you must bear in mind, as I pointed out in my last lecture, that a similar form of paralysis may take place, as the result of hysteria, where there is no appreciable lesion at all. In this case there is little reason to suspect hysterical paralysis, because the face is affected, and because the mode of moving the leg is essentially different from that of the hysterical palsy; the patient is also of the male sex, which is not very liable to these hysterical affections.

The parts of the brain, the lesion of which is most apt to produce hemiplegia, are the corpus striatum and the optic thalamus, and the most frequent lesions of them are softening, a clot, or abscess. It is remarkable that lesion of the optic thalamus should produce nearly, or precisely, the same effects as lesion of the corpus striatum. This is probably explained by the intimate union of the two bodies, so that neither can be affected without the other suffering more or less the morbid influence; but if the optic thalamus be the part diseased, the corpus striatum will suffer more in consequence than the optic thalamus would, if the corpus striatum were the seat of lesion, because of the great size and extensive connexions of the former, and the smaller size and more limited connexions of the latter. Disease also in the immediate vicinity of these parts will cause paralysis; but if the lesion be situated quite near the surface of either hemisphere of the brain, and be not of such a nature as to produce pressure, there will be no paralysis.

A clot, or an abscess, or a tumour, in the middle of the centrum ovale, will not produce paralysis if it do not cause pres-

sure, or interfere materially with any of the fibres of the corpus striatum.

Another condition capable of producing hemiplegia is inflammatory or other disease of the membranes of the brain. The dura mater cannot suffer long from inflammatory disease without implicating the arachnoid or pia mater. When you get inflammation of these membranes, you have effusion of lymph or of pus, which, as it increases, causes pressure on the surface of the brain, which is then extended to the corpus striatum and optic thalamus, and thence results the paralysis.

If some of the deeper-seated parts, such as the crura cerebri, are affected, we also have paralysis; because the crura cerebri, as the bond of union between the corpora striata and spinal cord, form a part of the great centre of volition. Disease of the cerebellum or its crura, provided it be deep seated, will also produce hemiplegia; this is probably due to the connexion which is formed between the hemispheres of the cerebellum and the fibres of the pyramids in the pons Varolii.

Now, in the case of Hardwick, the first symptoms were those of irritation, producing convulsive movements of the right side; and these were followed by incomplete paralysis of the limbs. This slow accession of the paralysis, following symptoms of irritation, gives us some clue to the nature of the exciting lesion. These phenomena are precisely such as one would expect, where the lesion consisted in inflammation of the membranes of the brain, accompanied by effusion of lymph. In the first stages of the inflammatory affection you would have irritation, and consequently convulsive movements; and in a later stage, where the lymph came to be effused, we should have pressure and paralysis; but as the pressure was not excited immediately, but only indirectly, upon the centre of volition, the paralysis would be incomplete.

A very interesting and important feature in the paralysis in this case is the accompanying spastic or rigid state of the muscles. This rigidity, according to my experience, if it supervene *early* in the paralytic seizure, or simultaneously with the paralysis, indicates irritative disease within the cranium. It is not uncommon, however, to meet with cases in which there has been very complete paralysis, with perfect resolution of the

muscles; but after a time these muscles slowly become rigid, the fingers become flexed, and sometimes firmly pressed against the palm of the hand, the hand bent upon the forearm, and the forearm upon the arm, with a tense and spastic, although wasted, condition of the muscles. This *late* form of muscular rigidity you must carefully distinguish from the *early* one, inasmuch as the former indicates that there has been loss of substance in the brain, and that the cicatrix is undergoing contraction.*

You will meet, in practice, four different conditions of the muscles in paralytic limbs in different cases. The first differs scarcely at all from that of the healthy muscles; the muscles exhibit, perhaps, less firmness, and are less excitable by the galvanic stimulus, when the paralysing lesion is not of an irritative kind. A second condition presents complete relaxation of the muscles: they are soft, imperfectly nourished, and waste with wonderful rapidity; so that under a paralysis of a few days' duration the size of the limb experiences a very marked diminution. In these muscles there is very little excitability to the galvanic stimulus—sometimes almost none. This is the most complete condition of paralysis, in the strict sense of that term, and it is sometimes accompanied with phenomena which denote a depressed state of the general nutrition of the limb: the pulse in the large arteries of that side is weaker; there is sometimes more or less of œdema, especially if the limb be kept in a dependent position; and the heat of the limb is imperfectly maintained. Some of these cases get well; others continue paralysed, although the general health of the patient improves, and the muscles become wasted to mere membranes; others, again, continue paralysed, but the muscles gradually assume a condition—the third to which I wish to call your attention—of contraction and rigidity, the flexor muscles always exhibiting this state to a greater degree than the extensors. The muscles are still wasted, but they are stretched like tense cords between their origins and insertions. The biceps in the arm, and the hamstring muscles in the thigh, project beneath the skin like tense membranes. This condition is due to a chronic shortening

* Vide infra, Lecture XLIII and XLIV.

of the muscles themselves: they are tense, but not firm nor plump; it is undoubtedly a form of muscular atrophy, of which a contracted and rigid state is a prominent feature. A fourth condition is illustrated by our present case. The muscles suffer very little, or not at all, in their nutrition; they are either constantly firm and rigid, or become so on the slightest movement of the limb; the paralysis is seldom complete. In these cases there is more or less of an exaltation of nutrition;—the circulation in the limb is vigorous, and its heat is not below the standard of the other limb; and it is frequently more excitable by galvanism than the corresponding muscles on the other side.

I must beg your particular attention to these various states in which the muscles of paralytic limbs are found. You may draw practical inferences from them of great value in treatment: when the condition of rigidity is present early, your patient will bear local bleeding or local counter-irritation, or both; and may derive benefit from these measures, provided other symptoms do not contra-indicate them. The state of complete relaxation affords no indication for antiphlogistic treatment, but on the contrary, in many of the cases in which it occurs, it should be regarded as pointing out the propriety of an opposite mode of treatment. As to that condition in which the muscles assume the contracted state gradually, and some time after the paralytic seizure, I wish much it were in my power to suggest some means of arresting it. Some slight benefit is gained by subjecting the limb to frequent extension at stated periods in the day: this, I believe, will retard the contraction, so long as it is diligently persisted in; but when it has been laid aside, the contraction will go on just as if the extension had never been employed. The case is analogous to that of stricture in the urethra, or of the cicatrix after a burn, which exhibits a remarkable tendency to contract, requiring in the former case the long-continued use of the bougie, and in many instances its frequent employment throughout the entire life of the patient. In both instances, indeed, I believe I am correct in saying that surgeons have hitherto failed in finding any means to check effectually the tendency to contraction.

I may add, that long-continued and forcible extension of the limb gives rise to considerable pain when the muscles are in a

state of chronic contraction—pain so severe that the patient cannot bear extension for any length of time.

But to recur to the case of Hardwick. From the various symptoms I have detailed to you, I have been led to the following diagnosis in this case—namely, that the lesion is of an inflammatory kind, and that it is principally and primarily meningeal; so far I can speak without hesitation: but in determining the precise locality, more difficulty is experienced; I have no doubt, however, that it is so situated as to affect the optic and third pair of nerves; and from the position to which the man has always referred the pain, namely, to the left parietal bone, I should assign as its locality the dura mater, and the other membranes in the vicinity of the anterior and inferior angle of that bone; thence the disease has extended perhaps along the fissure of Sylvius, and thus it has come to involve the optic and third pair of nerves. It must be obvious to you, however, that the disease might readily have been set up first in the pia mater, and may have involved the origin of these nerves through some other parts of the brain, producing precisely the same train of symptoms.

The treatment adopted in this case has been chiefly counter-irritation to the scalp, by tartar emetic ointment, and the use of mercury. These remedies have produced no good result; the patient's intelligence and memory are becoming affected, and I fear that the hemispheres of the brain are getting involved, either by extension of disease or by pressure. It is not improbable that ere long we shall have the opportunity of ascertaining how far the diagnosis is correct or otherwise.*

Case CLIII (vol. xxii, p. 103).—The second case is that of Catherine Williams, aged, as she stated, fifty, but looking at least sixty-five: she had been long addicted to habits of intemperance. This woman stated that she had been suffering from pain in the head for four months, and also from pain in her limbs: the pain in the head was not at all fixed, it was accompanied by drowsiness. She was a thin, pale, ill-nourished woman, and looked like one who drank more than she ate.

The week before her admission she complained of severe pain

* Vide infra, Lecture XXXV.

and numbness in the left hand and arm: this was probably of the nature of a subjective sensation, due to an affection of the nerves at their central extremity, and not at their periphery. Affections of this kind not unfrequently depend upon disease of the brain; sometimes, however, they are confined to the trunk of the nerve, and are strictly of a neuralgic character. In this case, however, the concomitant headache and drowsiness pointed to a cerebral affection.

Previous to her present attack coming on, it appears that she had been working very hard, and drinking in proportion. One morning, whilst at breakfast, she suddenly lost the use of the left arm and side of the face, and, on attempting to get up, fell to the ground: in the evening, the leg on the same side became paralysed; the paralytic seizure was not accompanied either by stertor or by loss of consciousness. On admission (Nov. 15, 1847) two days after this seizure, we found this woman completely hemiplegic on the left side, with the most perfect resolution of all the muscles; the facial palsy was also complete, and there was deviation of the tongue to the left side. The muscles of the paralysed limbs were wasted, and there was a slightly œdematous state. On examining the heart, we found a loud systolic bellows sound, indicative of an imperfect action of the mitral valve, allowing of regurgitation through the mitral orifice; affording one of many examples of the association of cardiac with cerebral disease. From her habits and age, it seems probable that the mitral disease is due to atheromatous deposits on or in the valve, or to shortening of the chordæ tendinæ; similar deposits will also probably be found in the arteries of other parts of the body, and in those of the brain affecting the vessels perhaps on one side more than those on the other.

In this case the paralysis seems justly referable to disease of the brain; the paralysis has all the characters of that produced by such lesion. The lesion is not meningeal, because there are no symptoms of irritation, and because the paralysis supervened suddenly, and was complete. We must look for the cause of it in the substance of the brain, and I should be led to locate it either in, or in the immediate vicinity of, the corpus striatum or the optic thalamus, as these are the parts most frequently affected in such cases, and as lesion of both or of either

of them, but especially of the corpus striatum, gives rise to the most complete paralysis.

When the attack took place, there was no stertor or loss of consciousness: this shows that the paralysing lesion, whatever it may have been, caused no pressure on the brain, nor any great shock to that organ. The lesion, therefore, did not arise probably from effused blood, but rather from some degeneration of the cerebral matter itself—such as white softening; and this is a form of lesion which very frequently occurs in a subject so ill nourished as our patient, whose blood is poor, many of whose arteries are undoubtedly in a diseased condition, and whose heart, from the extensive regurgitant disease of the mitral valve, is not capable of supplying the brain with its due amount of blood.

My diagnosis of this case, therefore, is, that there has been white softening of the brain, situated at the parts which I have already named; this softening has probably existed for some time without any distinct symptoms, when suddenly some of the fibres giving way, paralysis followed with equal suddenness. It is neither impossible nor unlikely that some small clots (not of sufficient size to produce pressure) may exist in the white softened substance, produced by rupture of minute vessels.

The case has been treated upon this view of its nature. Indeed, the constitutional state of the patient afforded no indication for any other mode of treatment but that which would contribute to support and uphold. There is, however, but very little hope than any mode of treatment will be permanently beneficial, the whole nutrient function of the patient seems so seriously impaired, and it appears very unlikely that her vital powers will long enable her to struggle against the distressing influence of the cerebral disease.

I shall conclude this lecture, by calling your attention to the influence of electricity on the paralysed limbs, in each of the cases which I have narrated.

Most of you have frequently witnessed the trials with electricity made on these patients, and can bear me out in the statements I shall make. I may first, however, call to your recollection the doctrine of Dr. Marshall Hall, that when the influence of the brain upon a limb has been withdrawn, the irri-

tability of the muscles of that limb becomes considerably augmented, and that, therefore, in hemiplegic paralysis, the muscles of the paralysed limb are more excitable by the galvanic stimulus than those of the sound limb. The results of my experiments have led me to a somewhat different conclusion from that of Dr. Hall; and I would refer you to an account of these experiments published in the thirtieth and also in the thirty-sixth volumes of the 'Medico-Chirurgical Transactions.' If, however, I have ventured to express a difference of opinion from Dr. M. Hall, I can truly say that I have no wish to treat with disrespect any views which he may have put forward; but I cannot shrink from stating what I believe to be the truth, even though it be at variance with previously received opinions, however eminent the authority by which they may be sanctioned.

My experiments led me to arrange cases of hemiplegic paralysis in three classes, according to the manner in which the electrical stimulus affects the paralytic limbs. In the *first* class, to which belongs the vast majority of the cases, the paralytic limb was acted upon by electricity very slightly or not at all, and in every instance to a less degree than the sound limb. In the *second* class, no perceptible difference existed as to the effects of electricity on the two limbs: these were cases of recent paralysis, the cause of which was not of a very depressing nature. In the *third* class, the electricity produced a greater effect on the paralysed limb than on the sound limb; the difference, however, was never very great, and such cases are not numerous: in nearly all such the paralysis was accompanied by recent rigidity of the muscles.

Now, of the two cases which we have been describing, it was found that, in the man Hardwick, electricity produced more effect on the paralysed limbs than on the sound limbs; and in this case you will recollect there is muscular rigidity. After the patient had been some time in the hospital, the paralysis became more complete, and the muscles less rigid, and, in the same proportion, their excitability to the galvanic stimulus also diminished.

In the second case—the woman Williams—electricity produced scarcely any contractions in the paralysed limbs, whilst

it caused distinct but somewhat feeble contractions in the sound ones; and you will remember that we applied electricity in this case, not only by the electro-magnetic machine, but also by the simple galvanic trough, and that with each instrument the same results were obtained.

The conclusions, at which I have arrived upon this subject, are, that when the paralysed limbs exhibit an early spastic or rigid state of the muscles, as in the case of Hardwick, they will be more excitable by electricity than the sound limbs; but if the paralysis be accompanied by a state of complete resolution of the muscles, the sound limb is most excitable to the galvanic stimulus, and the paralysed limb is sometimes scarcely at all to be excited. In the latter case, the nerves of the paralytic limb are in a depressed condition; in the former they are in an irritated condition; and the different effects of electricity in the two cases will depend on the difference of cause of the paralysis. If the paralysing lesion be irritative, the paralytic limb will be more excitable by the galvanic stimulus; if, on the other hand, it be depressing, the paralytic limb will be less excitable. Thus this difference in the effect of electricity on the two limbs may serve to guide us in our diagnosis; and we may conclude that the lesion is irritative or depressing, according as the paralytic limb is more or less excitable by the galvanic stimulus.

LECTURE XXXV.

ON PARALYSIS.

ON CASES OF PARALYSIS DEPENDENT ON LESION OF THE BRAIN.

In the lecture of to-day I have to direct your attention to the concluding history of the two cases of diseased brain which I brought before you in my last lecture. Both of these cases have terminated fatally, as we had anticipated; and the opportunity is thus afforded us of comparing the diseased condition of the brain with the symptoms noticed during life.

The first of these cases was that of the man named Hardwick. You will remember that he was suffering from hemiplegia, with rigidity of the paralysed muscles; and that we treated him with galvanism, and, as is usually the case where there is recent rigidity of the paralysed muscles, these were more affected by the galvanic current than those upon the sound side—a circumstance which is due, as I think, to the exalted polarity of the nerves supplying the rigid muscles. You will not have forgotten that we derived, from the application of the galvanism, some aid to our diagnosis, and drew, from its greater influence on the paralysed than on the sound limb, the inference, that the lesion of the brain was one of an irritative kind. In the diagnosis that I then gave of this case, I expressed my belief that it was one of meningeal disease primarily, and that the brain itself was secondarily affected. This patient's death was preceded by symptoms of effusion: he became comatose for a day or two previous to this event. Upon making a post-mortem examination, we found effusion into the lateral ventricles: this was evidently recent, for the brain did not appear to have been much compressed.

In making the diagnosis in this case, you will remember that

I spoke with confidence respecting *the nature* of the disease, but hesitatingly as to *its locality*. The various segments of the encephalon are so closely connected with each other by commissural and other fibres, that the parts in the immediate vicinity of the diseased part sympathise with it to a very great extent—almost as much as if they were themselves diseased. Hence, it is very difficult, and sometimes impossible, to distinguish disease of the optic thalamus from disease of the corpus striatum, the intimate union of these two bodies causing a close sympathy between them. For this reason, lesion of the hemispheres, if situate close to the corpus striatum, gives rise to symptoms similar to those which would arise from disease of that body itself; and, for the same reason, deep-seated lesion of the cerebellum causes the same symptoms as would be produced by lesion of one side of the pons Varolii. You will not wonder, then, that it is exceedingly difficult to diagnose the exact locality of cerebral lesions. Certain broad distinctions may be made, with sufficient accuracy, by giving due attention to the general principles which physiology points out as to the functions of the great subdivisions of the brain; but I look upon it as impossible to determine the position of cerebral lesions with that minuteness and accuracy, with which we can discover the locality of lesions of other organs—the lungs, for instance.

I stated to you, that we should probably find the disease principally located in the dura mater, the arachnoid, or pia mater, near the fissure of Sylvius, and at a part corresponding to the squamous portion of the temporal bone. I also thought that the optic nerves or optic tracts, and the third nerves, would be involved in the disease, either at their origin or in some part of their course. In coming to this conclusion, I was mainly influenced, so far as regards the meningeal disease, by the fixed pain which the patient suffered about the squamous portion of the temporal bone, and just in front of the meatus auditorius externus. My first impression was, that the optic thalamus was the principal seat of the disease; but this view I afterwards gave up from observing the intensity and the constancy of position of the pain; and, although I was quite prepared to find disease of a part so nearly connected

with the optic and the third pair of nerves, as the optic thalamus, still I did not give it that importance which I had at first deemed it worthy of, and which our post-mortem examination showed that it really deserved.

I particularly wish to call your attention, gentlemen, to this subject now, while the details of the post-mortem examination are fresh upon your memories; and the more so because the diagnosis does not appear to have been quite exact. It is a duty we owe ourselves to scrutinise particularly any errors we commit in either diagnosis or practice. Depend upon it, if you do this faithfully, you will derive great benefit from it: your experience will be infinitely more profitable than if you slur over your mistakes, without explanation or inquiry. On this account, I make it a rule never to pass by any mistake made here in diagnosis or practice; and I feel that, in commenting upon such to you, I am far more likely to benefit both you and myself, than were I to dilate at length upon successful cases. The successful cases speak for themselves; the failures we would fain throw a veil over; but be assured, in so doing, we neither benefit science, nor do we confirm our own principles of truth and honesty.

A diagnosis may be erroneous in two ways: the one, in which it is altogether wrong; the other, in which, although the principles upon which the diagnosis is framed may have been sound, and have not been violated, the details may not be correctly defined. The first of these is likely to happen when our examination of the patient's condition has not been sufficiently careful; and when we have neglected to investigate his symptoms with all that accurate scrutiny, which alone can enable us to ascertain his real state; or where the information supplied to us, notwithstanding careful inquiry, has been incomplete or inaccurate. The second may occur from the absence of symptoms of a sufficiently distinctive character to give us the precise information we require, or where the attention has been unduly occupied by the inordinate development of some particular symptom: I say that, under such circumstances, although our diagnosis may have been conducted upon perfectly sound principles, it may still be erroneous in detail. Now I must tell you that, in this case, the diagnosis has been perfectly correct in principle, but erroneous

in some of the details; and I think the error has been caused partly by the difficulty to which I have already alluded, of determining the precise locality of lesions in brain disease, and partly by the prominence which the pain assumed, and by its very local character, pointing to a spot not exactly corresponding with that at which the disease was situated.

I founded my diagnosis principally upon the *pain;* the *imperfect* paralysis, and the spastic state of the muscles, denoting that the paralysing lesion was of a kind which likewise caused *nervous irritation.* Acute pain of the head is a symptom which indicates the site of the disease as either in the membranes themselves, or in some superficial part of the brain in contact with them; and it very commonly is felt at that spot in the head, which corresponds to the diseased part within the skull. Again, the imperfect character of the paralysis indicated that the morbid change was of some superficial part; for we find that the most complete paralyses are those caused by deep lesion—the nearer the lesion is to the surface, or the further it is from the corpus striatum and the crura cerebri, the less the paralysis, and *vice versâ:* and experience also shows, that an irritated state of the nerves and of the muscles of the palsied part is most frequently connected with superficial lesion of the brain, or with disease of the membranes.

I shall now detail to you the results disclosed by the post-mortem examination, and point out how far they correspond with or differ from the diagnosis formed during the life of the patient.

In the first place, we found that the disease was on the *left* side of the brain, the opposite to that on which the palsy existed,—so far, principles have not been violated; next, we found extensive *meningeal* disease, this also comporting with the inference which principles led us to draw from the persistence and the severity of the pain in the left side of the head; thirdly, we ascertained that the disease was *inflammatory,* for the products of inflammation were distinctly developed,—and, in this point likewise, the diagnosis was correct in principle.

It was wrong, however, in assigning the dura mater as being involved in the disease, for this membrane was healthy; and it was equally wrong in fixing the site of the disease at the ante-

rior inferior angle of the parietal bone; it failed, likewise, in not having indicated that the optic thalamus was the part of the brain secondarily affected by the meningeal disease.

The membrane principally diseased was the pia mater, and that part of the arachnoid connected with it, just where, at the fissure of Bichât, the former membrane is extended into the ventricles of the brain as the velum interpositum, passing over the quadrigeminal bodies, and closely connected with the optic thalamus. Here the pia mater was much thickened by the deposition of lymph: it was extremely red, and its vessels much enlarged. It formed, indeed, quite a large, soft, vascular tumour, which must have excited a good deal of irritation in the subjacent nervous matter. In this disease of the pia mater there was quite enough to explain the severe pain in the head, and the other signs of irritation present; but it is difficult to understand the precise localization of it to a part so much anterior to the seat of disease as the anterior inferior angle of the parietal bone; and this circumstance no doubt contributed very much to lead us astray. The optic thalamus on the diseased side appeared to be double its natural size, and by its great bulk compressed the crus cerebri of that side, which became flattened out by the pressure, and obliterated the locus perforatus. The crus cerebri of the right side must likewise have suffered some compression. This extensive compression necessarily affected the third pair of nerves on both sides, but chiefly on the left; and thus we obtained a satisfactory explanation of the peculiar convulsive movements of the eyeballs which this patient exhibited.

The corpus striatum was essentially healthy, but somewhat, though slightly, compressed; and its function was probably weakened.

The augmentation of size in the optic thalamus in this case was more apparent than real; for, in truth, this body was in part wasted. A large quantity of new material was deposited beneath the inflamed pia mater, which added considerably to the apparent bulk of the thalamus. In cutting into this body, it was found to be at one part soft and gelatinous, and at another indurated. The first portion was that in immediate connexion with the pia mater, and consisted of more or less perfectly

formed pus. The second portion, which consisted of the posterior third of the optic thalamus, exhibited much change in the proper texture of this part of the brain. At one part, quite close to its posterior extremity, there was a small cyst about the size of a pea, which contained pure pus, as determined by the microscope. Here and there we found minute spots of opaque, somewhat gritty matter, in which the microscope detected masses of phosphate of lime. Similar concretions of phosphate of lime were found in the diseased pia mater.

This indurated portion of the optic thalamus was submitted to chemical analysis, by my friend and pupil, Mr. Lionel Beale, jun.,* to whose skill and expertness as an analytic chemist I have been indebted on several occasions. He found that the indurated portions of the thalamus contained as much as 6·9 per cent. of the phosphatic salts; healthy cerebral matter containing, according to F. Simon, no more than 0·1 per cent. This remarkable retention or deposition of the phosphates in the diseased portion is no doubt connected with inflammation, and the arrest of the proper nutrient changes produced by it.

Thus the post-mortem inspection afforded us the most satisfactory evidence of the inflammatory nature of the cerebral lesion: the red and thick pia mater,—the puriform matter beneath it and upon the optic thalamus,—the cyst in this body containing pus,—the indurated portion of the thalamus: all these were signs of cerebral inflammation which even the most sceptical could not gainsay.

And it likewise proved the correctness of the diagnosis as to the cause of the paralysis. You will remember that I stated that the paralysis was caused by pressure, exerted not immediately, but indirectly, on the centre of volition. The pressure was found to be exerted on the optic thalamus, and through it on the corpus striatum and the inferior layer of the crus cerebri, both of which parts form a portion of the great centre of volition.

The inflammatory or irritative nature of the paralysing lesion corresponds in the most interesting manner with the augmented excitability of the paralysed muscles to galvanism, as we had ascer-

* Now Professor Beale.

tained more than once during the life of the patient. The explanation of this augmented excitability, which seems to me to be the correct one, is this, not that the muscles have experienced any increase in their irritability, but that the polarity of the nerves is augmented by the propagation of irritation from the compressed and inflamed brain to that segment of the cord in which they are implanted. The nerves, in these cases, are more or less in the condition into which they are apt to be thrown by strychnine: their vital force—their polarity—is exalted, and they are excitable by the slightest stimulus.

In fine, we learn from the review of this case that our diagnosis was sufficiently correct for all practical purposes—that, following the general principles which our present knowledge of cerebral physiology indicates, we obtained all the information we could desire for the proper treatment of the case. This treatment possibly might have been completely successful had the patient been submitted to it at a sufficiently early period before any great quantity of morbid deposit took place.

The subject of our second case, Catherine Williams, lingered on for some weeks; no improvement whatever took place in the condition of the paralytic limbs: they became extensively anasarcous, and the muscles extremely attenuated; and the patient died from sheer exhaustion.

The post-mortem inspection afforded very satisfactory proof of the correctness of the diagnosis in this case, both as to the nature and as to the locality of the lesion. The disease was in the very centre of the right corpus striatum, one third of which must have been destroyed by it. The whole brain was shrunk; and the quantity of subarachnoid fluid was much increased. Several of the arteries at the base of the brain were studded with atheromatous spots. The convolutions were small, and the sulci between them large.

The general shrinking of the brain explained the increased quantity of the subarachnoid fluid.

As to the nature of the disease: the middle third of the corpus striatum was excavated into a small cavity, which was filled by fluid and softened brain-substance; probably, also, by half-dissolved softened clots of blood. On examining the con-

tents of this cyst by the microscope, we could find no trace of any inflammatory product; there were some remains of nerve-tubes, and the rest consisted of an undefined granular matter, derived probably from the destruction of the vesicular matter of the corpus striatum.

I think there can be no doubt that in this case there was first simple softening (without discoloration) of the middle third of the corpus striatum; next came the solution of continuity of some of the fibres of the corpus striatum; and, possibly, at the same time, the rupture of some minute vessels, and the effusion of blood in small quantity. At this time the limbs became paralysed; and, owing to the total destruction of the brain-substance in a part so important as the corpus striatum, and the entire absence of any reparative effort, they never evinced the least sign of improvement, or of any other change, save wasting.

It would be difficult to find a more perfect example of a brain suffering from imperfect nutrition than this. No doubt the local softening was due to some defect in the local nutrition, the precise nature of which, however, we were not able to detect.

The nerves of the paralytic limbs were depressed in their vital powers; the want of their wonted stimulus, the will, suffered them to fall into decay; and very probably the morbid state of the corpus striatum exercised a depressing influence upon them. Hence their polarity was much below par; and the galvanic stimulus, which excited free action in the sound limbs, produced little or no effect on the paralysed limbs.

There is one point in which these cases present an interesting contrast with each other, to which I must allude before I conclude this lecture. The man, Hardwick, as you will remember, died comatose, and we found an undue quantity of fluid *within* the ventricles, and no subarachnoid fluid around the brain. The woman, Williams, died from exhaustion, without any symptom immediately referable to the brain. In this case, the subarachnoid fluid was abundant, but there was no fluid in the ventricles.

You will find, I think I may say invariably, that the accumulation of fluid in the ventricles, when it exceeds a certain

amount, produces coma. In the adult the comatose symptoms come on earlier, and with a less amount of effusion, than in the child, from the resisting nature of the cranial wall in the former, whilst, in the latter, the still open state of the fontanelles, and of some of the sutures, allows the skull to expand, as the fluid in the ventricles increases in quantity.

On the other hand, the increase in the subarachnoid fluid is not in itself accompanied by any special symptoms. This augmentation of a fluid which naturally occupies the subarachnoid space is due entirely to a shrinking or diminution in the bulk of the brain, from whatever cause; and its quantity bears an inverse proportion to the bulk of the brain. You find it in large quantity in the crania of persons dying anæmic, and also when the brain has been much impaired in its nutrition, so as to cause a diminution of its bulk; and even if there be a local diminution of bulk, as when one or two convolutions have shrunk, or have sunk in from the destruction of the subjacent cerebral substance, you will find an accumulation of fluid opposite the shrunk or depressed convolutions.

LECTURE XXXVI.

ON PARALYSIS.

ON A CASE OF PARALYSIS OF THE FACE, DEPENDENT ON LOSS OF POWER OF THE FACIAL NERVE (PORTIO DURA OF THE SEVENTH PAIR).

GENTLEMEN,—The case upon which I propose to comment to-day is one of not uncommon occurrence—a form of paralysis of the face dependent on the loss of power of the facial portion of the seventh pair of nerves.

As every form of palsy has a formidable appearance, and as this is particularly the case when the face is affected, and the

more so in proportion to the greater distortion of the countenance, I advise you to make yourselves well acquainted with the various kinds of palsy that affect the face. The alarm, which a loss of power on one side and the consequent distortion of the features occasion to the patients or their friends, is very great—and naturally so. Paralysis is a formidable symptom; and on its first appearance it is apt to be looked upon as a sign of the break-up of the patient's constitution—an indication that his doom is sealed. It is very important that, under such circumstances, the medical attendant should display a perfect acquaintance with the real state of the case, and be able to allay the patient's or his friend's fears, when it is possible to do so. Since in the generality of palsies, such as the patient now in the hospital suffers from, you may give with confidence, at least as regards the patient's life, a favorable prognosis, you ought to possess a thorough knowledge of the signs and the symptoms of this malady, so as to enable you to recognise it readily and with certainty whenever it comes before you.

Case CLIV (vol. xxi, p. 228).—The subject of this case is John Garrey; he is in Fisk ward, and you can scarcely fail to recognise him by the peculiar expression of his countenance; for on one side his look is most doleful and melancholy, while on the other it is very much the reverse.

He is thirty-nine years of age, and was admitted into the hospital on the 6th of January, 1848: the report in the case-book of that date goes on to say, that "he has lived in London ever since he was nine or ten years of age; is a married man, a carpenter by trade, temperate, and has always had good general health until a week ago, when, after keeping in-doors for a fortnight in consequence of having a bad leg, he was obliged to go out in search of work, and was exposed all day to very cold weather; in the evening he had an attack of shivering, and twitching in the under lip on the right side; after supper he found his lip drawn to the left side, but it was free from pain. About four days after this he was seized with a severe pain behind the right ear, which still continues, as do the distortion of the face and inability to close the eye on the right side, even during sleep; he cannot whistle, and he frowns only

on the left side. Upon trying to shut the eye he carries the ball upwards and inwards, but does not bring the lid down over it, excepting a very little. He protrudes the tongue in the median line."

The leading character of cases of facial palsy such as this is the inability to close the eyelids, from paralysis of the *orbicularis palpebrarum* muscle; this is the pathognomonic sign which determines the peculiar nature of the palsy, and distinguishes it from the more serious form of facial palsy which is dependent on disease of the brain and palsy of the fifth or of the third nerve.

It is remarkable how seldom the seventh pair of nerves is affected by disease of the brain. I cannot say that I ever saw an instance of complete paralysis of the orbicular muscle of the eyelids due distinctly to uncomplicated disease of the brain; and I have only seen a few in which the power of the muscle appeared to be enfeebled from that cause. Thus we have a point favorable and consolatory to a patient afflicted with *portio dura* paralysis; namely, that the affection being seated in that nerve need not excite the same alarm as to disease of the brain as in other cases of partial palsy, that of the third nerve, for instance. Moreover, disease of the brain would give rise to a different form of facial palsy.

You have only to examine this patient with care, and you will find that he has almost every sign which indicates that the paralysis has its seat in the portio dura nerve. He cannot close his right eyelids; in making the attempt, however, he seems not to have lost the power altogether, for the upper lid is slightly depressed; yet if you put your finger on the orbicular muscle you do not find the slightest contraction of it. How, then, is this slight depression of the upper lid produced? Watch him closely while he shuts the left eye and attempts to do the same with the right, and you will perceive that at the moment the left eye is closed, the right eyeball turns upwards and inwards to such an extent, that the cornea is nearly or wholly concealed by the upper lid, and by this upward movement of the ball the upper lid is slightly depressed. The same upward movement of the eyeball takes place on the sound side at the moment of the forcible contraction of the orbicular

muscle. It is a very curious instance of an involuntary movement which cannot be controlled, accompanying a forcible action of another kind; and it has reference to the complete protection of the eyeball against those sources of injury which would occasion the forcible closure of the eyelids.

Sir Charles Bell, to whom we are so much indebted for our improved knowledge of the paralytic affections of the face, dwelt much on this upward movement of the eyeball. He affirmed that it took place in sleep, and that during sleep the eyeball retained this position. I doubt much the correctness of this assertion. I have had many opportunities of satisfying myself that in perfectly tranquil sleep the eyeball is directed forwards, and seems suspended in the orbit, being equipoised among its muscles. Close the eyelids slowly and gently, and the eyeball remains quiescent,—contract the orbicular muscle forcibly, and the eyeball instantly turns upwards and inwards. When the orbicular muscle is made to contract strongly as a reflex action, as when you try to push any object into the eye, the upward movement takes place. But in ordinary winking you have none of it. This movement of the eyeball, then, accompanies only forced contraction of the orbicular muscle of the eyelids.

If you will take the pains to watch persons sleeping, whenever you have the opportunity, you will find that in *sound* and *tranquil* sleep there is no indication of active contraction of the orbicular muscle; there are no wrinkles of the eyelid, and no depression of the brow, as when that muscle is in strong contraction;—if, with the greatest care and gentleness, you raise the upper lid, you will find the eyeball directed forwards, maintained in this position by the equilibrium of its muscles. Should your attempt to raise the lid give rise to a reflex action, you will encounter a distinct resistance from the contraction of the orbicular muscle, and the eyeball will be turned upwards and inwards, more or less forcibly in proportion to the force of the reflex action. I think, therefore, we are justified in asserting, that in sound sleep the position of the eyeball is one of quiescence, that it is maintained in that position by the passive contraction of all its muscles, and that the eyelids are kept closed by the passive contraction of their orbicular muscle, and that during

sleep there is no effort or influence of the nervous system directed upon any of these muscles. It is only when sleep is disturbed, when the mind is more or less active, as in dreaming, that you will find active contraction of the orbicular muscle of the eyelid.*

Our patient is unable to frown on the right side, while he does so distinctly on the left; neither can he move his scalp on the right side; the corrugator supercilii, and the frontal portion of the occipito-frontalis muscles, on that side being paralysed. The levatores alæ nasi, and the zygomatic muscles, are likewise paralysed on the right side, and therefore the right nostril is motionless, and the angle of the mouth hangs on that side. The orbicularis oris muscle is paralysed as to its right half; the patient is consequently unable to purse up his mouth, and if you ask him to whistle, he will afford you indications of his inability to perform this, as well as other actions. In making the attempt to whistle, you may perceive that he contracts the orbicular muscle of the mouth on the left, but not at all on the right, and so he is quite unable to get his lip into the position necessary for the production of sound; and while trying to adapt his mouth for this purpose, he smiles or laughs, as is so often the case when you ask a person to whistle, and he thus affords you the opportunity of seeing how completely the action of the features is confined to the left side. The act of smiling or laughing is exaggerated on the left side, and the reason is because the left muscles have completely lost the resistance of those of the right side, which remain perfectly motionless, and

* Since this lecture was delivered, I have repeatedly examined this point in patients under the deep sleep produced by chloroform. And in every instance, reflex actions being in such cases in abeyance, the eyeball remained undisturbed when the upper eyelid was raised.—I have notes of one case (Case VII, Nov. 1840), in which the left eyeball (the palsy being on that side) was turned upwards and *outwards*, when the eyelid was raised. It is probable that this was due to a slight and transient palsy of the third nerve, or of the ciliary nerves, as there was at the same time dilatation of the left pupil. The patient was a gentleman of decided gouty constitution;—the palsy was removed in a fortnight. In this case I noted a symptom which is alluded to by some continental writers—namely, a slight defect in the power of taste on the left side of the tongue. This was probably due to a coincident affection of the superficial nerves of the tongue.

which from disease have lost their tone, and have suffered much in their nutrition. For the same reason all the movements of the features which act in symmetry, and which at the same time counterbalance each other, are found to take place to an exaggerated extent on the healthy side. Hence in smiling, laughing, and speaking, the face is drawn more or less to the left side: the distortion takes place on the healthy side, the paralysed side remaining unmoved. The popular notion, in cases of this kind, is, that the disease is on the side to which the mouth is drawn. No medical man, however, can fall into this mistake if he be at all acquainted with the real condition of the patient.

Another muscle which is paralysed in this case, and in all cases of the same kind, is the buccinator. Hence the cheek hangs loose, and as the patient speaks, it flaps to and fro. This extreme looseness of the cheek is not an early symptom of this form of paralysis; it manifests itself more and more, the longer the duration of the disease, and ultimately becomes the cause of symptoms very troublesome to the patient. It interferes not only with articulation, from its looseness and the flapping movement while the patient is speaking, but with mastication likewise. The paralysed muscle allows the food to accumulate between the teeth and the jaw, and fails in its function of supplying the mill with its proper amount of material to be ground. After a little time, patients learn to remedy the defect of articulation which the paralytic condition of the buccinator muscle causes, by supporting the cheek with the hand; and a similar kind of support helps to remove the inconveniences of mastication.

Increasing flaccidity of the cheek, and especially a rapid development of that condition, is a symptom of unfavorable omen as regards the patient's prospects of complete recovery.

You will observe that all the muscles paralysed in this affection are *superficial:* they are all muscles more or less concerned in the expression of the countenance. The deep-seated muscles are not affected—these are muscles of mastication; the only muscle paralysed, which is concerned in mastication, being the buccinator, which is, however, only accessory to that function, and is as much or more a muscle of expression.

And now we come to a most important question—What is the exact nature of this disease? Is it a disease of certain muscles, or of a certain nerve or nerves, or is it an affection of the brain? Its one-sided character would denote its being a cerebral affection: it may, however, occur simultaneously on both sides, and I have myself seen two instances of this kind. Experience, however, as I have already told you, assures us that it very rarely indeed accompanies cerebral disease; sometimes it occurs as the result of *intra-cranial* disease, less frequently from lesion of the brain itself.* What, then, is its nature? Sir C. Bell clearly pointed this out long ago, and to him we are especially indebted for our knowledge of the precise nature of the disease; so much so, that some designate the disease "Bell's paralysis of the face." Not that I should recommend you to adopt this name; for I must say that I cannot regard it as a compliment to the great names of our profession, to attach them to any of the numerous ills which flesh is heir to.

Sir C. Bell first pointed out the true nature of this palsy, because he was the first to unravel the intricacy of the nerves of the face. He showed that one nerve, and one nerve only, was at fault in this disease, and that it was strictly a local paralysis, due to a lesion in some part of the course of this nerve. The affected nerve is the portio dura of the seventh pair, the proper facial nerve, which supplies all the muscles paralysed in this affection, and is the only motor nerve which supplies nearly all of them. The fifth pair is not affected, because the muscles of mastication are free, and because the sensibility of the face remains intact. Sometimes the patient complains of slight pains

* This remark applies to simple palsy of the portio dura, unaccompanied by any other symptom referable to brain disease, such as pain in the head, giddiness, confusion of ideas, affection of speech, delirium, or by any other form of paralysis. Dr. Watson records a very interesting case, in which palsy of the portio dura was among the first symptoms; but, in this case, there were other and very decided symptoms, too, of brain mischief, and the lesion proved to be "a cancerous tumour, occupying the right hemisphere of the brain; at its under part, a clot as big as a hazel-nut" ('Lectures,' vol. i, p. 541). A careful analysis of the particulars of this case shows, I think, that this lesion operated only indirectly in the production of the palsy. The portio dura and the portio mollis were compressed and hardened by the superimposed tumour, which pressed down the intervening brain-substance upon them.

in the face, which may probably be due to a slight affection of the filaments of the fifth.

There is, however, one muscle paralysed in this affection, which does receive a supply from the fifth—namely, the buccinator. This muscle has two motor nerves,—a branch of the facial, and the long buccal nerve from the fifth: the former may be regarded as its nerve of expression; the latter as its nerve of mastication. How comes it, then, that if the first be paralysed and the muscle ceases to act in expression, it likewise ceases to act in mastication? The two nerves are distinct; and the buccal nerve is one of considerable size, and to all appearance would seem perfectly adequate to the maintenance of a different action independent of the portio dura. It is not easy to find an explanation of this curious fact, which is equally true if the nerve first palsied be the fifth—as in cases of hemiplegia, in which the hanging of the cheek is due to paralysis of the buccal nerve and of the buccinator muscle. The advocates of Dr. Hall's views would doubtless explain it by assigning to the facial nerve a specially spinal character, and to the fifth, a cerebral. The palsy of the facial nerve would, according to these views, not only destroy the influence of the will over the muscle, but also cut off its supply of irritability. Without going into other objections fatal to this explanation, it is quite enough to state that it is inadequate to explain the complete palsy of the buccinator muscle when the fifth is the only nerve affected, as in common hemiplegia.

In some instances the velum of the palate participates in the paralysis; and when you look into the patient's throat, you find the uvula inclining away from the paralysed side, and the velum drawn to the sound side. It is probable that the portio dura exercises some influence on the muscles of the palate, through the greater superficial petrosal nerve of Arnold, which arises from the knee-shaped swelling of the trunk of the portio dura in the aqueduct of Fallopius, and communicates with Meckel's ganglion, whence the palate-muscles derive their nerves. Possibly this influence may be more direct in some cases than in others.

Romberg considers this symptom as indicating that the paralysing lesion affects the nerve in the Fallopian aqueduct. I

have seen undoubted instances of disease of the aqueduct causing paralysis of the nerve in which the symptom did not exist. In my experience it is a symptom of very rare occurrence, and I incline to think it may be looked upon as a coincidence, like the partial defect of taste to which I have already alluded.*

As this is a local palsy, its causes are generally strictly local. Thus a common cause of it, and especially in strumous children, is *otitis*, and the subsequent caries of the petrous portion of the temporal bone. In such cases the paralysis is generally very complete: it is caused by inflammatory or destructive disease of the nerve in the Fallopian aqueduct, and it is often associated with a discharge from the ear, and with deafness. Injury to the trunk of the nerve may give rise to this form of palsy: hence it often follows surgical operations on the face, and accidental wounds in the parotid region; and formerly, before the true function of the facial nerve was known, when surgeons used to

* A curious instance of this attraction of the uvula to one side occurred not long ago (Feb. 1855—case of James Osborne, Vol. xlvi, p. 27) in the hospital, in connexion with a *spasmodic* condition of all the muscles supplied by the portio dura, as if that nerve were the seat of a continued irritation. The left side of the face was affected, and all the superficial facial muscles were in a state of spasm, including the orbicularis palpebrarum, which kept the eyelids constantly closed. There was imperfect paralysis of the left arm and leg, and the symptoms seemed to indicate a lesion paralysing to the left arm and leg, but approaching sufficiently near the point of implantation of the facial nerve of the left side to keep all its fibres in a state of irritation. There was a remarkable unsteadiness of gait, and the power of co-ordination was very much impaired, especially on the left side. If he stretched out his hand to touch an object, it went zigzag, and ultimately far wide of the mark; and he could not carry a cup of tea to his mouth without spilling it. At first starting to walk it was necessary to support him, and he walked like a drunken man; in attempting to turn, he was always in great danger of falling. There was slight failure of sensibility of the *right* side. The patient improved under treatment, left the hospital with more power in his left side, and better able to balance himself, but with the spasmodic state of the facial muscles unchanged, and the uvula still inclining. He has not been heard of since.

I may remark here that there is an *acute* affection of the portio dura nerve which induces most painful spasms of the facial muscles. This is essentially different from the affection in the case referred to. It occurs in connexion with gouty or rheumatic conditions, and is analogous to the acute affections of the sciatic and median nerves.

divide this nerve for *tic douloureux*, this form of paralysis used to be regularly manufactured by chirurgical skill.

A very common cause of this palsy is the influence of cold; as by exposure at an open window in a coach or railway carriage to a current of cold air. The case under our consideration was one of this description, the patient having been exposed the whole day to a cold atmosphere while in search of work. These are instances of what has been called "*peripheral paralysis*,"—cold acting directly on the peripheral ramifications of the nerve.

Sometimes you meet with cases which cannot be satisfactorily traced to exposure to cold; the patients, however, will be found to be out of health, and to have had pains about the face and neck for some days. It is probable that in all cases which have not a traumatic origin, or are not caused by disease of the petrous bone, there may be some constitutional fault which may show itself in this local malady, just as painful affections of sentient nerves—the fifth, for instance—are undoubtedly generally of constitutional origin.

Periodical neuralgic affections are, I believe, very frequently due to the determination of some poison to a particular nerve—as the paludal poison, or some matter generated in the system, gouty or rheumatic. There is no reason why such morbid matters should not affect a motor nerve as they affect a sensitive nerve, causing paralysis in the one case, and neuralgia in the other.

Mr. Bowman tells me he has met with several cases of distinctly rheumatic paralysis of the portio dura, and also of some of the nerves of the orbit, among the patients at the Ophthalmic Hospital, Moorfields.

Case CLV.—Very lately the following case has come before me, being one of palsy of the portio dura, which had marked evidence of being rheumatic in its origin. A man, aged twenty-eight, had severe rheumatism of some of the intercostal muscles of the left side. This got well, and then the muscles of the hip became affected, and he was completely lamed in consequence. As these were getting better, he found his face to become suddenly paralysed on one side, with all the symptoms

of palsy of the portio dura. During the first few days of his illness, the urine deposited lithic acid and lithates freely. The case ended favorably.

Case CLVI.—In November, 1843, I attended, with Mr. Wetherfield, a gentleman, living in Regent-street, eighty-five years of age, in whom complete and well-marked palsy of the portio dura came on under the following circumstances. He had in general enjoyed excellent health, but began to suffer from pains in the right arm and right side of the face, which he regarded as rheumatic, and which did not seem referable to any other cause. These continued for several days uninfluenced by treatment, when one morning, as he was proceeding to shave, he found the right cheek hanging loose, and the face drawn to the left side. I saw him soon after, and found all the signs of palsy of the portio dura unusually well marked. The cheek became flaccid and wasted with remarkable rapidity, and our patient suffered great inconvenience from it both in mastication and in speaking. I never saw more striking deformity than this patient presented within three weeks of the invasion of the palsy, from the staring eye and the flaccid and hanging cheek. He never evinced the slightest effort at recovery, and died within three months of senile gangrene of the toes, but without any fresh symptom referable to the nervous system.*

* Other forms of local paralysis may occur in states of constitution, if not rheumatic, at least allied to it, with imperfect action of the kidneys.

The following affords a good example of this:

Case CLVII.—A medical man, aged fifty-three, extensively engaged in practice in the county of Berks, applied to me in August, 1847, with complete paralysis of the deltoid muscle. He was a stout, full man, tall, of large build, and very active in his habits; fed well, and drank beer, but not to excess. He had been subject to a shifting neuralgia of the scalp, and to a discharge from the right ear, where he thought the tympanic membrane was destroyed; he was deaf on that side. Six weeks before he came to me he suffered from pain in the left side of the neck and shoulders, followed by complete paralysis of the left deltoid muscle and weakness of the whole arm. On examining, I found a total inability to raise the left arm to a right angle with the trunk, or to perform any of those actions which are usually effected by the deltoid muscle, which was very much wasted. He could, however, grasp perfectly with the left hand, and execute all the other movements of the arm and of the forearm. There was some degree of numbness of the arm. There were no symptoms

This form of palsy comes from exhaustion, as in that exhausted state of system which occurs after parturition and from nursing. The following is a good example.

Case CLVIII.—A married lady, twenty-five years of age, of highly nervous temperament, was brought to me by Dr. Westmacott in May, 1851. On the 5th of that month she had a miscarriage, which was followed by considerable hæmorrhage, with debility and a hysterical state. On the 13th she had inflammation, with slight ulceration of the right tonsil, which yielded readily to treatment. On the 19th, having been more than usually hysterical, after a fit of laughter, she found her face drawn to the left side, and on the following day there were unequivocal signs of paralysis of the portio dura on the right side. There was slight tenderness of the parotid region on the right side. The only treatment adopted in this case consisted in the use of warm fomentations over the right parotid region and the exhibition of the tincture of the sesqui-chloride of iron. In three weeks she was quite well.

Case CLIX.—As an example of this affection coming on under exhausting influence, I may refer to the case of Mr. H., aged thirty-five, an overworked medical practitioner in a very populous district, largely employed in midwifery practice. After an unusual amount of night-work (March, 1848), he found

distinctly referable to the head. His tongue was coated; appetite good: the discharge from the ear had ceased. The urine was pale, of low specific gravity, and contained albumen in small quantity.

I viewed the case as one of local palsy, connected with a deranged state of system, rheumatic or gouty. I regulated his diet, and gave him small doses of the mineral acids. After a fortnight of this treatment, he improved considerably; and could raise his arm slightly. The albumen in the urine had much diminished; and crystals of lithic acid were precipitated. He was now ordered three grains of iodide of potassium, with ten minims of liquor potassæ, thrice a day. He only followed this treatment for ten days, as the iodide of potassium purged him. Still he was improving. I continued the liquor potassæ, and advised galvanism to the muscle. This plan was diligently pursued for a fortnight, at the end of which time he had so far improved that he could raise his arm nearly to a right angle, he could put on his coat and tie his cravat; and in three weeks more he was quite well. All signs of albumen had disappeared from his urine.

himself paralysed on the left side of his face. All the signs of palsy of the portio dura were present. The attack was not preceded by headache, nor by any pain in the face or neck, nor any affection of the ear. This patient speedily recovered on going to the country, and taking tonic medicines.

The cause of the palsy, in our patient Garrey, appears to have been the direct influence of cold. This view is confirmed by the pain which the patient suffered at first in the neighbourhood of the ear; as if the ear itself and the nerves about it were chilled, and some degree of inflammation excited in them in consequence.

The duration of this palsy varies considerably: it rarely, if ever, lasts a shorter time than ten days, whilst it very often extends to as many weeks: perhaps three or four weeks may be assigned as an average duration for the non-traumatic cases.

The prognosis in cases of this kind should always be founded upon the cause. When the paralysis has been caused by mechanical injury, your prognosis must generally be unfavorable, more especially if any distinct solution of continuity have taken place in the nerve. Nerve-substance is very slow of regeneration; and when it is reproduced, the new fibres do not adapt themselves with precision to the old ones, and so they form very imperfect conductors of the nervous force. But if the paralysis is due to cold or to some constitutional cause, it almost invariably gets well. But you should bear in mind, that even in cases which are incurable by reason of the solution of continuity of the nerve, there is little in this form of paralysis tending to shorten life, or calculated to prove otherwise than inconvenient, by causing imperfection of speech, mastication, and vision, and sometimes of deglutition.

I must, however, beg of you not to lose sight of the fact that sometimes paralysis of the portio dura may be the forerunner of much more serious disease. The disease of the temporal bone, on which the palsy depends, may pass on to caries, and may excite meningeal inflammation and even abscess of the brain. A very striking example of this you will find recorded in Dr. Graves' 'Clinical Medicine,' vol. ii, p. 569.

In Garrey's case I have given a favorable prognosis, believ-

ing that no serious mischief has been done to the nerve, and that it has not been the subject of destructive disease. He begins to gain some power over the orbicular muscle of the eyelids, and the distortion of the face is somewhat less. The duration of the palsy has already been quite three weeks, and it seems probable that the patient's recovery will not be rapid, as so little amendment has as yet shown itself. I have observed that when the symptoms begin to mend early—that is, within a week—complete recovery takes place very rapidly; but if the first signs of improvement show themselves late, the recovery is slow, or only partial.

You will likewise find it necessary to be guided by the cause of the palsy as to the course you will pursue in its treatment. If otitis be its cause, and the inflammation be of recent occurrence, it may be necessary for you to have recourse to the usual antiphlogistic measures for its suppression; and in such a case it may be desirable to carry the use of mercury to ptyalism. In the palsy from division of the nerve, all medical treatment is useless; and when the disease has been caused by cold, or has arisen from any constitutional cause, much medical interference is not requisite. If there be pain of the face, warm fomentations will prove useful. Sometimes a few leeches at the angle of the jaw, or over the parotid space, or behind the ear, may be tried, or a blister, or iodine paint. I cannot say that I have ever known clear and distinct benefit produced by any of these latter remedies in shortening the duration of the palsy.

In the use of internal remedies you must be guided by the diathesis, and the existing condition of your patient. Mild purgatives are generally useful, and sometimes alkalies and sudorifics, and I have seen decided benefit from the use of the iodide of potassium. In the cases which follow exhaustion, tonics are clearly indicated. I cannot name to you any remedy which will act specifically on the palsied nerve. Strychnine is of no use in such cases.

As to local remedies, I advise you to abstain from the use of them, if possible. Blisters are open to this objection, that they sometimes cause enlargement of the neighbouring glands of the neck, which, by their pressure, may increase the evil we wish to

remove. Galvanism, employed carefully, may be useful,—always remembering, in the application of it, to vary the direction of the current, and never to continue it so long as to exhaust any small amount of nervous force which the nerve may be capable of maintaining.

Our patient, Garrey, has been treated chiefly by leeching and fomenting, and purging, in the first instance, and afterwards by the iodide of potassium. He has been completely relieved of pain, and his muscular power is beginning to return. I propose shortly to try the effects of galvanism with him.

Garrey suffers from a very troublesome symptom of frequent occurrence in these cases, and which is very difficult to deal with —I mean irritation of the conjunctiva, occasioning free lacrymation and soreness of the eye. This is obviously due to the constant exposure of the eye, occasioned by the loss of the power of winking; and it can only be obviated by attention on the part of the patient to the protection of the eye, or by his wearing a shade to cover it.

[This patient got quite well, galvanism having been applied for two or three days. Four weeks elapsed from the occurrence of the palsy to his complete cure.]

The following cases are of sufficient interest to induce me to subjoin a brief detail of them in further illustration of the clinical history of this form of paralysis.

Case CLX (vol. xvi, p. 96).—F. M'Casey, aged twenty-eight, a silversmith, of very dissipated habits, had syphilis ten years ago, health otherwise good. On the night of the 6th Dec. he was out till a very late hour, and next day observed a redness of the conjunctiva of the right eye, which did not subside readily. There was no accompanying sore throat nor any glandular enlargement. On the morning of the 13th, on getting out of bed, he found himself unable to spit. He went to his workshop, and there the peculiar condition of his features excited the risibility of his fellow-workmen, which induced him to come to the hospital to have the peculiarity investigated. All the usual signs of paralysis of the portio dura were present,—the right eye stared, and the right cheek was flaccid,—mastication was im-

paired on the right side, but the masseter and temporal muscles acted perfectly. The conjunctiva of the right eye was red and irritable, and tears flowed freely and to a troublesome extent. The right parotid gland was slightly painful to pressure; and he stated that since he had been salivated, six months ago, the parotid glands were liable to become tender and swollen upon the accession of the slightest cold. There was slight deafness of the right ear.

This patient was treated by the application of four leeches over the right parotid, and iodide of potassium was exhibited in the dose of three grains thrice a day. The latter was omitted on the eighth day. Under this plan the paralysis gradually subsided, and he left the hospital, cured, on the 31st of December, eighteen days after the attack.

Case CLXI (vol. xxiii, p. 46).—Harriet Winter, aged eighteen, a servant, previous health good, excepting that she has for the last two years suffered occasionally from pains in the head. On the morning of the 11th February, she awoke with severe pain across the forehead and top of the head, and numbness of the left side of the face, which was much swollen from inflammation of the gum of a carious tooth.

At the same time she was seized with severe pain as of otitis in the right ear, and the day after (Feb. 12) the face became drawn to the left side. She has also been much troubled with pain and numbness of the upper and lower extremities on the left side. She was admitted into the hospital on the 28th February with well-marked symptoms of palsy of the portio dura of the right side. "She frowns, laughs, and talks with the left side only,—cannot close the right eyelid; there is tenderness on pressure over the trunk of the portio dura of the right side: complains much of pain in the right ear, from which there is now a free discharge of pus." With these symptoms of local affection on the right side, there were pains and numbness of the left arm and leg, and headache, but no distinct paralysis on the left side.

After this patient had been a week in the hospital, she began to complain of pains in the right arm and leg, similar to those she had been suffering from on the left side, and on the 13th of

March all her large joints became affected with rheumatism, they were red on the surface and swollen; she sweated freely, and had a furred tongue, and her pulse rose to 100. The attack, in short, put on the characters of mild rheumatic fever. In about twelve days the fever and articular swelling were subdued, and the otitis had also improved very much. The pulse was now 80. She had so far regained power as to be able nearly completely to close the right eye. There still remained, however, considerable distortion of the face when she laughed or spoke. The discharge from the ear ceased, but some degree of deafness remained. This patient continued in the hospital till the middle of May, in all ten weeks, without any further improvement.

In consequence of the otitis, and the apparent threatening of intracranial mischief as indicated by the disturbed sensation on the opposite side of the body, mercury was at first used in this case for a short time; subsequently the rheumatic affection was treated with sudorifics and blisters to the joints.

There can be no doubt, I think, that this was a well-marked instance of rheumatic otitis causing palsy of the portio dura, the nerve having been so much damaged that it probably would never perfectly recover its functions.

Case CLXII.—Charles Pottage, aged eighteen (vol. xxxi, A.), a compositor, works a great deal by gaslight; his mother is highly rheumatic, and he has himself suffered a good deal from rheumatic pains, especially of the right arm, which have weakened it very much. For six months past he has suffered very much from dyspeptic symptoms. A week before his admission his right eye became very weak, and ran a great deal. He then applied to me, and I discovered all the signs of paralysis of the right portio dura, and sent him into the hospital on the 4th of January. The right side of his face exhibited all the usual signs. There was complete palsy of all the superficial muscles except the orbicularis palpebrarum, which acted slightly in winking in harmony with its fellow of the opposite side.

As this patient's general powers were very much depressed, he was treated by bark and mineral acids, and very mild aperient medicine, and liberal diet. His improvement was marked and progressive, and he was discharged cured on the 13th, being

the 9th day of the treatment, and the 16th from the commencement of the paralytic attack.

Case CLXIII.—For the outline of the following remarkable example of paralysis of the portio dura on *both* sides, I am indebted to my friend, Mr. Holthouse, of the Westminster Hospital.

W. B., aged forty-two, formerly a water-cress gatherer, became a patient of the Public Dispensary, Carey-street, Lincoln's Inn, in December, 1848, for rheumatic or gouty effusion into the left knee-joint. As the patient was perfectly deaf, and his face was paralysed on both sides to a remarkable extent, Mr. Holthouse was led to inquire into the circumstances which preceded this extraordinary amount of facial palsy.

From a very imperfect statement which the patient furnished to Mr. Holthouse, it appears that the illness which issued in this formidable face paralysis began so long ago as thirteen years. He was then seized with a severe pain in the left foot, so bad that he could not put his foot to the ground that day. In the night he was attacked with violent pain in the forehead, and that in the foot left him; soon after this he appears to have become paralysed on the right side of the face—the eye, as he describes it, staring open, and looking large and prominent. He used to suffer from pains in the head, chiefly on the right side and in the right ear, with more or less of noise in the head. His hearing now became affected, at first so that talking to him was exquisitely painful, but afterwards deafness came on, affecting both ears. A discharge now came from the right ear, and he describes the lower lip as "dropping down," and the jaw-bone on both sides appearing out of place. Probably both buccinators became at this time paralysed. When he ate or spoke he was forced to put his hand to his lower lip to hold it up.

I subjoin the description of the phenomena which this patient presents, as given me by Mr. Holthouse.

"The features generally lack expression, and their muscles are wasted; the eyes protrude, and the eyelids are farther apart than natural, so that a large portion of the sclerotic is thus exposed to view; the conjunctivæ are suffused with tears and slightly injected; winking takes place at about the usual

intervals, though less completely than natural; he can approximate the lids voluntarily when told to shut his eyes, but cannot bring them into contact one with the other, and in endeavouring to do this the eyes are rolled strongly upwards. Can neither frown nor raise the eyebrows, nor laugh, nor whistle, nor put in action any of the muscles supplied by the portio dura, unless it be the orbicularis palpebrarum to the extent already indicated. The upper lip is so elongated that its border is on a level with the margin of the gum of the lower jaw, while the lower lip is everted and pendant as if a weight were suspended from it; by an inspiratory effort both lips can be brought into apposition, and maintained so as long as the effort is continued. The cheeks are wasted, sunken, and flaccid, and the patient has lost the greater number of his teeth. The paralytic condition of the lips and cheeks, together with the loss just referred to, renders the pronunciation of the labials and dentals impossible; consequently his articulation is very imperfect, and mastication is performed with some difficulty; he is obliged to eat slowly, and to make use of his fingers as aids to the lips in keeping in the food, as well as to remove it from between the gums and the cheeks, where portions of it are liable to accumulate. In drinking he presses the rim of the glass against the cuticular surface of the lower lip just below its mucous margin, whereby the pendulous free edge is lifted up and bent inwards towards the cavity of the mouth; in consequence of the lips being constantly apart, the labial glands on the lower lip can be seen to pour out their clear fluid secretion, which stands in minute and separate drops, like fine dew covering its surface; this fluid is perfectly clear, transparent, slightly viscid, neutral to test-paper, and free from all organic matters, as seen through an eighth-of-an-inch object-glass. Is perfectly deaf with both ears, the loudest sounds, even through a trumpet, being quite inaudible to him; notwithstanding this high degree of cophosis, like many deaf persons, he fancies he can hear, and is a regular attendant at church; can hear, he says, the playing of the organ and the rumbling of the carriages in the street, and I have satisfied myself that he is really conscious of the passing of vehicles; but it is evident that he derives the information from the vibrations conveyed to the general surface of

his body, and not to the auditory organ; in other words, he feels the vibrations, but does not hear them; has occasional tinnitus, which he likens to the noise produced by putting a shell to the ear—"a kind of hissing noise;" sudden snappings are likewise occasionally heard in the left ear. On examining the ears, nothing abnormal is detected on the left side; but from the right meatus there flows a fœtid, purulent, and occasionally bloody discharge. A fungus growing from the cavity of the tympanum occupies the bottom of this passage, the membrana tympani being destroyed and the ossicula absent."

CLXIV.—I am induced to add another case, as a rare and instructive example of paralysis of the portio dura, in combination with temporary paralysis of a portion of the fifth nerve, and of the third, and apparently connected with secondary syphilis.

Catherine Regan, aged thirty-six, admitted February 15th, 1845 (vol. xiv, p. 37), married, and mother of two children. In the summer of 1843 she was under surgical treatment for syphilitic periostitis of the scalp and tibia, and for iritis of the left eye. For these complaints she took mercury to salivation. In January, 1844, some tubercles formed in the subcutaneous tissue of the left arm, right leg, and right side of the face. These all suppurated; that on the face was situated in the right cheek, just in front of the ear, and burst spontaneously, leaving a deep and indelible cicatrix. Four months after this, she observed her mouth to be drawn to the left side: she noticed it while she was eating; it was not preceded by any particular pain in the head. She also found that she could not close the right eye. Soon after this her right cheek became numb, and deprived of feeling. A fortnight prior to her admission into the hospital the right eyelid dropped, and she became unable to raise it. Previous to this she suffered occasionally from headache, but since then, the headache has been constant, of a darting character, and confined to the right temporal and parietal regions; deafness has also come on in the left ear, followed by a discharge, but she never experienced deafness or suffered from a discharge from the right ear.

The following is the description of her condition at the

time of her admission, as recorded by my clinical clerk of that year, Dr. Hensley.

The face is drawn to the left side, the right cheek hangs and is very flaccid; there is ptosis of the right upper eyelid, and she is likewise unable to contract the orbicularis palpebrarum muscle of the right side, cannot purse up her lips to whistle, the right cheek flaps as she speaks or attempts to blow; the muscles of the jaw on the right side are not paralysed; there is decided loss of sensation of the right cheek and numbness of the right temple, but the eyelids and the conjunctiva are sensitive. The right side of the tongue is numb, but she can protrude the tongue straight: has no perception of the bitterness of quinine on the anterior part of the right side of the tongue, but tastes it immediately on the left side or at the base of the organ.

She complains of a constant, dull, heavy pain in the right temporal and parietal region, also of a noise in her right ear on moving the jaw, and occasionally a severe pain about the right temporo-maxillary joint. Hearing of the right ear is perfect, deafness of the left complete.

The right eyeball squints outwards and a little upwards: cannot direct the eyes in harmony, so that objects appear indistinct and sometimes double; can see better with the right than the left, probably from the previous iritis of the latter.

This patient was treated with five-grain doses of iodide of potassium thrice a day; and, in consequence of there being great tenderness on pressure in the region of the right temple, an incision was made, on the 22d February, down to the bone, and through the periosteum, with immediate relief to the pain of the temple. After she had been under this treatment nine days, the sensibility of the right cheek began to return, and she was able to raise the right eyelid a little. On the 4th of March, the following report was made:—She sleeps well, can now open her right eye, has less numbness of the right cheek, which, however, still hangs; can feel when the right side of the tongue is touched: power of motion of the right eyeball is much improved. On the 18th, she could open the right eye perfectly, but could not close it.

On the 29th she was discharged, all indications of palsy of the

third and fifth nerves having disappeared, but that of the seventh remaining permanent and irremediable, this latter having been, doubtless, due to the cicatrix of the suppurated tubercle.

LECTURE XXXVII.

ON PARALYSIS.

SOFTENING OF THE BRAIN.

I PROPOSE to call your attention to two cases of brain-disease that have recently terminated fatally in the hospital, which fatal termination gives us an opportunity of ascertaining positively what were the diseased conditions before death. A fatal termination to cases of this kind, although casting opprobrium on our art, yet affords us this consolation, that the post-mortem inspection clears away that amount of uncertainty which overhangs most cases of brain-disease, in consequence of the various complicating sympathies that accompany disease of the central organ of the nervous system. In many cases of cerebral affection, while it is sufficiently easy to determine the nature of the disease, it is very difficult to decide upon its locality. It is only, therefore, by a careful observation of cases during life, and an unprejudiced comparison of the post-mortem changes with the symptoms, that we can arrive at accurate conclusions respecting the precise value of certain symptoms, or the exact interpretation of them. A clot in one hemisphere of the brain, encroaching more or less upon the corpus striatum, will produce symptoms exactly the same as those of a similar clot deep in the substance of the corresponding cerebellar hemisphere; and it is only by the observation and careful collation of numerous cases, made complete by their post-mortem examinations, that we can expect to arrive at such conclusions as may hereafter enable

us to distinguish a paralysing clot situated in the cerebrum from a similar one situated in the cerebellum.

It is curious that in heart-disease the main difficulty is not as to the situation of the lesion, but as to its precise nature. We can easily enough tell whether this or that valve is diseased, or this or that ventricle dilated; but we cannot always be so exact as regards the precise nature of the lesion. On the whole, however, we have attained in heart-cases a certainty of diagnosis far exceeding that in brain-diseases. This we owe mainly to the much greater knowledge which we can obtain respecting the living organ from auscultation, but also to post-mortem investigations. In a large majority of heart-cases, post-mortem examinations are *corroborative;* but in brain-cases, they often give us the only clear information we possess, certainly as regards the site of the lesion; but sometimes they entirely contradict our anticipations.

Case CLXV.—The first case is that of William Ware (vol. xxvi), a ploughman, from Kent, who has been in the hospital some time. He unfortunately caught erysipelas, and died of it, and we had thus an opportunity of ascertaining the precise cause of his symptoms.

This man was thirty years of age, and of temperate habits. About fifteen weeks before his admission, he was suddenly seized with paralysis of the right side of his body and face. It is not certain that he lost his consciousness at first, as he woke out of his sleep in a state of paralysis. After this, according to his own account, he lost his consciousness, and continued in that state for three weeks; but it is improbable that this unconscious condition was complete coma, for coma of three weeks' duration is very unusual. He fell, perhaps, into a lethargic state, unable, as he says, to speak distinctly, or to comprehend perfectly what was said to him, or to feed himself, and he was deprived totally of the power of the right side.

It is always important to see what state the patient was in before the attack: in most cases you will find something wrong. Our patient had complained of a numbness of the extremities of the affected side three or four days before the attack, no doubt dependent on some morbid change going on in the brain.

After the recovery of his consciousness, he continued for three weeks in a state of complete hemiplegia: he used to gape very much, and frequently cried in a childish manner, as persons thus afflicted are very apt to do. At the end of the three weeks, he began to recover the palsy, the leg first regaining power, then the arm; but as there had been no improvement for some time, he was sent up to town.

At the time of his admission into the hospital, our patient exhibited the following symptoms:—There was hemiplegic paralysis of the right side, no longer, however, complete; for he could use the leg very well, and the arm slightly. He could elevate the arm to a right angle with the trunk, and bend the elbow-joint and flex the fingers so as to grasp feebly. In protruding the tongue it deviated, as is usual, to the paralysed side; but he could move it from side to side. The face was still slightly distorted, the cheek hanging on the right side: none of this distortion, however, was due to paralysis of the facial nerve; for all the movements of the superficial muscles were perfect: he could wink, and frown, and whistle; and when he laughed, the distortion of his face was not nearly so great as you find it when the portio dura nerve is affected. The facial palsy was due to paralysis of the buccal nerve of the fifth pair affecting the buccinator muscle. The sensibility of the affected side was slightly impaired. The muscles of the paralysed limbs were, as usual, somewhat wasted: they were, however, evidently recovering their state of tone gradually, and they were free from rigidity. Galvanism had been used several times, more experimentally than curatively, and the man was encouraged to take as much exercise as he could without fatigue. With the notion of reducing any inflammatory process which might be going on at the seat of cerebral lesion, we gave him mercury; and while he was in a state of salivation he was exposed to the contagion of erysipelas, and was seized with that disease in a very severe form, and died.

The symptoms justify a diagnosis with regard to locality. As the paralysis was very complete, and as the motor power only was affected, it seemed in the highest degree probable that the lesion of the brain was situated in some part which exercises an important influence on voluntary motion. Now there are the

best reasons for believing that no part is so intimately connected with this function as the corpus striatum. In a case, then, like the present, in which the paralysis of motion had been complete, without any considerable injury to sensation, the lesion would most probably be seated in the corpus striatum, or in that part of the hemisphere which is most intimately connected with it; and of the two corpora striata we would fix on that of the left side, since the paralysis is shown on the opposite side to that in which the cerebral disease exists; and here the paralysis was on the right side.

Taking, then, this *locality* as granted, what was the nature of the disease? It might either be occasioned by a clot of blood from a sudden rupture of a blood-vessel, *i. e.* apoplexy, or a more slow disease, which, however, was of such a nature as to be capable of producing sudden paralysis. The suddenness of the attack, and the succeeding loss of consciousness, favour the former supposition. But it is wrong to suppose that apoplexy is the only cause which can produce this sudden paralysis (by apoplexy I mean the rupture of a blood-vessel, and the consequent escape of blood into the brain)—there may be other causes. 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 impaired way perhaps, but not sufficiently so as to call attention to it. Suddenly, probably from some temporary excitement, the fibres give way, and the power of voluntary motion is lost, as suddenly as the galvanic current ceases on breaking the circuit. This is one cause of paralysis in cases where there is no apoplexy.

It is now clearly established that such a lesion may ofteu exist alone. It is, likewise, very frequently—I incline, indeed, to believe almost always—the precursor of apoplexy; and, therefore, we frequently find in these patches of white softening one or more clots of blood of various sizes. The artery or arteries leading to the part are diseased; that portion of the brain fails in its nutrition; it passes into the state of white softening; and the minute vessels, losing the support which they must receive from the firm brain-texture, and being them-

selves often more or less diseased, give way, and allow the blood to escape into the tissue of the brain.*

There is another way in which paralysis may take place—viz., by an inflammatory state of the brain-substance, which produces softening, although of a different kind to that which I have just described, in which there may or may not be rupture of fibres; but there may be deposit as the result of the inflammation, which, by its pressure, may interfere with the conducting power of the fibre.

Now I have mentioned the conditions of *white* and *red* softening, or as they might be better called, *atrophic* and *inflammatory*. These are apt to come on under opposite circumstances. *White* softening may be caused by anything that diminishes or cuts off the supply of blood to the head. In some cases, in which the carotid artery has been tied for aneurism, and the supply of blood to the brain on that side thus stopped, the patient may go on very well for a short time, and there may be no impairment of function; but during that time the process of softening has been gradually proceeding on the side of the brain which is supplied by that carotid; and in a day or two the softened fibres cease to maintain their continuity, give way, and paralysis ensues.

Some years ago I attended, along with my friend Mr. Street, of Norwood, a very remarkable case of this sort. The supply of blood had been cut off by a dissecting aneurism, which had plugged up the common carotid artery on the right side, and paralysis on the left side of the body took place. We were much puzzled to account for the paralysis till after death, when the post-mortem examination cleared it up. The account of this case was published in the twenty-seventh volume of the 'Medico-Chirurgical Transactions.'

There is abundant evidence to show that under ordinary circumstances white softening is *atrophic, i. e.* dependent on imperfect nourishment of the brain, and non-inflammatory. But it may exist on the confines of genuine inflammation, the nutrient fluid being diverted from it to the inflamed portion, or it may exist around an effusion of liquid in the ventricles, that

* See further on this subject, Lects. XLII, XLIII, XLIV.

effusion being possibly inflammatory in its nature, or around an indurated portion of brain, or a tumour.

The second kind, the *red* softening, is inflammatory. I show you here a very good representation of it, which was made from a case of mine some time since. The portion of the brain affected was of considerable extent; there was paralysis, of course, on the opposite side, a stupid, comatose condition, but not complete coma, and loss of sensibility, which continued till the patient died. But the paralysis came on gradually, and not till after these comatose symptoms had existed some time.

Now of these two conditions I think we may very justly attribute the symptoms in our patient Ware's case to a white softening, followed by a rupture of blood-vessels and an effusion of blood.

The numbness, which he described as having occurred before the paralysis, indicated that morbid changes were slowly going on, and after a time rupture of fibres, and giving way of the blood-vessels, took place, when followed the paralysis and the three weeks' stupor. The former, being very complete, denoted a solution of the continuity in some part of the centre of volition; the sudden stupor implied that pressure existed within the cranium, but only to a moderate extent, for a large effusion, capable of producing such a complete paralysis, would undoubtedly have caused profound coma.

This, I say, seems the *most probable* train of morbid processes at the commencement of the case; but we cannot speak on this subject with great certainty, for we cannot fully depend on the accuracy of the patient's history of himself. It tallies, however, very well with what we observed of his subsequent history, and at the post-mortem examination.

A train of phenomena, however, very similar, but differing as regards the *sudden* supervention of the paralytic state, might have occurred, when the primary lesion was of the inflammatory kind.

Upon examination of the brain after death, it was found that a considerable portion of the corpus striatum of the left side was completely destroyed and excavated, and that the cavity was filled with a creamy fluid, having somewhat the appearance of pus: the anterior and inner part of the corpus striatum

was healthy, but the whole of the posterior and outer part was thus disorganized: a few bands of fibres, easily broken down, passed from one side of the cavity to the other. The creamy matter contained in the cavity was found on microscopical examination to contain great numbers of large cells, containing oily matter in large globules, and also in a state of extremely minute subdivision. These curious collections of oil-globules might suggest the idea, that some active proeess had been going on during life. What their precise signification is, I do not pretend to determine; but I think I may affirm that they are characteristic of a state of white softening of some duration, as I have found them in other cases, in which no doubt could exist of the atrophic nature of the lesion.

It seems almost certain, then, that this excavation of a portion of the corpus striatum must have been due to a white softening, followed by an effusion of blood, and which was the immediate cause of the breaking down of the fibres and of the pressure which gave rise to the paralysis and coma. No traces of blood remained in the diseased part, as no doubt there had been ample time for the disintegration of its particles, and for its absorption.

At first the paralysis was very complete, but afterwards the patient recovered a certain amount of power, especially in the lower extremity. If the paralysis had been accompanied with rigidity, I should have been led to the conclusion that the cerebral lesion was of an irritating nature. This rigid state of the paralysed limb (*when it comes on at the same time as, or very soon after, the paralysis*) is generally seen when some superficial part is affected, as the meninges or the surface of the brain, or when there is a growth from the skull, or a tumour in the hemispheres, in some cases of inflammatory softening, or in some conditions keeping up a constant irritation; but when there is simple rupture of the fibres of a deep-seated part of the brain, as the corpus striatum, with or without pressure, there is no irritation, and the paralysed muscles are quite lax.

There was no appearance whatever of rigidity in the muscles of the paralysed limbs. Now this is just the sort of paralysis accompanied as it was by comatose symptoms) which would

arise from a solution of continuity of fibres in the first instance, and from compression. A portion of the corpus striatum is destroyed, and the healthy part is compressed by the effused blood. On the absorption of the latter a certain amount of power had returned in the limbs; and it depended on the possibility of repairing the broken-down portion, whether a complete restoration would take place. That parts so much diseased would have ever been completely repaired seems extremely unlikely, for two reasons; first, because the arteries of the part did not seem in a perfectly healthy condition; and, secondly, because of the well-known tardiness of all reparative processes in the brain, where actual solution of continuity has taken place.

You remember that on several occasions we passed the galvanic current through the paralytic and the sound limbs in this case. This was done, as I explained to you at the time, for the purpose of ascertaining whether any irritant disease existed within the cranium at the seat of the paralysing lesion.

If, on passing the galvanic current, you produce less contraction in the paralysed than in the sound side, then you may judge the cause to be of a *depressing* kind: if, on the other hand, the contractions in the diseased limb are the strongest, then you may conclude that the condition of the centre which causes the paralysis is *irritative*. But then you must bear in mind that irritation is not always inflammatory.

The most important points in this case may be thus summed up:

First, through some diseased state of the assimilative process, the arteries of the brain become diseased, and an insidious, gradual process of softening takes place; rupture of one or more blood-vessels follows upon this, with solution of continuity of fibres, and compression of the neighbouring healthy brain-structure; after this we have absorption of the clot, and more or less attempt at reparation: it may be that some inflammation may take place around the clots, which may retard the process of recovery.

If you were called upon to treat a case of this kind from the

beginning, what course would you adopt? If you can clearly make out that the lesion is not inflammatory, but, on the contrary, due to defective nutrition, the less you interfere the better. Keep the patient in the horizontal position; let the head be kept cool; unload the bowels in such a way as will involve the least effort on the part of the patient: a large turpentine and castor-oil clyster is generally more expeditious than purgatives given by the mouth; but it may be often advisable to give such a purgative as croton-oil, which is easily introduced into the system, and operates freely and quickly. Bleeding is not advisable in such cases as this, as it tends to increase the atrophic condition of the brain, and would, under such circumstances, favour rather than repress hæmorrhage.

When this patient came under treatment in the hospital, we did not at first administer any medicinal agent: he was kept quiet and nourished moderately. After one or two trials with the galvanism we observed, on one occasion, a little more excitability in the palsied limbs than in the sound ones under the influence of the inverse current. Finding that he had made no progress for some days, I was led, from this effect of the galvanism, to fear that some inflammation might have been set up around the lesion; and, accordingly, I was induced to give him drachm doses of the solution of the bichloride of mercury—that is, as you know, the sixteenth of a grain of the salt, three times a day. No good effect following this treatment; on the contrary, I fear the salivation, which took place sooner than might have been expected, may have made him more susceptible of the poison of erysipelas.

After all, I must acknowledge that in this part of the treatment somewhat of the *nimia medici diligentia* was exhibited. It is a lesson hard to learn, and more difficult to act upon, that nature can do more than the physician; but it is a lesson which each succeeding year of increasing experience will impress upon you, and in no cases more than in those of chronic affections of the brain.

I had intended to have brought under your notice to-day another case of the same nature as Ware's, in which apoplectic effusions took place on both sides; but, as I fear I could not do

so without making this lecture too long, I shall reserve this case for my next lecture.

LECTURE XXXVIII.

ON A CASE OF WHITE OR ATROPHIC SOFTENING OF THE BRAIN, CAUSING DOUBLE APOPLEXY.

I PROPOSE to-day to offer you some remarks on the second case which was to have formed part of the subject of my last lecture. It is one of the same nature as that on which I last commented, and it serves to illustrate the mode in which softening of the brain is apt to take place, and the way in which that disease favours the production of apoplexy. We are fortunate in having a very full account of the case, taken with the most praiseworthy precision, by my clinical clerk, Dr. Vaux.

CASE CLXVI.—The subject of this case was named George Regan, aged fifty-nine (vol. xxiv); and was admitted into Sutherland Ward, 20th of January, 1849. He was a glass-cutter by trade, and had lived well all his life; he called himself temperate, but admitted that he had been in the habit of drinking a good deal of beer and spirits.

This man seems to have suffered long from what he called rheumatism, but which, from his habits and his age, I should think was rather of the nature of gout; he had, however, no regular fit of gout, but complained of pains in various situations, and for these he was for some time an out-patient at the hospital.

About two years before his admission, he was seized suddenly while at work, at nine o'clock in the morning, with a feeling of stupor; as he happened to be working at home at the time, he laid down on his bed, hoping to sleep it off by dinner-time; but when he attempted to get up, he fell, and found that he had

lost the use of his right side; he then became comatose, and so continued for some days. Whilst in a state of insensibility he was taken to a neighbouring hospital, where he remained for two or three months, and at the end of that time in some degree recovered from his attack, but not so as to enable him to work as he had done before his illness.

During last October he had a *second* attack, of the same kind as the first, but less severe; the limbs were paralysed, as before, on the right side; he remained a few days in bed, and recovered in some degree the use of the arm and leg, but he has never been able to work since.

On Christmas day last, at nine o'clock in the morning, before he had risen, he had a *third* attack; on this occasion he was delirious for a short time, and afterwards became insensible, but his consciousness returned in the evening.

This third attack brought on an increase of the paralysis on the right side, so that on this occasion not only was the motor power affected, but the sensibility likewise. This latter function was so far affected that he could not pick up small objects, and he would frequently let fall things which he meant to retain in his hand, and he staggered when he attempted to walk. It was more than three weeks after this attack when he was admitted, and the paralytic state had not improved; on the contrary, he thought it worse. His condition on admission was as follows: there was imperfect palsy of the right side, as shown by slight ptosis of the right upper eyelid, and a slight hanging of the cheek, the features being a little drawn to the *left* side. He would protrude his tongue straight, but his articulation seemed somewhat difficult. In walking, he slightly dragged the right leg; the grasp of the right hand was moderately firm, but not *so* firm as that of the left; the muscles of the right leg and arm were flaccid, and less nourished than on the left side. The impulse of the heart was very strong, and a mitral systolic bellows sound could be distinctly heard.

For some days after his admission he suffered very much from constipated bowels, and it was necessary to give the strongest purgatives. He then began to complain of heaviness of the head, a disposition in his thoughts to wander, and a difficulty in collecting them. These symptoms appeared to me to portend

the approach of another attack, such as he had on three former occasions. His remarkably sallow complexion and general leucophlegmatic appearance led me to suspect the existence of renal disease, resulting from that particular form of kidney—small and atrophied—which so frequently accompanies a gouty condition. This opinion was confirmed by the characters of the urine, which was pale, of low specific gravity, and slightly, but distinctly, albuminous. With this view, and imagining that the uneliminated urea might be contaminating the blood and affecting the brain, I ordered him to be freely blistered at the back of the neck. This seems to have somewhat relieved him, for the next day he was reported to feel rather easier as to his head, and to have more power over his thoughts. At nine o'clock, however, on the following morning, the house-physician, Dr. Armitage, was called to him, in consequence of his having been suddenly seized with a fit; he found him completely paralysed on the *left* side, both as to sensibility and voluntary motion; his left eye squinted, and was twisted downwards and inwards; at every expiration his left cheek puffed out, from want of power of the buccinator; the right leg and arm moved when pricked, but the left remained motionless; his breathing became louder and more stertorous, his coma deeper, and at last he died.

There was in the attacks to which this patient was subject a curious combination of the epileptic and apoplectic, the one following upon the other. At the foundation of them, no doubt, was the diseased state of kidney. The first attack of sudden stupor was probably a slight epileptic seizure, the effect of which was a disturbance in the circulation of the brain, and the giving way of some small vessels—a slight apoplexy, with compression and rupture of fibres, and consequent paralysis.

Whilst the one or two small clots which had been effused on this occasion were undergoing absorption, and some attempt at reparation was taking place, he had another seizure of the epileptic kind—a further disturbance of the circulation and nutrition of the brain on the same side, probably in the corpus striatum, or among the fibres which pass from it to the hemisphere. But as the paralytic state appears to have been only

slightly increased, and as the coma was of short duration, it is not likely that any effusion of blood took place on this occasion.

The epileptic character of the third attack was manifested in the delirium with which it was ushered in, and which ended in coma. There was a decidedly increased paralysis after this attack; but since quite as much paralysis is apt to follow the simple epileptic seizure, it by no means follows that any effusion of blood took place, although it is probable that such must have been the case, from the fact that the palsy showed no signs of improvement, as is generally the case with the epileptic palsy. As this attack occurred at a time when Christmas festivities are more or less prevalent with all ranks and classes, it is very likely that the immediate exciting cause of the attack was due to over-indulgence of some kind.

The fourth attack had in its premonitory signs all the characters of a threatening epileptic paroxysm; and knowing, as we did, the existence of renal disease, we were prepared for it. The epileptic coma, however, soon passed into the profounder coma of a compressed brain; and as a new hemiplegic paralysis of a very complete kind showed itself on the left side instead of the right, it was easy to infer that a new and extensive apoplectic effusion must have taken place on the right side of the brain.

Assuming that the first three attacks were epileptic, followed by an apoplectic effusion, and that this effusion was due to a weakened condition of the arterial coats as the result of disease, it was quite consonant with experience to attribute the fourth attack to a similar cause, and to infer that arteries similarly diseased had given way on the right side of the brain. Most cases of rupture of vessels in the brain, at the age of this patient, take place from disease of the arteries; and it is very common, as was first pointed out by Bizot, for the arteries of the brain to be affected in a symmetrical manner, *i. e.* corresponding arteries of opposite sides will be similarly diseased, and to nearly the same extent. In this way disease had been, for some time, making progress in this man's brain symmetrically; there was first palsy of the right side, and then a similar condition of the left; and this simple fact of symmetry

pointed to the arterial system as the seat of disease, and therefore to its usual result—apoplexy.

On opening the body we found there was a double apoplexy corresponding to the double paralysis—one of long standing, and one recent—the former on the left side, the latter on the right. The original hæmorrhage affected the corpus striatum and optic thalamus on the left side; and it was evident that the hæmorrhage on this side did not take place at once, but on two occasions at least. The substance of the corpus striatum had evidently been the seat of the older effusion. It exhibited on section several bloody clots, and that peculiar yellow discoloration which always succeeds to a hæmorrhage. The more recent effusion was a clot which had formed quite on the surface of the optic thalamus, extending likewise to the corpus striatum, and lodged in a depression on the surface of those bodies. We had thus an explanation of the cause of the original paralysis of the left side; and its imperfect nature was plainly due to the fact, that there was but little destruction of the corpus striatum, and that the compression of the later clot affected chiefly the optic thalamus, and but slightly the corpus striatum.

The apoplectic effusion on the right side was much more extensive; it involved parts corresponding to those affected in the first seizures, but to a much greater extent; and the brain-substance was more completely torn up and destroyed. The clot was very large; it filled the right lateral ventricle, and even broke through its roof, and tore up the white substance of the cerebral hemisphere; it likewise broke down the corpus striatum, optic thalamus, and the septum lucidum, all which parts were completely swept away. This remarkable destruction was no doubt owing to a previous diseased state of the brain. I mentioned to you in my last lecture that it is very common for apoplectic effusions to be preceded by white softening; and when the softening has got to a certain extent, the brain substance no longer affords an adequate support to the vessels, which, themselves more or less enfeebled by disease, give way, not in one point only, but in many, and the abundant effusion ploughs up the softened matter, quite destroys it, and takes its place; all this was the case in the present instance. The arteries of the brain were very generally dis-

eased on both sides, and exhibited that symmetry to which I have alluded.

We found likewise, as had been anticipated, a diseased state of the kidneys; they were very much contracted, the cortical substance wasted, fissured, and granulated on its surface, the tubular substance healthy, and the capsule thickened. This condition of kidney, formerly described as the third stage of Bright's disease, is, in reality, a chronic degeneration or wasting of the kidneys, due to a deranged and damaged nutrition, for which I should be glad to find some other name than chronic nephritis. I have called it *gouty kidney*;* and in this and many other cases this name is very appropriate. But it occurs in cases where there is no evidence of gout. The result of the disease is to render the kidneys imperfect emunctories for the elimination of the urea and other elements of the urine, which accumulate in the blood, and give rise to various morbid changes throughout the body, and are especially mischievous to the functions of the brain.

The heart and the arterial system were likewise extensively diseased. In the coats of the arteries were very numerous deposits of atheromatous matter. The heart was much dilated and hypertrophied, especially as regards the left ventricle. The fibrous basis of the valves was extensively thickened, apparently by some deposit, which rendered it opaque, and impaired its flexibility; and as is generally the case in this particular form of disease, *all* the valves were altered in this way; those of the right side, however, being much less diseased than those of the left. The semilunar valves of the arteries, especially of the aorta, had the fibrous festoons at their basis much thickened, the curtain of each valve being very little affected, and its functions, therefore, not impaired; and the cordæ tendineæ of the mitral valve were much thickened, and somewhat shortened, and the curtains of the valve also thickened. Similar changes had taken place in the tricuspid valve and its tendinous cords, but to a much less extent.

Taking, then, a general retrospect of this case, we find there is quite enough to account for all we have seen. The sequence

* See on this subject Lecture XXVIII, on Gouty Kidney.

of the events may be thus described:—First, the man gets into a general gouty condition, and the elimination of this morbid material gives rise to an irritation of the kidney, which at length assumes the form of gouty kidney, or, if you will, chronic nephritis; and the kidney, thus damaged and incapacitated for the perfect discharge of its function, is the great promoter of all the subsequent evils; the blood becomes still further contaminated, additional deposits to those which doubtless had already formed take place in the tissue of the heart's valves, in the large systemic arteries, and in those of the brain; the diseased arteries of the brain become insufficient channels of supply; white softening is the consequence, and many of the unsupported and unhealthy capillaries at length give way; and thus all the circumstances, from first to last, fall in regular order as cause and effect.

The deposits in the arteries produce a twofold influence upon the circulation—by roughening the inner surface of the arterial channels, they create a certain amount of direct obstacle to the flow of blood from the ventricle; and by diminishing, or nearly destroying, the elasticity of the arterial walls, they impair one of the most important forces by which the circulation is carried on in the arterial system. Thus the arteries, from being elastic, yielding channels, with perfectly smooth inner surfaces, are changed into resisting, inert tubes, with rough interiors. It is plain, then, that, under these circumstances, the heart has to encounter great obstacles, and to do a great deal more work than when the arteries are in their normal state; hence the dilatation of the cavities of this organ, caused by the obstacle to the free flow of the blood; and the hypertrophy, by the greater exercise and effort of its muscular fibres. The increase of force in the heart's action, however, is merely remedial, to meet the increase of obstacle, and is one of those beautiful instances of self-adaptation to change of circumstances which the animal organism, especially the muscular system, so abundantly exhibits.

As these deposits go on, they impair the structure of the arteries of the brain; the degenerated walls of these vessels become less strong, and less able to support their contents. And I would particularly impress upon you, that there is no

excessive determination of blood to the brain in these cases, but the reverse; for the blood that goes to the head has, in the erect posture, to be pumped up against the force of gravity; and, therefore, any obstacle in the course of the arteries would tell more in this than in any other direction. It is a common notion, too easily accepted by many excellent practical writers, that hypertrophy of the heart gives rise to apoplexy, by sending the blood with an undue impulse to the head; but for the correction of this error we need only remember that the additional force is merely such as is necessary for the exigencies of the circulation, and such as shall preserve the force of the blood's current as nearly as possible to the normal point, in spite of the existing obstruction. Indeed, the actual force with which the blood circulates in the morbid arteries is, most probably, less than in health. The apoplexy is, in fact, due to the diseased state of the arteries, which renders their walls an inadequate support to their contents, and to the diseased state of brain, which imperfectly supports the arteries.

A vast number of the cases of apoplexy which occur about the period of life of our patient, or after the age of fifty, are of this kind—a fact that has an obvious and an important bearing upon the question of treatment.

There is a practice, unfortunately too common, but which, I think, is every day becoming less so—namely, that of following an attack of apoplexy, very much as a matter of course, by depletive measures. However, it may be, that some justification may be found for such a mode of treatment in the cases of strong, young, hale, and plethoric subjects, I presume no one will say that it is very well adapted to patients who have passed the meridian of life, whose blood and tissues are more or less contaminated by morbid matters, and with whom a diseased state of the arteries of the brain has already greatly weakened the nutrition of that organ. The case, indeed, which I have just detailed to you is one of many which proclaim loudly, that a depletory system ought not to be pursued indiscriminately, or even generally, in apoplectic cases.

With reference to this question of depletion in apoplexy, let me refer you to an interesting and very useful work by Mr. Copeman, in which he has collected, from a great variety of

sources, a large number of cases which presented the symptoms of apoplexy. Of 155 cases in which the treatment was specified, 129 were bled, and only 26 were not. Of the 129 persons who were bled, 51 recovered, and 78 died—the recoveries being about 1 in $2\frac{1}{2}$, the deaths 1 in $1\frac{2}{3}$. Of the 26 who were not bled, 18 survived, and 8 died, the proportion of recoveries being 1 in $1\frac{1}{2}$, and of deaths, 1 in $3\frac{1}{4}$. Eighty-five of the cases were bled generally and copiously, and of these only 28 recovered, and 57 died —in other words, two in every three cases terminated fatally. I am quite aware that the small number of cases not bled casts some doubt on the validity of the conclusion to be drawn from the comparison of the results of the treatment. But the fact that considerably more than half of those treated by bleeding died (and we owe much to the industry of Mr. Copeman for bringing it out), is a highly significant one, and should arrest attention.*

I hope I have now said enough to convince you that the treatment of patients with apoplectic symptoms must not be regarded as a matter of routine, but as a question of grave import, and which demands the most anxious consideration of the practitioner. Let me add, that it sometimes requires the exercise of no small courage and self-possession to resist adopting that practice; for the popular feeling, led by a formerly too prevalent medical practice, is entirely in favour of it, and would readily condemn a practitioner as guilty of the death of his patient who suffered him to die unbled. It is a far more dashing and courageous thing to open a vein on the spot and in the presence of a number of anxious friends, than to adopt less showy, and apparently less active, measures.

But, indeed, you need not be inactive, even if you decide against adopting the plan of bleeding. Having placed your patient in an easy position, in which no excitement of muscular action is likely to take place (for you must bear in mind that reflex actions may often be readily excited in these apoplectic cases), you should immediately direct your attention to the state of the stomach and intestinal canal. Sometimes in these cases the stomach is overloaded, or the bowels are confined, and the

* A Collection of Cases of Apoplexy, by E. Copeman, 1845.

administration of a quickly acting emetic, or even of some purgative medicine, will often provoke a moderate sickness, which unloads the former. Nor can there be any objection to adopting measures to clear out the bowels, either by an active purgative administered by the mouth, such as calomel or croton oil, or by a stimulating and purgative enema, or both.

If, upon full inquiry into all the particulars of the case, you find that your patient is of plethoric habit, with too much blood in his body, and with a sufficiently strong heart, you may bleed him with reasonable chance of benefit; but if he has been of intemperate habits, is labouring under organic disease of the heart and arteries, is of gouty or rheumatic constitution, then, whatever popular or medical custom may say, my advice to you is, hesitate much before you deplete by bleeding.

The objects which it is proposed to gain by bleeding are a diminution of the cerebral congestion, and the stoppage of the hæmorrhage into the brain; and where it is quite plain that cerebral congestion does exist, and that that congestion causes the cerebral hæmorrhage, this is clearly a rational practice. But you must bear in mind that in a large number of the cases—probably the majority—there is in reality no cerebral congestion, and that the hæmorrhage is of a kind not likely to be stopped by taking away blood—by establishing another hæmorrhage elsewhere.

On the whole, then, I think that the results of experience denote that the majority of cases of apoplexy are best treated by purging, shaving the head and keeping it cool—perhaps blistering; that bleeding is rarely applicable, except to the young, vigorous, strong, and plethoric; and that the practitioner should aim more at keeping the heart's action quiet than at any other single indication.

LECTURE XXXIX.

ON PARALYSIS.

ON SOFTENING OF THE BRAIN.

Gentlemen,—We can illustrate to-day the influence of disease of the brain in the production of paralysis, and also the pathological changes in that organ which are capable of cutting off the influence of the will from a large portion of the body. For this purpose I shall request your attention to the particulars of three cases.

The first case ran so short a course that I fear many of you missed the opportunity of seeing the patient during life. The facts observed in the post-mortem examination of this case are of especial interest, inasmuch as they afford an explanation of the early changes which take place in the brain's structure, leading ultimately to such alterations as favour rupture of the blood-vessels, and the consequent escape of blood, in greater or less quantity, into the surrounding texture, constituting that condition which is called Apoplexy, or Sanguineous Apoplexy.

Case CLXVII.—The patient, by name William Thurston (P.M. Book, page 15), was an auctioneer's porter, a muscular, well-nourished man, about the age of fifty; he had always been of intemperate habits, and about six months before the fit had lost his wife; under the influence of an ill-regulated grief he was led to yield too much to self-indulgence, and to console himself with the bottle. On the morning before his admission he fell senseless on the floor, but it did not appear that he bit his tongue during the fit. In about half an hour he recovered so much as to be able to attend to his business. It seems most probable that this was an epileptic fit, which, however, wanted the very characteristic feature of biting the tongue. That

symptom, however, is not always present, and, although when it occurs along with the other symptoms, it may be regarded as pathognomonic of the epileptic fit, its absence by no means proves that the attack was not of that nature. The fact, that a patient has bitten his tongue in the attack, shows in a striking point of view the profound insensibility that accompanies the fit; for, in the insensibility of the patient to pain, the tongue is not withdrawn from between the teeth, and so it gets bitten by the violent and spasmodic closure of the jaws. In the course of the day, while at his work, our patient fell down in another fit, in which he remained quite unconscious, but did not struggle. He was brought to the hospital in a state of profound coma, at four o'clock p.m.; his eyes were shut, and the pupils were quite insensible to the stimulus of light. The fall was so sudden and heavy that he bruised his chin, and loosened four of his teeth.

He lay in a totally unconscious state, and the breathing was accompanied with a snoring noise, not of the loudest kind. The pulse and respiration were not particularly rapid at this time, nor could any evidence of a paralysed condition of either side of the body be obtained. So things went on until another fit commenced, in about an hour after his admission, which was accompanied by violent spasmodic action of the muscles of the whole body, affecting chiefly those of the lower extremities. There was no indication that the convulsions were greater on one side of the body than on the other; this point was so closely watched, that if there were any difference it must have been very slight. The convulsions were accompanied by an acceleration of the respiration, and by increased noise in the throat; in about ten minutes the patient was quiet again, but in an hour more the same train of phenomena was repeated, with still more noise accompanying inspiration, and a puffing out of the cheek at each expiration. After this fourth fit several other attacks manifested themselves at still shorter intervals, and he died in the night, in a very exhausted state.

Now we often meet with phenomena such as have been exhibited by this patient, without being able to find any post-mortem appearances which will explain their occurrence; hence I gave a doubtful diagnosis. Before the body was opened, I stated that we should find one of three conditions—first, a super-

ficial clot extending over a great part of the surface of the brain; or, secondly, a clot in the arachnoid sac, or between the dura mater and the bone, or even between the layers of the dura mater; or, thirdly, a state of brain apparently normal, which would be quite insufficient in itself to account for the symptoms during life, in which case the epileptic condition would probably be found to have been associated with disease of the kidney. We were not able to determine the condition of the kidneys during life, as we had no opportunity of examining the urine. The difficulty of the diagnosis was much increased by the fact that one side of the body did not appear to have been more affected than the other. This point was carefully attended to by those who watched the patient whilst he was suffering from the convulsions. Had the convulsions been greater on one side, one would have been led to the inference that a clot was present in the opposite side of the brain; but in the absence of such evidence I was quite prepared to find that the convulsions were due to the change in the state of the blood which is induced by kidney disease. The symptoms, however, only extended over a period of twelve hours, which is a much shorter time than the epileptic attacks depending upon kidney disease generally take to run their course. Such cases usually last much longer (unless the fits are very violent and at short intervals) than those in which cerebral hæmorrhage has taken place; the quantity of blood effused, which is often very considerable, tending, in the latter cases in some measure to expedite the fatal result.

Let me, however, describe the post-mortem appearances in detail.

The dura mater was remarkably adherent to the calvaria, over the situation of the Pacchionian bodies, a condition which we very frequently meet with in persons who are much addicted to drink, or are prone to violent fits of passion, and in these cases the Pacchionian bodies themselves are often observed to be unusually large.

Upon cutting through the dura mater a large clot was found in the cavity of the arachnoid, about the situation of the squamous portion of the left temporal bone. In the posterior part of the head a much larger quantity of blood was found effused, chiefly between the hemispheres, and on the left side of the falx.

The quantity could not have been less than two ounces. The pia mater over the whole surface of the brain was found to be highly injected with blood.

Now comes the most interesting by far of all the appearances which we noticed. On the superior surface of the right hemisphere, one or two circumscribed patches of gray matter were observed to be of a much darker colour than the surrounding parts. On closely examining these, it was found that the colour was due to the presence of a number of minute red points, which under the microscope were found to consist of little extravasations of blood into the gray matter of the brain. Upon submitting portions of these dark patches to microscopical examination, the capillaries were found to have undergone fatty degeneration, the deposits of fat (consisting of aggregations of very minute oil-globules) being in the walls, and arranged in alternate patches on the sides of the vessels, apparently in the situation of development cells. Of this appearance, I show you an excellent sketch by my late clinical clerk, Mr. Curme. The white matter of the brain appeared to be healthy and of a natural consistence. The ventricles were free from effusion, as was also the base of the brain.

The depositions of fatty matter in the minute vessels may interfere mechanically with the passage of the blood through them, and they would certainly interfere with the development of the force of vital contractility, which, as you know, is present in healthy vessels, and upon which the strength of these vessels mainly depends.

No more important observation has been made of late years in minute anatomy than that which showed that the minute blood-vessels are apt to become the seat of an atrophic process, in which the normal tissue (probably the muscular) of the capillary walls is replaced in great part by fat. It had long been noticed that the larger blood-vessels were more or less diseased in cases in which hæmorrhage had occurred on or into the brain; but it was evident that the hæmorrhage did not depend on the rupture of a large vessel; it had all the characters of a bleeding which came from several minute points, and had merged into one great clot. The sources of these small hæmorrhages are well illustrated in the condition which I have described in the right

hemisphere of this patient. This fatty degeneration of the capillary vessels will hereafter take its place with other degenerations of a similar character which modern research has brought to light, such as the fatty disease of the kidney, one of the forms of disease of that organ in which albuminous urine occurs; such, likewise, as fatty degeneration of the muscular fibres of the heart, in which many a case of broken heart originates.

In looking into the pathology of this condition of the minute blood-vessels, you may fairly ask, is it, indeed, the primary and essential disease? or is it not rather the effect of a depraved nutrition of the tissues to which these canals are the carriers of nutriment? Now, a little reflection would lead you to say that both these questions may be answered in the affirmative, and that the evil of which we speak may either commence in the brain-tissues (the brain-fibre or the brain-cell), which consequently cease to draw upon the blood-vessels to their wonted extent; and the blood-vessels, therefore, deliver to them their nutrient matter in diminished quantity; their activity is, consequently, proportionably diminished, and an atrophic state ensues. Or the evil may commence in the blood-vessels, and this will probably be the favorite view. We know that the larger vessels are frequently the seat of numerous atheromatous deposits, and not only the cerebral vessels, but the aorta itself, and the radial arteries, as we can often learn by feeling the pulse in the living person. Why should not this atheromatous state extend to the minutest vessels? I do not express any strong opinion in favour of either of these views; but I would say that it seems to me that pathologists do not enough regard the tissue itself as being the starting point of morbid change; and are too apt to overlook the fact, that the power of attraction of the tissue for certain elements of the blood is not the least important agent in determining the greater or less flow of blood to the organ or tissue.

And, indeed, whichever of these two views we adopt, who will say that in the blood itself we do not find the chief source of evil, which may tell either upon the blood-vessels or upon the elements of the tissues?

Without waiting to decide this point, we may lay it down that, whether in consequence of disease of the blood-vessels or

otherwise, the nutrition of the brain becomes impaired, and this shows itself mainly in an altered consistence of the nervous matter. Its colour does not undergo any appreciable alteration, and if you look at a portion through the microscope you will not detect any obvious change; the vesicles remain the same and the fibres remain unchanged; but, as I have said, the consistence is diminished, instead of being firm, the tissue of the organ has become soft and pulpy, and in some cases almost diffluent and of the consistence of cream. The diseased blood-vessels lie in the midst of this pulpy mass for some time without undergoing any further change, but sooner or later, under some mental emotion, or during some increased heart's action depending either upon a strong mental emotion or an increased mental effort of any kind, or upon derangement of the digestive organs or upon some bodily exertion, the blood is sent with an undue force, or in unusual quantity, into the vessels, and, in consequence, the vascular canals in the pulpy portion of the cerebral tissue, being deprived of their usual support, give way, and blood is effused into the softened part of the brain, which it breaks up, and the more readily in consequence of its already diminished consistence.* This is the *rationale* of the development of many an attack of apoplexy, from which the patient may or may not recover, according to the extent of brain previously softened, and according to the amount of blood effused.

In the case of Thurston, the presence of the red points above described afforded evidence of the occurrence of small extravasations. Other vessels in the pia mater were doubtless in a similar condition to those of the right side, and these giving way in the manner I just now referred to, led to the extensive effusion of blood which we met with on the surface of the brain. Had the extravasations been limited to the dark patches of gray matter, I should imagine that the attack would have been but slight and transient, and the patient might for a time, at least, have recovered. Such cases we often meet with, when the only symptom is a transient confusion of ideas, or perhaps

* An epileptic fit, by retarding the venous circulation, increases the blood-pressure in the arteries, and is, I have no doubt, a very common cause of cerebral hæmorrhage.

only a sensation of faintness or giddiness; after a lapse of a longer or shorter period, this slight attack is followed by one of a more serious nature, in which loss of consciousness, or paralysis, or both, may occur.

We had no evidence in this case, during life, that any paralysis existed, or that the convulsions were excited more on one side than on the other. This was sufficiently explained by the post-mortem appearances. The lesion was too much on the surface to cause paralysis, and the compression caused by the effusion of blood was exerted over an extended surface, but not to any great depth. The clot, by extending between the hemispheres, compressed both, and although it was larger over the left hemisphere, the right was more diseased, and was itself the seat of small effusions in some of its convolutions.

In such cases as this, the paralysis, when it occurs, will be due to the solution of continuity of fibres connected with the corpus striatum, or optic thalamus, or to compression, direct or indirect, of those bodies; the loss of consciousness will arise from the effusion of blood, and the consequent compression of and shock to sound parts of the brain. It is, I think, not unreasonable to suppose that a simple solution of continuity of brain-fibre may occur without any extravasation of blood; and there are cases of paralysis which occur under these circumstances, to which I shall take another opportunity of calling your attention. But I suspect that, in every case where consciousness is affected, and where more or less of coma takes place, there will be found more or less of extravasation of blood, excepting always uncomplicated cases of renal disease and of true epilepsy, or cases in which the softening involves a very large portion of the convoluted surface, or of the white substance of the hemispheres of the brain.

I need scarcely tell you that the condition of brain to which I allude is called white softening. This disease, when connected with a morbid state of blood-vessels, occurs most frequently at the more advanced periods of life, and is essentially an atrophic condition. After some time, the diseased brain-substance exhibits not only the diminished consistence which I have mentioned, but also the development of a peculiar series of large cells filled with fatty matter. These cells, as they occur

in the more chronic cases, are generally found in the immediate vicinity of capillary vessels, and sometimes are disposed, in a very striking manner, in rows parallel to them; they may perhaps be interpreted as abortive attempts at new fibres, or as products of some change in the secondary destructive or assimilative processes.

There is one other point which I must briefly notice in the history of this case, namely, that the fatal symptoms were ushered in by an attack distinctly epileptic in its character, from which the patient perfectly recovered, nor did a fresh attack come on until some hours after. I think it important that you should especially notice these cases of epileptic paroxysms coming on in states of brain in which the supply of blood is rather deficient than superabundant.* The fact has an interesting bearing on the pathology of epilepsy in general.

Let me now pass on to another case, which affords a good illustration of the *symptoms* which accompany this lesion during life, while the case just related illustrates the morbid anatomy.

Case CLXVIII.—H. B—, aged seventy-two (vol. xxxvii, p. 64). Our patient is a full, plethoric man, the beau ideal of the popular conception of an apopletic subject, with a short neck and a rubicund countenance. He has seen much better days, and, indeed, for many years represented an important constituency in Parliament. He used to live well in the common sense of the expression, and to enjoy the good things of this life while he could get them. But he acted too much on the principle, "let us eat and drink, for to-morrow we die." In short, he has been a jolly, hearty sort of fellow, who has lived a dissipated and a careless life, and who latterly has had to undergo much privation and distress, interlarded every now and then with a bout of intemperance to drive away care. On the 15th of June, while walking in the street, he suddenly became giddy, and fell backwards to the ground. He had experienced a transient feeling of giddiness once or twice within the same week, but did not fall on either of these occasions. He became lethargic and stupid, but retained consciousness when the fit

* *Vide* Case CLXVI, Lect. XXXVIII.

occurred, for he informed us he could distinctly recollect a medical man coming up to bleed him, and he also felt the prick of the lancet. When taken up, he was found to have lost all voluntary power over the left side, and he was unable to speak; but sensibility was not destroyed in the affected side. He remained under treatment in his lodgings until the 21st, when he was brought into the hospital, and the following note was made of his condition at that time:

There is no power of motion on the left side; but the sensibility of that side is unimpaired. The left cheek hangs, and the face is drawn to the right. The tongue, when protruded, moves to the left side. The muscles of the paralysed limbs are in a relaxed state, excepting the biceps muscle of the arm, which is slightly rigid, and becomes more manifestly so when you attempt to extend the forearm upon the arm. His articulation is imperfect, and he cannot swallow solid food, although fluids pass down readily.

The artery at the wrist is found to be in a thickened state from atheromatous patches. In all cases of apoplexy, especially in advanced age, you should try to ascertain the actual condition of the radial artery, as regards these deposits. If this vessel be freely studded with deposits, it is highly probable that other arteries are so too, and not the least likely those of the brain. The heart's sounds are feeble, as if the nutrition of the muscular substance of the organ had been impaired, and, upon listening carefully, a roughness accompanying the first sound is heard at the apex; the pulse is intermittent in character. Being very much depressed on his admission, the patient was ordered to take small and frequent doses of ammonia, and suitable nourishment.

There is a point connected with this case to which I may refer briefly. On examining the cornea of the eyes, we found a large and well-marked *arcus senilis* around each cornea. This condition has attracted some attention of late, in consequence of Mr. Canton having shown that it is a fatty degeneration of the cornea, commencing at the circumference. It occurs, as we had long known, generally after fifty; sometimes, though rarely, at an earlier period. Sometimes it encroaches very largely upon the cornea. I know a gentleman of seventy-eight,

in whom it forms nearly a third of that tunic. It is supposed to have a connexion with fatty degeneration of the arteries and with fatty heart; but it by no means follows that because the first exists, the others should also; although no doubt the three often coexist. And if you find a thickened condition of the radial arteries at the wrists, with indications of atheromatous deposit and a large arcus senilis too, it would be highly probable that the fatty degeneration of the small arteries of the brain had taken place, and this suspicion would be greatly increased if there have been an apoplectic attack.

Our patient, H. B—, has improved slightly since his admission. He is now (June 29th) much less lethargic, and has acquired some power in the paralysed limbs. Reflex action is more easily excited in the lower extremity on stimulating the sole of the foot.

What is the diagnosis in such a case as that of H. B—?

The case is not likely to be confounded with epilepsy, because there has been no absolute suspension of consciousness from the first moment of the attack. I think the phenomena of the case are best explained as follows:

There have been taking place, very gradually, and probably over a long period of time, deposits in the larger arteries of the brain, impairing more or less the nutrition of the parts which are supplied by these vessels, and generating a soft and friable condition of the nervous matter. Ere long a decided hæmorrhage takes place, which breaks down the soft cerebral matter, severs a large number of fibres, and creates shock by the suddenness of the laceration of the brain-substance, and by the compression of the healthy parts of the organ, consequent upon the escape of the blood from its normal channels. The result of the combined influence of shock and compression is the development of a more or less comatose state, which is, in the main, proportioned to the quantity of blood extravasated, although also to a degree dependent on the position of the clot. And the effect of the rupture of a large number of nerve-fibres is to disconnect the centre of volition from a greater or less portion of the body, according to the number of nerve-fibres lacerated.

It is important, however, to bear in mind that an equal

amount of paralysis might be induced without any laceration of brain or rupture of fibres. The compression of the surface of the brain would produce this effect, but for this purpose the clot must be a large one. There are two points that render it improbable that, in the case under discussion, the lesion is of this nature; first, that a clot of sufficient magnitude to cause by simple compression so much paralysis would probably give rise to a much greater amount of coma than existed; and secondly, that so much compression of the brain would be accompanied with much greater rigidity of the paralysed muscles.

Can we form any exact opinion as regards the precise position of the paralysing lesion in the brain? I have already stated why I do not believe it to be a clot on the surface. From the completeness and extent of the paralysis, it seems probable that a considerable portion of the centre of volition is in some way interfered with. Assuming the lesion to be white softening, and a clot, with ruptured and torn brain-fibres, it may be situated deep in the hemisphere, just outside the corpus striatum, and perhaps encroaching upon it, or in the corpus striatum, or the optic thalamus, or in both, or in the crus cerebri, or deep in the substance of one hemisphere of the cerebellum.

This patient remained in the hospital for nearly four months. The paralysed limbs had gained but a slight degree of power; but consciousness was completely restored; he never, however, gained strength; he became imbecile, and dirty in his habits, and extremely restless. His friends removed him into lodgings, where he soon died. An examination of the body was not allowed.

I must hasten to notice a third case, of which I have only time to take a brief survey. It affords a good example of what appears to me to be simple white softening, with solution of continuity of fibres, and without clot.

Case CLXIX.—Comfort Winning, aged sixty-one. Lonsdale ward, admitted June 16th (vol. xxxvi, p. 76). This woman had several confinements, and some severe ones; and she appears to have suffered from bronchitis in September, from

which, however, she recovered perfectly. In February last she lost the use of the *left* side of the body suddenly, and without affection of consciousness. The paralysis, according to her statement, was not complete, and she recovered perfectly within three months. Exactly twenty-four days before her admission she had a second attack. She was sitting at breakfast, and suddenly lost the use of the *right* arm and leg, with no other affection of consciousness than a sense of confusion and giddiness, and some pain in the head. This time the paralysis was very perfect; the arm and leg were completely paralysed as regards motion, sensation being slightly affected; the right side of the face was paralysed in the ordinary way. Her articulation also was much affected.

When admitted into the hospital, there was still palsy of the right arm; the leg had acquired a little power, she could move it slightly, and reflex actions were readily excited under the influence of stimulation of the sole of the foot. The palsy of the tongue and face had recovered, and the speech was less affected. Common sensation was but slightly impaired; but she suffered a great deal of pain in the paralysed limbs, especially in the arm, and there was a feeling as if the gums were swollen and the tongue too large for her mouth.

The paralysed limbs are completely relaxed. There is no rigidity of the muscles whatever, and the forearm may be flexed or extended on the arm without the least resistance. The fingers are slightly flexed towards the palm, and offer a little resistance to extension. There is a distinct arcus senilis in each cornea. The pulse is natural, but feeble, and there is no evidence of disease of the heart.

This woman has now (June 29th) been thirteen days in the hospital. During that time she has manifested little or no improvement. She has suffered chiefly from a very distressing symptom of not uncommon occurrence in similar cases, namely, severe pain in the paralysed limbs—partly referred to the joints, partly to the course of the principal nerves of the limbs. These pains are most severe in the arm, and are most troublesome at night, interfering much with rest. The only advance of power is seen in the leg.

This case is in all probability one of white softening from

obstructed circulation by diseased arteries. There is probably no clot, or at most a very small one. The grounds upon which I have formed this opinion are these:—First, the age of the patient, which is favorable to the morbid change of the vessels with which white softening is so often associated. Secondly, the fact that the present is a second attack, on the opposite side to the first. You know that, as was first made out by Bizot, the cerebral blood-vessels are apt to become diseased symmetrically. The former attack was probably due to a diseased state of blood-vessels; if there have been diseased vessels on the right side, it is probable such exist on the left, and this second attack might have been predicted with some degree of probability on the occasion of the first. Thirdly, the arcus senilis is well marked in each cornea.

The recovery from the first attack was in all probability due to a collateral circulation, which was sufficient to restore the nutrition of the softened fibres and to heal the breach. This attack, however, was comparatively slight, and it is not likely that much of the brain was involved. The second attack has evidently resulted from a much more extensive lesion.

I think you will admit that it is improbable there has been any extensive effusion of blood in this case, or, indeed, any effusion at all, if you consider that a clot of sufficient size to create all this paralysis would inevitably have caused more or less coma. The suddenness of the occurrence of the palsy appears to me to be sufficiently accounted for, on the supposition that a number of fibres connected with the centre of volition became softened up to a certain point, and giving way suddenly by a sort of deliquescence, severed the communication between the centre of volition and one half of the body.

The prognosis respecting this case is not favorable as regards the recovery of power in the limbs. A second attack, involving a large extent of brain, is not likely to be recovered from, because a considerable number of blood-vessels are doubtless diseased; and it is not probable that a sufficient collateral circulation can be obtained to bring the requisite supplies, through other channels, to the diseased portion of brain. But as to the duration of life, it is difficult to assign the period, as that will depend upon general care and nourishment, and the absence of

causes which may unduly excite either the cerebral or the general circulation.

The treatment adopted in this case was simply that of supporting by an appropriate and rather generous diet. Quinine was administered for a short time, and she took also iodide of potassium in five-grain doses every night with benefit to the pains in her limbs.

[This patient remained in the hospital till the end of July, and left improved in general health and as to the power of the leg, but without any increase of power in the arm.]

LECTURE XL.

ON RENAL COMA, AND ON DELIRIUM.

ON A CASE EXHIBITING RENAL SYMPTOMS CONNECTED WITH RENAL DISEASE, AND TERMINATING IN SANGUINEOUS APOPLEXY; AND ON A CASE OF DELIRIUM.

Gentlemen,—Two cases have within the last day or two terminated fatally in the wards of this hospital; these I mean to make the subject of my observations to you to-day; they are both of a very interesting nature, highly illustrative of important points in pathology, and such as will well repay careful study by those who have narrowly watched them.

Case CLXX.—The first is that of Moses Jeffrey (vol. xxvi, p. 56), a heavy, lethargic patient, whom you must all remember as having been long an inmate of the Sutherland ward, where he was admitted on March 7th, 1849. His disease belongs to an interesting class of cases, which, while they exhibit a remarkable similarity of symptoms, may be referred to very different causes. Our knowledge of the real nature of these cases is deduced from the union of the study of clinical history with post-mortem examination. Indeed, without the opportu-

nity of examining the state of the principal organs of the body after death, we should never have arrived at any accurate knowledge of the real nature of these cases; for, did we look alone to the symptoms, we should be led to refer them to only one and the same cause—as, indeed, had always been done, until modern researches, by means of that union to which I have already referred, of diligent clinical study with post-mortem investigation, taught us to interpret the symptoms, and to appreciate their real value and indications.

Our patient was one of a class of men addicted to intemperate habits; he was a compositor by trade; he had formerly drunk a good deal, but for eight years he had been a teetotaller, and had strictly adhered to the pledge. His recent temperance, however, notwithstanding that it extended over so many years, was not enough to save him from the consequences of his former habits.

Five years before his admission he began to have œdematous swelling of his legs, but this did not last long; it was probably owing to some temporary defective action of the skin. His next symptom, which commenced six or eight months before he was admitted, was frequent micturition at night; he was compelled to get out of bed six or seven times of a night. This was probably due partly to an irritable bladder, and partly to the quantity of urine secreted. He next became affected with head symptoms; he suffered from violent pain in the vertex, which continued ever after unabated. Soon after this his sight began to get dim, the little and ring fingers of his left hand became benumbed, and he felt very drowsy and unable to work.

The report goes on to describe him as "a heavy-looking man, thin and pallid; the complexion peculiarly sallow, as if not only the red particles of the blood were deficient, but also their hæmatine altered; frequently staring with a vacant gaze, answering questions very slowly, and appearing as if he felt it irksome to be made to reply otherwise than in monosyllables; moving his limbs about very slowly, and apparently with effort; pupils equal, contracting sluggishly."

Now, a patient coming to you with such a catalogue of symptoms as this, you would be tempted to set the case down

as one of disease of the brain, in which the primary mischief is seated in that organ; and, if you made no further inquiry, you would immediately direct the whole force of your treatment to the head. Indeed, some years ago, this is what all practitioners would probably have done; for, in the state of knowledge then, it could not have occurred to them what else they should do; but we now know that such symptoms as I have already detailed to you, referable as they certainly are to the organs within the head, ought, nevertheless, to direct our attention to other organs likewise—namely, to the kidneys, and more especially if, in addition, the patient has a pale, sallow complexion, such as was very well marked in Jeffreys. Then, turning our attention in this direction, what information did we get in the present case? Why, we found that the quantity of urine was considerable, and that its specific gravity was very low; that it was acid, contained albumen, and that, on being allowed to stand, it threw down a scanty precipitate, which was found to consist of epithelium, of transparent casts of uriniferous tubes, and of a few cells containing a considerable quantity of fat.

Here, then, we get a satisfactory explanation of the head symptoms—a true and sufficient cause for them; we find them to be *secondary*, resulting from previously existing and primary disease of the kidney. It was evident that the general nutrition of our patient was considerably impaired; and, as the result of this impairment of nutrition in general, there was of course an impairment of the nutrition of the brain in particular; and moreover, although there was a considerable quantity of urine passed—for some days, indeed, the quantity of urine was so large (as much as nine or even ten pints) as to have led us to test for the presence of sugar—still it is probable, from its low specific gravity, that the due quantity of urea and other solids of the urine was not eliminated; and thus the blood, and the various organs, were poisoned at the same time that they were imperfectly nourished. And this twofold aberration from the healthy standard — this actual poisoning, as well as imperfect nutrition of organs — this nourishment, by not only a poor, but by a poisoned blood, which inevitably results from chronic renal disease—explains how a train of symptoms similar to those above detailed may

arise from a particular state of the kidneys. I do not say that these symptoms may not recur in cases where there is no kidney disease, but I do say that, in a considerable number of cases in which they are present kidney disease will also be found.

The circulation was feeble, the pulse small and weak; but the heart's impulse was strong, and was felt over a greater surface than was natural. There was, however, no correspondence between the heart's force and that of the pulse, nor was there evidence of any valvular disease. At no time was there much dropsy. It never exceeded a moderate anasarca of the legs and a slight puffiness of the face.

This man continued in the hospital for four months, with scarcely any variation in his symptoms. He always attracted attention by his pale, sallow, and heavy look. He complained constantly of more or less headache, referred for the most part to the vertex, was always more or less lethargic, slept a great deal during the day, always heavily at night, but was frequently awakened up by frightful dreams. He was often sick, and vomited his meals. He had no paralytic symptom, except a numbness of the left side of his face, and a similar numbness of the little and ring fingers of the left hand; which, however, sufficiently indicated that the impaired state of blood and of general nutrition was doing its work in the brain. Up to the day of his death he continued to pass pale urine, in quantity varying from five to ten or eleven pints, never exceeding 1012 in specific gravity, and always containing a notable quantity of albumen, although not a very abundant precipitate in each portion examined.

For some days we compared the quantity of urine which he passed with the quantity of his drink, and found that they maintained a pretty close relation—sometimes the latter exceeding the former, at other times the reverse; but in neither case was the difference more than from half a pint to a pint.

You will remember—those of you, at least, who are accustomed to accompany me through the wards of the hospital—that I had no difficulty in saying that the disease was in the kidney, and in indicating of what sort it was. I told you that the kidney was contracted, shrivelled up, from the atrophy and

obliteration of numbers of the uriniferous tubes, altering the relations of the tubular and intertubular structures, creating congestion in patches and obliteration of some vessels, and interfering with the healthy formation of epithelium. This diagnosis rested upon the absence of more decided symptoms of brain disease than the pain, the lethargy, and the numbness of one or two parts, and upon the known sufficiency of renal disease to disturb the nervous system in the manner and to the extent to which that disturbance existed in our patient. It was likewise indicated by the peculiar complexion of the patient (no hæmorrhage having taken place from any other organ than, perhaps, the kidney), by the presence of albumen in the urine, by the dropsy, and by the large quantity of urine passed.

This last symptom, with the low specific gravity of the urine, the comparatively small amount of albuminous precipitate, and the moderate extent of the dropsy, led me to infer that the disease was *contraction* of the kidney, due to a chronic degeneration of that organ. In cases of the enlarged kidney, which owes its increased size to the deposition of fat in the epithelium-cells, or in that caused by the undue formation of epithelium distending the tubes, the urine is deficient in quantity; it has a smoky hue, often contains blood, the albuminous precipitate is very abundant, sometimes as abundant as in the serum of the blood, and the specific gravity is higher.

The diuresis which is often present in these cases, and which was so marked a feature of that which we are now considering, is explained by the condition to which the renal structures are reduced, as the result of chronic disease. The tubes being stripped of epithelium, and consisting, in great part, solely of basement-membrane, allow that to take place throughout their whole length, which in health would occur only at the Malpighian bodies, namely, the percolation of the aqueous part of the secretion. This great discharge of water was, however, a fortunate thing for our patient, for it enabled him to get rid of a much larger amount of solid matter than he would otherwise have been able to do. Had this free excretion of water been checked at any time, serious symptoms would no doubt have instantly shown themselves in epilepsy or profound coma. By Dr. Beale's analysis, it appeared that, shortly after his admis-

sion, the urine, of which he then passed about five pints daily, contained twenty-two parts of solid matters in a thousand, and these consisted chiefly of *albumen* and *extractive matters*, whereas twelve or fourteen parts at least ought to have been urea. Even the increased flow of such urine as he was passing would not have carried off enough of the organic and saline constituents to purify the blood. On the other hand, the constant drain of so much albumen, about 300 or 400 grains per diem, must have greatly injured the blood, as the nutrient fluid of the nervous system, by depriving it of so large a quantity of its chief staminal principle. And you can readily understand how even a slight check to the renal secretion would prove mischievous, by reducing still further the small amount of excretion of the proper organic matters of the urine.

The prognosis of a case like this is always unfavorable. If the secretion be copious, the patient may linger on for a considerable time, as happened in the present instance; but if the secretion be materially checked, he will presently die. And although in our patient the flow experienced no diminution, he died suddenly, yet not unexpectedly; the manner in which he died differing somewhat from that of many other cases of the same disease. For some time his symptoms had been becoming aggravated; he had been more drowsy and lethargic, more doughy-looking and blanched, and the dropsy had gained on him, although not to any great extent. One night, without any special premonitory symptoms, he suddenly became comatose, breathed with stertor and puffing, his pulse became slow and languid, his respiration interrupted with increasing intervals, till it gradually and finally ceased. There were no convulsions. The time from the first access of the coma to his death was not more than a quarter of an hour.

The manner of death showed that it was owing to some change in the brain; and the short duration of the coma, and the speedy supervention of death, would go far to localize the lesion. Of this supposition the post-mortem examination was quite confirmatory; the following were the conditions that it revealed:—On opening the cranium the membranes were found quite healthy; on removing the brain a clot was found in the fourth ventricle, which proceeded from the left lobe of the cere-

bellum, having broken down the substance of that centre to enter the ventricle. The blood had passed through the iter into the third ventricle; having filled which, it passed, on either side, through the foramen of Monro, into the lateral ventricles, which it traversed, following the course of the choroid plexuses, and finally descending into the inferior cornua. The blood had coagulated into a firm clot, so that the whole could be taken out as a mould of the cavities and connecting passages. The choroid plexuses were lying on the inside of the clot, apparently healthy. The anterior cornua were filled with serum. The corpora striata and optic thalami, and the hemispheres of the brain, were healthy.

Now, in all cases where death supervenes very speedily on apoplexy, the clot is very likely to be found in a similar situation to this, involving, if not confined to, the mesocephale. Death is the more speedy the nearer it is to that important and central portion—that nucleus of the brain. In such cases death occurs much as it would do when an animal is pithed.

I may also refer you to a symptom which occurred in this case, and which is, I think, a very fatal one in apoplectic cases —namely, the flapping of the cheeks in respiration. It is due to paralysis of both the buccinator muscles, and is the result of great compression of the brain, or of compression of it near the centre of respiration, which is also the seat of implantation of the facial and fifth nerves, upon which the action of these muscles depends.

The kidneys which I show you here were found in exactly the state which I described to you before the man died. Observe the surface of this kidney, how shrunk, granulated, and fissured it is, and on cutting it, how the organ is wasted, at the expense chiefly of the cortical substance. On a microscopical examination it was found that the tubes were stripped of their epithelium; in some parts the cells were filled with fat, but the chief feature as regards the epithelium was its scantiness and its imperfect organization. The thinness of the walls of the uriniferous tubes, as I have already explained, was favorable to the excretion of water, but not of organic matter, which is separated solely by the agency of the secreting epithelium; the former, holding albumen in solution, was, there-

fore, carried on with an energy proportional to its abundance, but the organic constituents of the urine would be secreted only in proportion to the quantity of epithelium existing in the kidney.

Coupling together the clinical history and the morbid anatomy of the case, there can be no doubt that the state of brain was dependent on that of the kidney; and had it not been for what we may call the *accident* of the apoplexy, the patient might have lived on for some time longer, might have had several epileptic fits, and would probably have died in one of these, or from the exhaustion which follows such attacks.

Taking together the state of the kidney and that of the cerebral functions, nothing can be clearer than this class of cases; but putting the state of the kidney out of the question, nothing can be more obscure. There is no doubt that the kidney is the *primum movens;* the blood becomes poisoned; this poison interferes with perfect sanguification, a poor nutrient fluid is carried to the brain, which becomes, as a consequence, ill-nourished; add to this the probable actual presence of urea, and other solids of the urine, in the brain, and we have quite sufficient to account for all we have seen.

In the treatment of this case there was really little to be done. It is not improbable that by the frequent blistering of the neck and scalp, and other means of counter-irritation employed, such as the long issue in the scalp, we may have retarded the mischief within the cranium, and prolonged life somewhat; beyond this we cannot claim much credit for any benefit the patient may have derived. This is one of those maladies in which, the more we know as to its intrinsic nature, the more we feel the impotence of our art to effect a cure. And so far, indeed, ignorance would have been bliss; in a certain sense, if it be not folly to be wise, our increased wisdom is attended, at least, with results less flattering and agreeable to our vanity. Still the knowledge of the disease, and a correct diagnosis, no doubt saved the patient from many blind and haphazard trials of remedies, which might have diminished his strength without any promise of good.

Another case that has recently come under our notice is, in one point of view, illustrative of what I have already said

as regards the occurrence of cerebral symptoms without cerebral lesion, and it is also interesting on its own account.

Case CLXXI. — "Maryanne Beasely, aged thirty-seven (vol. xxvi, p. 209), native of London, where she has always lived; married, and has five children; of very intemperate habits, being much addicted to gin-drinking; in appearance thin and haggard, looking much older than her stated age; very weak and restless; her manner nervous and agitated; she has not slept for a considerable period; tongue somewhat furred, tremulous; pulse large, soft, not frequent." She had psoriasis on the back of both hands; for which malady, and for her general cachectic condition, she was sent to the hospital. She was admitted on the 1st of June, "and in the course of that night," the report of my clinical clerk, Mr. (now Dr.) Hyde Salter, goes on to state, "in no part of which had she slept, she was seized with furious delirium, and talked loudly and coarsely. By the negligence of the night-nurse, the house-physician was not called up; but on visiting her the next morning, she was found in a state quite resembling delirium tremens, suffering from various hallucinations of black-beetles, black men, and especially policemen, whom she appeared to hold in great horror. Stimulants and opium were prescribed, but without any immediate effect, and she became so violent that it was found necessary to remove her to a separate ward, and employ a strait-jacket."

Let me here remark, in passing, that in your practice the use of the strait-jacket should always be a last resource. If you cannot get a sufficient number of attendants, and there is danger of the patient doing himself injury, then it is absolutely necessary, and you have no choice; but it should be avoided if possible; it creates opposition, excites the patient, tends to prolong his delirium, and exhausts him. Remember, I do not say *never* use a strait-jacket, but make its *necessity* the rule for its use; for in many cases it would be highly dangerous, and even fatal to the patient's life, to dispense with it.

But to return to the history of the case. The delirium, notwithstanding the opium and stimulants, lasted till the night of the 4th, when she got some sleep, and from this time to the 8th

she rapidly improved, but on that day she began to squint. The urine was carefully examined, and found not to contain albumen. The report of the 8th says:—"The patient appears the same in every respect as when last reported, except that she has a slight but distinct internal strabismus, affecting principally the left eye; pupils rather dilated, but both exactly alike. She is much agitated at this uncomfortable symptom, but her agitation has not at all the appearance of delirium tremens. She states that when she looks at an object with both eyes it looks indistinct and double, and she is totally unable to distinguish the distances and relations of surrounding objects; when, however, she covers up one eye, and it does not matter which, the vision is perfect."

She continued to improve, with the exception of the persistence of the strabismus, till the 12th, when she was taken back to the Augusta ward. Here she became delirious again, but the delirium was not violent; it was low and muttering; the squinting increased. The report of the 14th is as follows:—"The patient is a good deal weaker to-day; the squinting remains the same as when last reported. She is agitated, and rather more delirious, and the delirium is of a low character, but not resembling delirium tremens in the least; pulse 110, weak; passes her motions under her. She has a good deal of bronchitis, diffused pretty generally over the chest; she coughs a good deal, but does not spit up the mucus; and she passes her motions under her, apparently from listlessness and inability of exertion;" in fact, she passed quickly into a typhoid state.

In the evening of the 14th she seemed to rally a little, but about one o'clock on the morning of the 15th a change for the worse took place; she became comatose, which state gradually increased, and she died about half-past seven in the morning.

My chief object in calling your attention to this case is to point out to you the great apparent want of correspondence between the state of the brain and the serious nervous symptoms under which the patient laboured. Nor, indeed, did the post-mortem examination bring to light such a diseased state of any other organ as sufficed to explain the fatal result.

There was considerable congestion of the lungs, and an abundant secretion of mucus in the bronchial tubes. The

kidneys exhibited the appearance of passive congestion, but were otherwise healthy.

From the suddenness of the accession of the typhoid symptoms which came on at last, I was suspicious that our patient was suffering from purulent infection, that pus had formed somewhere, and had passed into the circulation. And this impression was the stronger from the resemblance of her symptoms to those of a woman who had been in the hospital some time ago. She was under treatment for chronic bronchitis, and was going on tolerably well, when one day she became rapidly typhoid, with low delirium, her tongue furred and brown, and full, quick pulse. This state continued for two days; then sudden coma came on, and she died. We found, on examining the body, an abscess in the septum of the heart; this had burst, and discharged its contents into the circulation. In many of these cases, if the patient does not die immediately, you get deposits in different parts of the body, particularly in the joints; but in such a case as I have related there was no time for such secondary abscesses. Among puerperal woman we may often meet a case of this kind; a woman has been safely delivered, and everything seems to be going on well; you visit her in the evening, and find a little uneasiness; you order her bowels to be cleared out; the next day you find her sinking, it may be, dying, and she may die within four and twenty hours of the first accession of the unfavorable symptoms. If you examine the state of the veins of the uterus, you will find the explanation —the woman had uterine phlebitis, and pus in her blood; these are cases that occur in the practice of every man.

But in this case we were not able to discover any source of purulent infection. Here is the brain; you perceive that it is decidedly paler than natural, and that those red points, which are produced by cutting across vessels, on slicing the white substance of the hemispheres, are perhaps too numerous and too large. With these slight exceptions, the brain and its membranes seem perfectly healthy in their structure; there are no unnatural appearances; there is no evidence of any distinct lesion. And this is the point that I am most anxious to impress upon you—that the delirium does not necessarily connect itself with any actual disorganization of the brain or its membranes,

nor does coma either; but that both these formidable states may take place in a brain that shall reveal, on the minutest scrutiny, no appreciable aberration from the natural standard.

Now let us ask, of what did this woman die? I have already told you of my suspicion regarding purulent infection; but this must be abandoned, from our having failed to discover any purulent formation. I incline very much to think that the patient was not benefited by having been moved from one ward to another soon after she had been brought into the hospital.

She had been in a very exhausted state during her delirium; from this she had recovered to a great extent, and then she was moved up several flights of stairs to another ward. The fatigue consequent upon this, added to the attack of bronchitis, must have tended greatly to produce the typhoid state in which she died.

I have met with more than one instance of bad consequences following upon the removal of a patient in delirium, or just recovered from it prematurely. About two years ago a man was admitted here for epileptic delirium. Finding that his delirium was very noisy, and disturbed the other patients, I had him placed in a separate ward, where he recovered from his delirium. It was found necessary to move him upstairs, and shortly afterwards he became delirious again, and died comatose.

I am satisfied, from these and other cases, that there is nothing respecting which we ought to be more cautious than as to moving patients either in or just recovered from delirium; even to move them from one room to another on the same floor is dangerous, still more moving to any distance, or to another floor. Let us take this case as a warning of the necessity of great caution and circumspection before we sanction the removal of a patient under such circumstances.

LECTURE XLI.

ON PARALYSIS.

ON HEMIPLEGIA.

I PROPOSE, in the present and two or three subsequent lectures, to make some remarks on the clinical history, pathology, and treatment of that form of paralysis which, as affecting one half of the body, is usually called Hemiplegia. The affection is a very common one—we are rarely without two or three examples of it in the hospital; and you may often see persons labouring under it, walking in the streets with characteristic gait. Persons attacked with what is commonly called a paralytic stroke, are generally seized with this form of palsy.

Now, let me first describe to you the precise features of hemiplegia, and the manner in which it differs from other forms of palsy, so that you may easily recognise it when you see it. You have an excellent example of it in the case of John Scott, in Fisk ward, and you may compare my description with the actual condition of that patient.

The term *hemiplegia* denotes a palsy stroke, affecting either half of the body; the parts actually involved are the upper and lower extremity, the muscles of mastication, including the buccinator, and also of the tongue on one side. This must be distinguished from *paraplegia*, which means paralysis of the lower half of the body, in which both legs, and perhaps some of the muscles of the bladder and rectum, are paralysed.

You may have hemiplegia either *complete* or *incomplete*, as regards motor power, there being also great variety as to the affection of sentient power. In the complete paralysis, the upper and lower extremity on one side exhibit a complete loss of the power of motion, the face is very much affected, and the tongue also.

When a patient is seized with this palsy, if he be standing or sitting, he falls, even though he retain his consciousness perfectly, because the power of maintaining his equilibrium is destroyed by the failure of the antagonising muscles of one half of the body. On coming to such a case, you find the patient lying on his back, with total inability to move either the arm or leg; both of which lie, as if lifeless, by his side. Sometimes all power of motion is destroyed, and no movement can be excited either by any effort of the will or by any degree of external stimulation. In other cases, a very slight stimulus, such as tickling with the finger, or with the point or feather of a pen, or the application of a hot spoon to the sole of the foot, will, to the great surprise of the patient, produce active movements.

It is a point deserving of your notice, that very often either the movement thus excited, or the stimulation applied, although in itself not severe, will cause the patient a good deal of pain. It has often struck me that there is in these cases an irritable state of the sentient nerves and of the centre of sensation, and that both the stimulus applied and the jerking action of the muscles create a good deal of pain. The patients frequently call out lustily from the pain thus produced, and express a great dread of, and dislike to, the repetition of the experiment.

You will also observe, that these excited motions, now so well known under the name of *reflex actions*, occur almost exclusively in the lower extremity. If you stimulate the palm of the hand or pinch the skin of the forearm, you cannot produce movements such as occur in the lower extremity, nor, indeed, as a general rule, any movements at all, unless it be that, when a patient has acquired some grasping power in the hand, tickling its palm while he is attempting to grasp, will sometimes increase the power with which he performs that action.

There is, however, a curious and very interesting involuntary movement, which you will sometimes witness in hemiplegic cases. It occurs simultaneously with yawning, and less frequently with the actions consequent on emotion, surprise, joy or pleasure, or grief, as in laughter or crying. I may here mention that yawning is a very frequent, sometimes a troublesome, and not always a favorable symptom after an attack of

hemiplegia. It is more frequent in proportion as the shock is severe, but it seems to come on as the first effects of the shock are declining.

You will now proceed to examine the face. In slight cases this will not be at all affected, and the palsy will be confined to the limbs; sometimes, on the contrary, it will be the part first and alone paralysed. It is very necessary that you should be well impressed with the characters of the facial palsy which accompanies hemiplegia, and that you should be able readily to distinguish it from that which arises from affection of the portio dura. Place the patient well opposite you, and observe the conditions of the face, as it is perfectly quiescent. You will perceive that the paralysed cheek hangs, and that the angle of the mouth on that side is lower than its fellow; the cheek is more or less loose and flaccid, in proportion as the paralysis is more or less perfect. If now you ask him to smile or speak, the want of equilibrium of the face becomes very apparent. The healthy muscles being relieved of the antagonism of the paralysed ones, contract to a much greater extent than is natural, while the palsied cheek remains quiescent, or allows itself to be drawn slightly towards the mesial line. This exaggerated action of one cheek attracts very much the attention of the patient's friends, or himself when he looks in the glass; and they are apt to refer all the mischief to the really sound cheek. "His face," the patient's friends will tell you, "is all drawn on one side;" and, they will hardly believe, when you assure them that the drawn side is all sound, and that its being drawn is merely the result of the want of a resisting power on the opposite side. But, with all this, the patient can shut both eyes well, and open them; and he can move the cheek, so far as can be effected by the zygomatic muscles, and he makes a very fair attempt at pursing up the mouth for whistling, this last act, however, being rather impaired by the intimate connexion of the buccinator with the orbicular muscle of the mouth. The muscles of mastication on the paralysed side act with less power, although it seldom happens in hemiplegia that their power is completely destroyed. Doubtless, much of their action is reflex, and this explains the fact, that while the buccinator muscle is very much paralysed, and even wasted, the

masseter and other masticatory muscles retain a considerable amount of power.

Your anatomical knowledge will explain all this to you; the fifth nerve is more or less involved in or influenced by the paralysing lesion. In proportion to the extent to which it shares the depressing influence will be the number of muscles engaged, and the degree of their loss of power. If the nerve be immediately involved in the lesion, then the face-palsy is at its highest point. Some of you may remember a striking instance of this in the case of a man named Coulson, subject to epileptic fits, who was a frequent visitor to the hospital, and was for some time a standing example of very complete palsy of the fifth nerve. In this man the face wore the same aspect as in hemiplegia, but, in addition, there was remarkable wasting of the temporal and masseter muscles, and a complete hollow in the regions which they occupied.

The facial nerve, or portio dura, is not generally touched by the paralysing lesion in hemiplegia. Hence you find the orbicularis palpebrarum and the superficial face-muscles unscathed. In a few cases, indeed, it has appeared to me that these muscles were weakened, as if the nerve participated slightly in the shock; but complete palsy of the nerve rarely takes place, I would almost say never, unless the paralysing lesion is situated near its implantation (Lect. XXXVI, p. 644).

In some cases the third nerve is paralysed, and this is shown by dropping of the upper eyelid and an inability to elevate it, and by squinting of the eyeball outwards, and more or less dilatation of the pupil. Not uncommonly palsy of the third nerve is a precursor of the hemiplegic attack, and its occurrence should always excite your fears of approaching more extensive mischief. Still, it is remarkable that this nerve so often escapes in hemiplegia; how many hemiplegics do we not see who have no trace of affection of the third nerve! Indeed, sometimes this will occur: a person will be seized with palsy of the third nerve, and of no other; he will get well of this, and remain well some time; he will then be seized with hemiplegia, the third nerve escaping any fresh attack.

You now ask the patient to put out his tongue, and you observe a very characteristic phenomenon. The tongue is pro-

truded with a more or less distinct deviation to the paralysed side. This is clearly owing to the impairment of the equilibrium of the protruding forces. Those on the right being paralysed or weakened, those on the left prevail, and push the tongue to the opposite side, that is, to the right. Very often the tongue-muscles are only weakened by the paralysing lesion, and the patient, on directing his attention specially to it, can protrude the tongue quite straight. I need scarcely tell you that this tongue-palsy is due to the ninth nerve being influenced by the paralysing lesion; the very same phenomenon may be caused by a local lesion affecting the nerve only.

In a physiological point of view, it is a very interesting inquiry how it happens that the fifth and the ninth nerves are so frequently—nay, almost universally—paralysed in hemiplegia, while the portio dura escapes. I cannot enter upon this point now, and must be content with remarking, that the discussion involves some highly interesting questions connected with the mechanism of cerebro-spinal actions.

The palsy of the face, and that of the tongue, conjointly, give rise to the imperfection of articulation often present in hemiplegia. The patient speaks thick, and is especially indistinct in the pronunciation of labials and dentals, giving a guttural character to all his words. But sometimes the power of speech is wholly destroyed, even in cases where these nerves have suffered but little or not at all, or the power of utterance is limited to "yes" and "no," or either of these monosyllables; and this is a sign of very unfavorable portent, as denoting, with the other symptoms, extensive lesion of brain, superficial as well as deep.

It is curious how rarely it happens that the muscles of the trunk, as the intercostals, or the abdominal muscles, are involved in the hemiplegic paralysis. It must be an extensive cerebral lesion which will paralyse these muscles. There is, however, a spinal hemiplegia of which this palsy is a prominent feature.

Deglutition is also sometimes impaired, the vagus or glosso-pharyngeal being probably affected; but this is not a very frequent symptom, and, when present to any great extent, it denotes a serious and extensive lesion of the brain.

In grave lesions, especially when of the inflammatory kind,

the sphincter ani muscle is paralysed. This symptom, which augurs most unfavorably, is, happily, of only rare occurrence.

Such being the phenomena in hemiplegic palsy, let me say a word or two touching the way in which such an affection may be produced. You know that paralysis may be caused by any lesion which interrupts the continuity of a nerve or set of nerves, and which interferes with the due connexion between these nerves and the centre of volition; or by lesion of the centre of volition itself. Thus, then, you may have hemiplegia dependent on peripheral affection of the nerves, the morbid process spreading from periphery to centre—this is a rare and an incomplete form of hemiplegia—or you may have it caused by a lesion in some part of the brain or spinal cord. If the lesion be situated within the cranium, above the point of decussation of the pyramidal columns of the medulla oblongata, the palsy will be on the side of the body opposite to the lesion; this is the most common form of hemiplegia. If it be seated in the spinal cord, below the decussation, the palsy will be on the same side of the body as the lesion; but in such a case, which is very rare, the phenomena present certain very essential points of difference from cerebral hemiplegia.

Looking at the matter, now, in a clinical point of view, I may state to you that the following forms or varieties of hemiplegia will occur to you in practice, of most of which I hope to bring before you cases in illustration. First, and most commonly, you have the typical hemiplegia of diseased brain, that is, a brain affected with some distinct and special lesion, such as an apoplectic clot, a softening involving a considerable portion of the centre of volition, or a tumour in this centre, or compressing it. With this you may contrast the rare but not less certain spinal hemiplegia caused by a lesion involving one half of the spinal cord, just below the decussation of the pyramids. Thirdly, you may have hemiplegia consequent upon an epileptic attack, in which the paralysing lesion is generally transient, and the palsy remains only a few hours, or at most a few days, after the epileptic seizure. From this close connexion between the paralysis and epileptic fit, I prefer to mark this form of hemiplegia (although it may strictly be classed with the cerebral hemiplegia) as *epileptic hemiplegia*. Fourthly, you may have hemiplegia

following, and sometimes, although rarely, preceding chorea—*choreic hemiplegia*. Fifthly, you meet with a peculiar and less perfect form of hemiplegia in hysterical women—the *hysterical hemiplegia;* and in nervous, hypochondriacal men I have seen an analogous form brought on under the influence of strong emotion. Lastly, you have a form which, from its mode of access, creeping as it were from periphery to centre, you may call *peripheral hemiplegia*.

Now, in all these forms of hemiplegia the paralysis is prominently a paralysis of motion. This will occur in various degrees, from a slight awkwardness in the movements to a completely paralysed condition, in which the patient does not possess the slightest power over the muscles of the limb. The extent to which sensation is impaired bears no constant relation to the degree of motor paralysis. You may have complete paralysis of motion with a sound or even an exalted state of sensation. In general, however, sensation is more or less impaired. In estimating this point, you must not be content with simply pinching up the skin of the patient, for there are many instances in which the sensibility is deadened, but in which it might escape our notice, unless we employed a more accurate method of investigation. The method I adopt is similar to that employed many years ago by Weber in comparing the sensibility of the surface in different parts of the body. It consists in ascertaining how near two sharp points of a pair of compasses may be approximated, and yet be distinctly felt as two points by the patient. Upon comparing the impression thus produced upon the sound limb with that on the paralysed limb, an idea and a definite expression of the extent to which sensation is involved may be arrived at. On the paralysed side the two points will be considered by the patient as but one, while on the opposite limb he will be able to distinguish clearly that there are two.

I propose in this and the subsequent lectures to illustrate these forms of hemiplegia by cases that have fallen under my notice; and I shall first speak of those cases which are distinctly caused by some special lesion of the brain.

I have many times had occasion to direct your attention to the condition of the muscles of the paralysed limbs in cases of

cerebral hemiplegia. This point has not hitherto received the attention it deserves, and many otherwise interesting narratives of cases are comparatively of little value, from the omission of all mention of this symptom. Now I hope, as we go on, to show you that from the state of the muscles of the palsied limbs, especially of the upper extremity, as being nearest to the seat of the lesion, you may draw inferences as to the nature of the lesion which will afford important aid for diagnosis and prognosis; and I propose to make this the basis for an arrangement of cases of hemiplegia, which I trust you may find useful in practice.

Looking, then, to the state of the muscles of the palsied limbs, I arrange cases of cerebral hemiplegia in three classes:

The first class consists of those cases in which the muscles of the paralytic limbs are completely relaxed. The limbs are loose and flaccid, and if you flex the forearm upon the arm, or the leg upon the thigh, you find no resistance or opposition to that movement. When you feel the muscles you find them lax and flabby, contrasting more or less with the firmness and plumpness of those of the sound limbs, and they are more or less wasted according to the period of time which has elapsed since the paralytic seizure.

In the second class I place those cases in which the paralysed muscles exhibit a certain amount of rigidity, *which rigidity has existed from the moment of or soon after the attack.* This rigidity varies in degree from an increased plumpness of the biceps of the arm and the hamstring muscles in the thigh, and a resistance on the part of these muscles to the extension of the forearm or leg, up to a contraction almost tetanic. The nutrition of the muscles in cases of this class is not materially weakened at first, and the wasting is consequently either *nil,* or is very trifling. If, however, the palsy persist, the muscles waste, although not so fast as in the first class of cases.

In the third class, we find cases with rigid muscles likewise. In these cases the rigidity is a late phenomenon. It does not occur for some time after the paralytic seizure. The cases of the first class often pass into this. The wasted and relaxed muscles after some time gradually acquire more or less of ten-

sion; they become shortened, and appear like tight cords stretched between their origin and insertion. The tension is most manifest in the flexor muscles, and the limbs assume the state of more or less flexion, especially the upper extremity. The forearm becomes strongly contracted on the arm, and the fingers flexed into the palm of the hand, which is liable to be irritated by the growth of the nails.

In all cases of cerebral hemiplegia, I advise you to pay minute attention to the investigation of the arterial system, and also of the heart. In old persons, or those somewhat advanced in life, we often find in the state of the radial or temporal artery a clue to the condition of the arterial system in general, and of the arteries of the brain in particular. In feeling the pulse you should roll the artery beneath your finger, and examine in this way as long a portion of it as you can get at. If the artery be diseased you will find a thickened state of its wall, and sometimes you will be able to detect distinct deposits in it, which now and then will be hard and resisting, owing to their admixture with earthy matter. You must be careful likewise to examine and compare the arteries of both sides, when you will often find corresponding states, and you will notice that the deposits exist more or less symmetrically. And this should confirm your suspicions, that the diseased state is not limited to the radial or temporal arteries, but exists pretty extensively throughout the arterial tree.

Your conclusion respecting the morbid state of the arteries will receive further confirmation if, on examining the heart, you obtain evidence of its being in a state of hypertrophy, for a morbid state of the arteries is a fruitful source of hypertrophy of the heart. But, indeed, a disease of the heart of any kind, in advanced life, is very liable to be accompanied with a more or less diseased state of arteries.

Recently the observations made by Dr. Kirkes in this country, and by Virchow in Germany, have rendered it probable that disease of the brain, capable of producing hemiplegia, may sometimes be caused by the obstruction of a principal cerebral artery by a plug of fibrine detached from an excrescence on one of the aortic or other valves of the heart, the result of former endocarditis. You should, therefore, in examining the heart

in young persons, look out for evidence of valvular disease caused by endocardial deposits.

Nevertheless, I must confess that I am not convinced that in cases such as Virchow and Kirkes refer to, the stoppage of the arterial circulation is always caused by a plug accidentally brought from a distant part of the circulation. I should be more disposed to refer it to a coagulum formed in the artery, promoted by an altered nutrition of its wall—*arteritis*, if you choose so to call it—and connected with a rheumatic or other morbid state of blood.*

That a softened state of brain—the state called white softening—follows the retardation and diminution of the cerebral circulation by diseased arteries, or its complete stoppage by a plugged artery, is now as well proved as any fact in pathology. To what extent the softening may go without producing paralysis, and whether, indeed, sometimes the mere shock of the sudden cutting off of a certain quantity of blood from a portion of the brain may not act of itself, without any softening of brain-tissue at all, these questions have yet to be decided. Neither has it been made plain, how the paralysis comes on so suddenly as it almost always does. Some would say it is because the stoppage of the blood-supply is sudden. But in many instances of softening from ill-nourished brain, the failure of the blood-supply is a very gradual process, and in some, in which the stream of blood is suddenly checked, paralysis does not come on for some time afterwards. Take, for example, some of the cases in which a ligature has been passed round the carotid artery. In Sir A. Cooper's well-known case, the palsy did not appear for seven days after the operation, and in one of Mr. Vincent's cases paralysis of sensation came on twenty-four hours after the operation, and four days afterwards paralysis of motion; and in my case of dissecting aneurism the paralysis did not ensue upon the plugging of the artery for three days.

I have been myself in the habit of attributing the sudden occurrence of the palsy to the rupture or rapid deliquescence of fibres which had been already softened, but not sufficiently so

* See an interesting case recorded by Dr. Gibbon in the 'Transactions of the Pathological Society,' vol. v, p. 11.

as to interrupt their powers as conductors of the nervous force. When hæmorrhage has taken place into the substance of the brain, the effusion of blood is sufficient to tear across some fibres and compress others. But when there has been no hæmorrhage, the simple melting down of a portion of the nerve-fibres (promoted possibly by the passage through them of the nervous force, just as the platinum wire will be consumed under the transit of the galvanic current) will produce a solution of continuity, and stop the propagation of the nervous force. Such a view as this serves to explain the recovery of the palsy, on the supposition of the restoration of the normal nutrition of the nerve-fibres and the reunion of those which had given way.

I need scarcely remark that, reasonable as this view is, and sufficient as it is to account for the phenomena in many of the cases, it is very difficult of demonstrative proof. Nevertheless, I am not without hope that diligent microscopic investigation may yet demonstrate that such a solution of continuity of fibres does take place.

Let me now proceed to the first of the three forms of the hemiplegia of brain-lesion which I have described, namely, that with relaxed and flaccid muscles.

This form of hemiplegia occurs in two ways—first, without loss of consciousness; and secondly, along with more or less of coma.

The first variety, for the sake of marking it distinctly, you may call *simple hemiplegia*. The patient is suddenly or rapidly seized with loss of power on one side, without any comatose condition whatever.

Case CLXXII (vol. xxxvii, p. 194).—An excellent example of this form of hemiplegia—*the simple hemiplegia*—is to be found in a patient now in the hospital, James Scott, æt. 53, in Fisk ward, who has been subject to attacks of gout. His fingers exhibit deposits of urate of soda about the joints. He has had no other serious illness. The lung-sounds are healthy; there is a feeble but distinct mitral systolic bellows-sound, best heard at the apex of the heart.

This patient was admitted on the 29th of November, 1852, and the following history was obtained by my clinical clerk, Mr. Liveing:—Ten days since, while at work, he felt suddenly ill, was obliged to sit down, and discovered that he was unable to use his right side, but he did not lose his senses at all.

On his admission his condition was as follows:—considerable palsy of the right side of the face; articulation slightly affected. Tongue deviates to the right side. He cannot move his right arm at all; he is also paralysed in the right leg, but can move it very slightly. Sensation somewhat impaired in the arm. The pain produced by pinching is considerable in the leg, and less than might be expected in the arm, of the paralysed side. There is marked reflex action on tickling the sole of the right foot. The muscles of the paralysed limbs are perfectly relaxed, and quite free from any indication of rigidity. The arteries do not feel sound, being rather thickened. Pulse, 79. There is no evidence of disease of the kidney; urine natural.

Now I shall best explain to you the view I take of this case, by reading to you the diagnosis which I drew up after I had seen him, and which was recorded by Mr. Liveing in the case-book.

"Diagnosis.—Dr. Todd believes that there is, in this case, white softening, most probably of the left corpus striatum, and, perhaps, also of some of the fibres of the hemisphere round about it. He conjectures that it may have resulted from obstruction of a cerebral artery (perhaps from gouty deposits), giving rise to a gradual softening, and ultimately to rupture or deliquescence of fibres, which is the immediate cause of the sudden paralysis. The obstruction may be temporary only, and if the force of the circulation be sufficient to overcome it, or a free collateral circulation be established, the nutrition of the brain may go on as before, and the patient recover."

The patient was, on the whole, in so fair a state of general nutrition, that I was led to augur favorably respecting him. Still I might have added, that the failure to overcome the arterial obstruction, or to establish a collateral circulation, would have resulted in confirmed palsy with relaxed muscles, or, after a time, in slow contraction of the paralysed and wasted muscles,

and a state of permanent flexion of the forearm and hand, and some stiffness of the leg.

The progress of this case was as follows :

On the 3d of November, five days from his admission and fifteen from the attack, the following note was made in the case-book :—"Much better altogether; he can move the fingers of the right hand, and flex the elbow-joint of the same side. Sensation is also improved; he can distinguish two points on the back of the hand at an inch and a quarter apart; feels pain now readily on pinching. The right leg is also better—he can move it better—no pain in it, and the pain on pinching is less than it was; passed three pints and a half of urine in the last twenty-four hours, specific gravity, 1017; right pupil larger than the left."

On the 7th, all the symptoms had improved excepting the paralysis of the arm; on the 9th, the palsy of the face was much better, and he was able to protrude his tongue nearly straight. The inequality of the pupils was less obvious; on the 16th, he had regained strength so much that he was able to walk with assistance on each side.

This man improved steadily and uniformly up to the beginning of February, when his further progress was checked by an attack of gout in his left knee; this yielded in a few days to treatment, and he remained in the hospital till the 22d of February, when he was discharged, having recovered the power of the paralysed side almost completely, nearly three months from the attack.

It was particularly deserving of notice, that in this case the muscular power was restored without any permanent state of rigidity or contraction of any muscle or sets of muscles of either limb. In consequence of this favorable restoration of the paralysed parts, I would infer that a collateral circulation restored the nutrition of the softened fibres, and that whatever solution of continuity had taken place then, a process of union (by the first intention, as one would say, of external parts) had been established.

In some cases the improvement in the softened part of the brain is, apparently, accompanied with a complicated process of cicatrization of the brain-substance, and connected with

that is a change in the paralysed muscles, which, without regaining their normal state of nutrition, become rigid, and throw the limbs into a more or less permanent state of flexion. Others, again, of which case CLXVIII, related in the seventh lecture, is an example, make very little progress towards recovery, the limbs remaining in the relaxed and flaccid state which they had assumed at first. I shall, in a future lecture, call your attention to those modes of termination of hemiplegic cases of this class.

If this man's (Scott) arteries are diseased, as is most probably the case, it is not unlikely that sooner or later he will have a second attack, and that then the softening and the solution of continuity of fibres, and, it may be, hæmorrhage, will take place in the right hemisphere of the brain.

I have already detailed a striking example of the tendency of both sides of the brain to become diseased, causing double apoplexy (vide Lect. XXXVIII, Case CLXVI). I will now give you a more perfect example, not only in illustration of the tendency of the brain symptoms to recur by disease of the opposite hemisphere, but also showing how complete may be the recovery from the effects of the first attack of simple softening, and of simple hemiplegia consequent thereon.

Case CLXXIII.—Mr. J. R—, æt. 75, a gentleman who had filled with distinction for many years the office of actuary in a leading insurance office in London; he was a short, stout, plethoric man, of sufficient active habits both of mind and body. One night, in the beginning of the month of December, 1850, having been previously in very good health, although for some time gradually failing in general power, on leaving the drawing-room to go to bed, he found, after taking hold of a bedroom-candlestick with the left hand, that he was unable to lift the candlestick, and the arm immediately fell powerless to the side, completely paralysed. His consciousness was not at all impaired; so perfect indeed was it, and so much did he retain his presence of mind, that he at once directed his servant to fetch from his library the volume of the 'Encyclopædia' containing the article "Paralysis," that he might ascertain whether he was attacked with that affection. His usual medical attendant,

Dr. Woolley, of Brompton, was sent for, and on his arrival the leg had become quite paralysed, as well as the face and tongue. He was bled moderately, and treated chiefly with aperients.

I saw him two days after the attack, and found complete paralysis of the left arm and leg, with a perfectly flaccid and relaxed state of the muscles. The face and tongue palsy were quite complete. Sensibility was only very slightly affected, and there was no pain in the head. The heart's action was good—rather quick—and a slight thickness was perceptible in the radial artery. Speech was but very slightly impaired, and the intelligence was perfect.

I continued to attend this gentleman, in conjunction with Dr. Woolley, for many months. In the month of May of the following year he had regained his power to such an extent, that he was able to go out and enjoy walking exercise; and, ultimately, towards the close of the summer of 1851, he so completely regained his power, that no difference could be observed in the movement of the left leg from that of the right, excepting, perhaps, a slight stiffness, and he could use his arm and hand freely, and could grasp with full force. There was but one defect in the hand, namely, a slight stiffness and semi-flexed state of the second and third fingers, which interfered with the perfect use of his fork at table. This symptom was never removed.

I had frequent opportunities of seeing this gentleman during the remainder of 1851, and throughout 1852, during nearly the whole of which time he retained the full power over his left side. At the end of the former year he began to suffer from an irritable and inflamed bladder, which secreted pus and phosphate of lime in considerable quantities, and he would, under these circumstances, pass highly alkaline urine. This state was kept in check effectually by large doses of the dilute nitric acid.

So he went on till the middle of 1852, when along with his bladder symptoms he manifested a good deal of debility, and an extreme fidgetiness and restlessness, and a certain amount of peevishness of temper; his memory likewise rather failed, and he could not walk as he had previously done, being very easily fatigued. I had referred these symptoms to a gradually and

slowly progressive softening of one or both hemispheres of the brain; and I had prepared his friends for some sudden change, either a fresh attack of paralysis, such as he had previously, or one accompanied with apoplectic effusion, which might speedily terminate his life. And I also intimated that the attack would not improbably affect the left side of the brain, as previously the right.

One Tuesday night, towards the end of December, 1852, having passed the day in his usual health, and been very cheerful, he was suddenly seized, soon after going to bed, with paralysis of the *right* side, and comatose symptoms very rapidly supervened. He quickly fell into very profound coma, with stertor and flapping of the cheeks, and died after some hours in the course of the night.

The examination of the brain was conducted by my friend, Mr. Dixon, surgeon to the Royal London Ophthalmic Hospital. The right hemisphere, including the corpus striatum and optic thalamus, which must have been the seat of the original disease, appeared healthy—we failed to detect any mark of previous lesion; but it is not impossible that such mark may have escaped our search, as it was conducted under very unfavorable circumstances, in a small room, on a dark day in December. The left lateral ventricle was full of blood, which had flowed freely into the right ventricle, the third ventricle, and through the iter into the fourth ventricle. The left corpus striatum and optic thalamus were completely swept away by the gush of blood, and the white substance of the hemisphere outside them was torn up by a large coagulum, the nervous matter beyond being evidently softened, and breaking down readily under a slight stream of water. The arteries were extensively diseased, and there were numerous fatty deposits in the walls of the smaller vessels.

A third case may be here quoted, showing complete recovery after a first attack, and partial recovery after a second in which the paralysis affected the opposite side.

Case CLXXIV.—Mary Anne Godfrey, æt. 29, a housemaid, of temperate habits, and without any history of syphilis, was admitted into the hospital on May 21st, 1850, with hemiplegia of the *left* side (vols. xxxi, p. 147; and xlix, p. 116). The

paralysis of motion was not quite complete, as she was able to move the fingers of her left hand *very slightly*, but the sensibility of the palsied limbs was almost entirely suspended; there was ptosis of the left upper lid, the tongue when protruded diverged towards the paralysed side, and her articulation was very imperfect. The sounds of the heart were normal, and the urine, upon examination, was found to be healthy. The account which this patient gave of herself was this :—Her health had been tolerably good up to December, 1849, when she had an attack of rheumatism, for which she was treated in a London hospital; and in January, 1850, she had a fit (the first in her life), which lasted two hours, and which, from her own description of it, appears to have been of an epileptic character, but out of which she came with the full use of all her limbs. In the beginning of the following March she had a second epileptic seizure, which also lasted about two hours, at the end of which time, when she came to herself, she found that her left arm was paralysed, and her face drawn to one side; the palsy, however, did not last longer than half an hour. Three weeks subsequently to this she had a third and similar attack, which was also followed by transient paralysis of the left side. From this time she continued without the occurrence of any fresh fit up to May 17th (1850), on the morning of which day, after having slept very heavily the preceding night, she awoke with complete hemiplegia of the left side, and in this state she remained until she was brought to the hospital four days subsequently. She was at once subjected to a supporting plan of treatment, and, under the administration of quinine, iron, and an occasional purgative, together with a nutritious diet, she gradually recovered the use of her limbs, and was discharged on July 4th (1850), *i. e.*, about six weeks after her admission, perfectly free from every trace of paralysis.

[I regret to say, the condition of the paralysed muscles during this attack was not noted.]

After leaving the hospital, the health of this patient continued pretty good, with the exception of her being more or less liable to rheumatic pains, until the evening of November 6th, 1855—a period of more than five years—when she suddenly fell down in a state of insensibility whilst walking in the street. She was taken home, and the following morning a medical man was sent

for, who cupped her on the back of the neck. She continued, however, perfectly insensible until the night of the 7th, when she recovered her consciousness, but only to find herself hemiplegic on the *right* side; and on the 14th, the paralysis continuing, she was again brought to the hospital. On this occasion there was complete paralysis, both as regards *motion* and *sensation*, of the right arm and leg, and of the right side of the face and tongue; articulation was much impaired, and the muscles of the paralysed limbs were perfectly flaccid and relaxed, offering not the least resistance to the movements of flexion or extension. No reflex actions could be excited by tickling the sole of the *right* foot, nor by the application of a hot spoon to the same limb. A bellows-sound, accompanying the systole of the heart, was distinctly audible, and was loudest over the apex of the organ.* The patient was again put upon a supporting plan of treatment, consisting in the exhibition of ammonia, chloric æther, and small quantities of brandy, along with a generous diet; and, for the above medicines iron and quinine were after a time substituted. Under this course she gradually but slowly improved, and on February 28th, 1856, after a stay in the hospital of rather more than three months, she was again discharged, having completely recovered the sensibility of the paralysed parts, and having regained the power of voluntary motion in the lower extremity to such a degree as to be able to walk about the ward with the assistance of a nurse. The arm continued quite paralysed as to motion, with some degree of contraction of the flexor muscles of the fingers.

* This bellows-sound was observed during the patient's former sojourn in the hospital, and was probably due to an endocardial affection at the time of her rheumatic attack in 1849.

LECTURE XLII.

ON PARALYSIS.

HEMIPLEGIA WITH RELAXED MUSCLES.

In my last lecture I introduced the subject of Hemiplegia to you, and enumerated the various forms in which (looking at it in a clinical point of view) the affection is apt to occur.

You will remember that I proposed to you two forms of the simple hemiplegia, or that with relaxed muscles. In both, the attack of paralysis is sudden, or at least very rapid; but in the first form it is not attended by any affection of consciousness, while in the second it is accompanied by a comatose condition which lasts a longer or shorter time.

Now the first of these forms of the simple hemiplegia I illustrated by the case of J. Scott, who had been in the hospital, and by another case which I attended in private. Both these cases are examples of the affection occurring in the advanced period of life, at which time, indeed, the attack is most apt to take place—because it is at that time that the arteries undergo the changes which render them less fit as carriers of blood to the brain, and also make them brittle.

You will meet with cases of this kind at the earlier periods of life likewise; but at these ages the obstructed circulation will not arise so much from chronic disease of the blood-vessels as from a sudden arrest of blood in them, either by the formation of a plug in their interior, or by its detachment from some other part of the vascular system, and its impaction in one of the chief cerebral arteries.

The cases of this kind of hemiplegia recorded by Dr. Kirkes were aged respectively thirty-four, twenty-four, and twenty-four; and Dr Burrows has also contributed a case at the very early age of eleven, and another, most probably of the same

nature, aged nineteen.* These communications are of very great interest and importance; and the proved existence of cases of this kind will of course lead every practitioner to investigate the condition of the heart, and to seek for evidence of a rheumatic diathesis, or of attacks of rheumatic fever in the antecedents.

The case of M. Godfrey, quoted in the last lecture, might be possibly referable to a lesion of this kind.

It is at the earlier periods of life that you will meet with, also, the cases of what I propose to call choreic hemiplegia, and also most of the cases of epileptic and hysterical hemiplegia. You will, therefore, bear in mind that in investigating cases of hemiplegia at the early ages, it will be necessary for you to determine whether they have any connexion with these states of constitution.

The pressure of a tumour so far impairs the nutrition of surrounding parts of the brain as to induce white-softening, and consequent paralysis or other symptoms.

By and by I shall have to detail to you the particulars of a case of hemiplegia in a young person of nineteen, in which the diagnosis was for some time obscure, but which was referable to the form of the malady now under our consideration.†

I must ask you to remember, that obstructed blood-vessels are not the only cause of the softening, upon which this form of paralysis depends. A poor or vitiated blood, excessive mental effort, grief, fright, an abnormal deposit, a severe epileptic fit, the syphilitic poison, may so impair the nutrition of the brain, as to cause softening and solution of continuity of brain-fibres, even although the arteries be healthy and pervious.

You will recollect that I insisted much upon the importance of noting carefully, in your records of cases of hemiplegic paralysis, the condition of the paralysed muscles; and that I proposed to make that the basis of my arrangement of such cases for clinical observation. This subject is so important, that I must beg of you to excuse my referring to it again. In all cases of this description I would ask you to observe and note

* 'Medical Times,' 1853. No. 136.

† Vide infra, Case CXCVIII.

the following points: 1st, whether the muscles of the paralytic limbs are absolutely relaxed and flaccid, and offer no resistance to the extension or the flexion of the limb; 2dly, whether, when you attempt to extend the forearm upon the arm, you experience resistance from the biceps muscle, or, if you flex the one upon the other, you find the triceps to cause resistance; and in the lower extremity, whether you encounter similar resistance from the hamstring muscles, or from the rectus femoris; 3dly, whether the muscles are rigid, and maintain the limb in a more or less flexed state; and lastly, if rigid, to note the time at which the rigidity came on,—whether it came on simultaneously with, or soon after the palsy, the muscles being well nourished — or, at a remote period, with wasting of the muscles.

A very little reflection will show you the reasonableness of attaching importance to the state of the muscles in brain and spinal-cord affections. If you observe the results of experiments on animals recently dead, you will perceive in what an intimate relation the nervous and muscular forces stand to each other. You cannot touch a muscular nerve, or a part of a nervous centre in which muscular nerves are implanted, without affecting the muscles to which those nerves are distributed, however distant they may be. How instantly does strychnine signalise its presence in the blood, by the tetanic condition of the muscular system! This is not because strychnine has any special affinity for the muscles, but because it has the property of exalting the nervous force, of exciting the polarity of the nervous centres, and this in its turn can excite the muscular force. If, then, the state of the muscular force is an index to the condition of the nervous force and of the nervous centres in poisoning by strychnine, why may we not use it to enable us to form some estimate of the state of the nervous force in paralytic cases? And may we not with justice affirm, that when the muscles of a paralysed limb are in a state of rigid and tetanic contraction, the nervous centre at or about the lesion is in a very different state, *quoad* its power of generating the nervous force, from that in which it is, when the muscles are lax, flaccid, and entirely inactive?

No observer, so far as I know, has paid attention to the con-

dition of the muscles, exactly in the way that I propose. Lallemand, and, more recently, Durand-Fardel, seem to me to have done most in this way, and to have had a clearer appreciation of the importance of looking to the state of the muscles, than any other observers. But they have not sufficiently noted the occurrence of rigidity as an early and a late phenomenon.

The lesion which gives rise to the paralysis in these cases, is "white-softening," *i.e.*, softening of the brain substance without any discoloration; the cerebral matter becoming so soft that a gentle stream of water poured on it from a height breaks it up (which has no such effect on a healthy brain), and in some cases, indeed, becoming diffluent, so as to assume a creamy consistence.

The suddenness of the attack of paralysis is due (as I suppose) to rupture of the softened fibres. It must, I think, be admitted, that fibres, wanting in their normal consistence, may, nevertheless, propagate the nervous force, and that a softened brain may more or less perfectly minister to volition or sensation. Persons, whose brains are wholly or partially in this condition, often experience uneasy sensations in the limbs, which subsequently become affected, such as neuralgic pains, numbness, coldness, &c. By and by fibres give way, sometimes all at once, often gradually one after another, and the paralysis ensues with proportionate rapidity.

The second variety of the first class of hemiplegia, is characterised by the suddenness of the attack, and by more or less of coma soon after, or simultaneously with the paralysis. This form of hemiplegia, to which I wish to direct your attention today, is more common than the simple variety of which I have hitherto spoken, and it is also of a much more serious nature, as it often quickly terminates in death. It comes on in this way:—A man is walking about, or following his usual occupation, or doing any particular action, when his arm or leg suddenly becomes paralysed; at the same time he experiences a certain amount of shock, and soon the other limb on the same side also becomes affected. In the course of an hour or two, he falls into a sleepy state, which passes into coma, the breathing becoming stertorous, the face drawn to one side, and the flaccid paralysed cheek flapping to and fro, which is always a symptom of

very bad omen. Sometimes, however, the coma and palsy come on simultaneously; at other times a fit of epileptic character occurs, from which the patient recovers paralysed, and then passes into coma.

In illustration of this form of hemiplegia, especially as regards its mode of invasion, I shall refer you to the following case very lately in the hospital.

Case CLXXV. — Elizabeth Harvey, aged fifty-four, who was admitted into Lonsdale ward, on May 3d, 1853 (the notes of whose case have been kept by my clinical clerk, Dr. Plowman, in the most praiseworthy manner). (Vol. xxxix, p. 258). She was in her usual state of health, until three days before her admission into the hospital, when she experienced a shock, on recovery from which she found that her left arm and hand hung useless by her side. She soon passed into a state of stupor, her breathing became stertorous, and she was brought into the hospital in an imperfectly comatose condition. By talking loudly to her, we could get her to answer questions, and in this manner we elicited the account which I have just narrated. There was complete hemiplegia on the left side, the muscles of the limbs being all completely relaxed, with the exception of the biceps of the arm, which, on extending the forearm on the arm, became firm and resisting. In this last respect, the case scarcely comes under the class of hemiplegia with relaxed muscles; but I quote it as illustrating that form in all the main points; by and by, I shall refer to this particular symptom. Our patient complained a good deal of headache; the left side of the face was paralysed, the mouth being drawn to the right; the tongue, when protruded, had a marked divergence towards the paralysed side; there was some difficulty of swallowing; and thus early in the case there was slight puffing of the left cheek, and she passed her urine and evacuations under her, as if from paralysis of the sphincters. The following day, May 4th, she became perfectly comatose, and the left cheek flapped to and fro with each expiration. This phenomenon of flapping of the cheek, as I have already remarked, indicates a very perfect paralysis of the buccinator muscle, and is usually associated with an extensive paralysing lesion. The pupils were dilated,

the right being larger than the left; and the pulse was slow, being on the 3d, 56, and on the 4th, 52, while the respirations were eighteen in a minute.*

Let me detail to you another case in which the muscles were completely relaxed, not even the biceps becoming rigid on extension of the forearm.

Case CLXXVI.—Jane Power, aged sixty-three, admitted September 18th (vol. xii, p. 118). On the 16th of September, she complained of pain in the right side, the nature of which could not be ascertained. This pain passed off, and on the 17th, she was as well as usual. On the evening of the 18th, she suddenly complained of being very ill, and put her hand to her right side. It was found that the whole of that side was completely paralysed, and that it was cold. She did not become unconscious at once. She was immediately brought to the hospital.

On admission she was found in a state of stupor, and took no notice of those around her, but when roused would answer questions apparently correctly. Her breathing was somewhat stertorous; deglutition rather difficult. She put out her tongue when asked to do so, and it deviated to the right side. The pupils were much contracted. Pulse 100, very compressible. The paralysed limbs were quite relaxed; when the arm was lifted up and let go, it fell upon the bed as if dead. All her evacuations were passed in bed, the sphincters being probably paralysed.

On the 20th and 21st, she seemed to have rallied a little, and answered questions more readily, and swallowed better; the pupils were less contracted. On the 22d, without apparent cause, she became again less conscious; pulse 120; extremities cold. She lingered on till the 28th, the paralysis remaining unchanged, and the circulation failing, the lungs also becoming much congested. She died at seven o'clock a.m., on the 28th.

The lesion in this case was extensive white-softening of the middle of the left hemisphere above the roof of the lateral ventricle, in the centre of which a clot as large as a small

* See the conclusion of this case at p. 733.

walnut had formed. A small opening had been formed into the ventricle. The cerebral substance around the clot was much softened, and had some small extravasations in it. There is unfortunately no mention made in the record of the case of the condition of the arteries, so that we are left in the dark as to the cause of the white softening.

The case of Ware, detailed in Lecture XXXVII, p. 664, is a good illustration of this form of hemiplegia with relaxed muscles. This patient, as you will remember, was suddenly seized with paralysis at night, so that it was impossible to say whether his consciousness was, or was not, affected at the moment of the seizure. When he awoke, he found the right side of his body paralysed, and soon after he fell into a comatose state, which lasted for a considerable time. On recovering from the coma, he was still hemiplegic, and the paralysed muscles were completely relaxed. In this state, he was brought into the hospital; he died of erysipelas of the head and face, and thus an opportunity was afforded of ascertaining the condition of brain associated with the relaxed state of the paralysed muscles. Upon examining the brain of this man after death, the left corpus striatum was found to contain a small cavity, which was full of fluid; the cerebral substance immediately around this cavity was completely softened; there appeared to have been no attempt at the healing process; and, with the exception of the softening surrounding the cavity, the rest of the corpus striatum was in a healthy condition.

Case CLXXVII.—There is at present a man in Rose ward, of the name of Shea, who well illustrates this form of paralysis and its progress (vol. xxvi, p. 98). This man was admitted into the hospital for the first time on April 4th, 1849 (four years ago), and his history is this:—On April 2d, 1849, while going down to Sydenham by rail, he had a fit in the carriage, and when the door was opened, he was found lying on the floor of the carriage, with paralysis of the right side of his body. A medical man, who was called in to see him, bled him to ten ounces, and ordered him some aperient medicines; and two days after the attack, he was brought to the hospital. He then had hemiplegia of the right side with complete relaxation

of all the paralysed muscles, but the biceps contracted on making extension of the forearm; his face was drawn to the left side, the tongue, when protruded, diverged towards the right, and his speech was a good deal affected. After a time he recovered to a slight degree the power of the right arm; since then he has been more than once an inmate of the hospital, and he is now again a patient; I shall presently have to refer to him more particularly in illustration of the changes which such cases undergo in the course of time (p. 735).

Having detailed to you these cases illustrative of this form of hemiplegia, I shall now speak of the different modes in which it may terminate.

This variety of hemiplegia may terminate in one of four ways:—1st, it may end in death, and that, too, pretty rapidly; 2d, it may terminate in permanent paralysis, the paralysed muscles remaining relaxed; 3d, partial recovery of the paralysis may be attained, the paralysed muscles becoming wasted, and passing into a state of rigidity; and, lastly, there may be complete recovery from the paralysis.

When the patient dies, death results from the exhaustion consequent upon the shock and the suspension of function of a portion of the cerebro-spinal centre. The rapidity of the fatal event will be determined mainly by the extent of previous brain disease, or by the quantity of blood effused, or by the part of the brain affected—sometimes by the intensity of the shock. With regard to the part of the brain affected, I think it is quite certain, that when the apoplectic clot compresses directly the pons Varolii, or the medulla oblongata, or parts immediately connected with them, death will take place much sooner than when any other part of the brain is involved. Moreover, in cases in which the brain-lesion has, of itself, exercised a very depressing effect upon the powers of life, it is by no means to be overlooked, that the duration of life may be materially influenced by the treatment adopted, at the time of, or soon after the attack. Some will recover, to a certain extent, the immediate effects of the brain-lesion, but will succumb sooner or later by the failure of general nutrition.

In the sequel of the case of Elizabeth Harvey, part of which

I have read to you (Case CLXXV), you have a good illustration of a common mode of fatal termination of hemiplegic cases, when effusion of blood has occurred.

On the 4th of May she passed into a state of complete coma, the palsy remaining as before; the pupils becoming more dilated, the right appearing rather the larger; the pulse as low as 58. On the 5th the comatose state continued, and she was evidently sinking. The pulse had fallen to 50, and it was interesting to notice that the heart's action was 90—a striking proof how much the force of the heart was weakened, for nearly each alternate stystole was so weak as to be incapable of generating a pulse at the wrist. She died this night at ten o'clock, six days after the first attack of palsy.

Before examining the body after death, I ventured to make the following diagnosis:—I stated that I believed the symptoms were due, primarily, and essentially, to white-softening, consequent upon disease of the cerebral blood-vessels; that hæmorrhage had taken place, with more or less laceration of brain-substance. The latter point rested chiefly upon the rigidity observed in the biceps muscle of the arm whenever the forearm was extended on it. From the flapping of the cheek, I thought that the hæmorrhage was probably considerable; and that it was not unlikely that blood had been effused into the lateral ventricles.

Upon examining the brain the following were the appearances noticed:—The membranes appeared healthy; there were two or three enlarged Pacchionian bodies: there was no subarachnoid effusion. The brain-tissue generally appeared somewhat soft, and the convolutions of the anterior and middle lobes of the right hemisphere (the paralysis was on the left side) were greatly wanting in consistence, and readily gave way under a fine and gentle stream of water carefully poured on them. The brain did not appear to be at all congested, and there was no effusion into either of the lateral ventricles. On slicing the hemispheres, a small clot of blood, about an inch in diameter, was found in the right corpus striatum, and the whole of this body, and the surface of the optic thalamus of the same side, were also extremely soft, and were readily broken down by allowing a gentle stream of water to fall on them. The left

hemisphere appeared to be healthy, as did also the cerebellum. We then proceeded to examine into the cause of the white-softening and apoplexy, and we found by microscopic examination evidence of most extensive disease of the small arteries and capillaries of the brain. The vessels of the softened portion of the corpus striatum immediately surrounding the clot were thickly studded with oil globules, which, in some situations, were aggregated into dark masses, so large as here and there almost to fill up the vessels. The minutest capillaries, as well as the larger arteries, exhibited these deposits, and few could be discovered free from them. Acetic acid rendered the walls of the vessels and the globules clearer, but did not bring out the muscle-cells, whence we infer that, in part at least, the fatty deposits resulted from the degeneration of these fibres. No effervescence occurred on the addition of this reagent, nor did any of the globules appear dissolved by it. When, however, a drop of ether was added to one of the vessels, the globules were dissolved, and a greasy stain remained on the glass on the evaporation of the ether. Thus we had sufficient chemical evidence of the fatty nature of these particles. Many of the vessels of the vesicular matter of the convolutions exhibited this same fatty disease, only in a less degree.

Our diagnosis, then, appeared to be correct in all the main points. It was wrong, however, in the estimate of the size of the clot, and in stating that the hæmorrhage had extended into the lateral ventricles. This error might, I think, have been avoided, had I allowed sufficiently for the length of time (six days) which elapsed from the seizure. The great extent of the softening sufficiently accounted for the comatose state, which ensued so early, and could scarcely have been produced by so small a clot, in the midst of healthy brain. The brain had already, before the rupture of the blood-vessels, been much diseased, and had no power to rally after the severe shock which was inflicted upon it; but death will not occur, under these circumstances, so quickly as when an extensive hæmorrhage takes place into the lateral ventricles.

I may refer you to the cases of Thurston and Regan, described in Lects. XXXVIII and XXXIX, for instances of the speedy death of patients where large clots are formed in connexion with

disease of the same kind as that observed in this patient. And, in the case of Catherine Williams, detailed in Lects. XXXIV and XXXV, you will find a well-marked instance of death under this form of hemiplegia, where there was no clot at all, and in which, if any blood had been effused, it must have been in very small quantity.

Other patients die from the effects of bed-sores, and with indications of the failure of general nutrition, promoted, no doubt, by the depressed state of the whole nervous system. The case of J. R., quoted in my last lecture, is an example of this.

This second mode of termination may be illustrated by the case of Ware (p. 664), in whom death did not ensue on lesion of the brain, for he recovered from the loss of consciousness and other immediate effects of the attack, and, indeed, from the paralysis to some degree (being just able to move the fingers slightly), but on erysipelas. I believe, however, that if he had survived the erysipelas, he would never have completely recovered the power of his limbs. Around the excavation which was found in the corpus striatum, there was no indication of any contraction or cicatrization, and the absence of this accounted for the condition of the affected muscles. There was just enough of the corpus striatum left to enable the patient to recover some power over the paralysed parts, but not sufficient to restore them to their normal state. You will remember that as much as six months had elapsed from the paralytic seizure to his death, affording ample time for a cicatrizing process to have been established.

An illustration of the third mode of termination is to be found in the case of Shea, now in Rose ward (Case CLXXVII). This man recovered from the immediate effects of the attack, but the paralysis remains; and all his symptoms favour the opinion that in his case there has been rupture of some blood-vessels, with the formation of an apoplectic clot in, or very close to, the left corpus striatum, and hence the very complete paralysis of the right side which at first took place. After a time he recovered some power over the leg and arm, but the muscles of the paralysed limbs have gradually assumed a remarkably rigid condition, the arm being drawn to the side, the forearm flexed on the arm, the wrist bent on the forearm, and the fingers closed

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on the palm. When an attempt is made to extend the arm, great resistance is offered by the flexor muscles, particularly the biceps, which feels hard and rigid, and as if in a somewhat tetanoid state. At the same time, all these muscles have wasted very much, and are much smaller than those of the other side. This represents a maximum of this condition; but, in many instances, contraction is limited to a few muscles only, or to the biceps, or the flexors of the fingers, or to portions of them, so that a patient will recover with permanent flexion of one or two fingers. I shall recur to this point in my next lecture.

Lastly, and last frequently, recovery may take place without any rigidity or change in the muscles, other than a gradual restoration to their normal condition. Of these, I related to you two striking instances in my last lecture in the cases of J. Scott, and J. R. In J. R.'s case a slight stiffness of two fingers remained, and also some rigidity of the semi-tendinosus muscle.

When recovery takes place, you should observe the order in which the palsied parts become restored. The leg, face, and tongue are generally the first to recover; sometimes the face and tongue are first, at other times the leg; almost invariably the arm, as if it were most severely struck, from being nearest to the seat of lesion, requires the longest time to shake off the paralysing influence of the seizure. But even this sometimes begins to recover before the leg.

Such being the Clinical History of the first form of Hemiplegia, in its two varieties, what can we say as to its Pathology?

It may, I apprehend, be pretty confidently affirmed respecting the first variety, namely, that in which there is simply paralysis without coma, occurring *suddenly* or *with great rapidity*, that it is always dependent on softening, without or with a clot; that the softening is of the colourless kind, and that when a clot exists, it is so small as not to exercise pressure on neighbouring healthy parts of the brain; and, respecting the second variety, in which more or less of coma prevails, that it is also dependent on softening of the same nature, without or with a clot. When without a clot, the softening is of considerable extent, and may perhaps be associated with some previous morbid deposit.*

* In illustration of this form of softening in connexion with strumous deposit, the reader is referred to Case CXCVI, Lecture XLV.

When with a clot, which is the more common case, this is either of large size, or it is situated in some part where it can compress central and important parts of the brain.

The evidence now accumulated respecting the lesions which give rise to these two forms of hemiplegia indicate, I think, very distinctly that they result from defective circulation through the brain, and enfeebled nutrition of the cerebral matter. In some instances, actual obstruction of important arterial channels can be shown; in others, there is a marked degeneracy of a large portion of the arterial and capillary system, which may have preceded or gone on simultaneously with the cerebral degeneration. In all cases the cerebral disease reaches such an extent, that the vesicular matter imperfectly generates the nervous force, and the fibrous matter becomes a bad conductor of it, or even a non-conductor, or its continuity is interrupted, and so its power of conduction is rendered, in the one case, physiologically, in the other, mechanically impossible. And, if the softening of brain have been of sufficient duration, there will be found in it the large vesicular bodies, containing fatty particles in a state of minute division, which indicate a further degeneracy of the brain tissue, or an attempt at a reparative process.*

There are some other points which I must notice, such as the differential diagnosis and the prognosis of this form of hemiplegia; but these I would rather postpone until I have spoken of the other forms.

Let me now ask your attention to a few remarks upon the treatment of cases of this description. You are called to a patient who has become the subject of sudden paralysis and relaxation of the muscles of one half of the body: what can be done? We will suppose that you are satisfied that the attack results from the changes connected with white softening and diseased arteries. You will, naturally, reflect upon the changes that have been going on in the brain. The nerve matter has been undergoing a change which, either by alteration of structure or by a solution of continuity, or by both, renders

* *Vide* p. 734.

it incapable of propagating the nervous force. One or more blood-vessels have given way; or, if not, their rupture is imminent. You will ask yourself, what can I do to prevent this threatening hæmorrhage, or to check it if it have begun, or to restore the conducting power of the nerve-fibre?

I believe that the most important end for the practitioner to aim at, in the early treatment of these cases, is to keep down the frequency and force of the heart's action, by means not depressing to the vital powers. For this purpose the strict maintenance of the horizontal posture is of the highest moment; and when the patient is conscious, it is most desirable that the mind should be tranquillised by every means. It will, of course, likewise be necessary to remove all local impediments to the easy flow of the circulating fluid; and it is as well that the head should be slightly raised, sufficiently to prevent gravitation favouring the escape of blood from the ruptured vessels, but not so as to create any impediment to the flow, which might embarrass the action of the heart.

To remove any distant source of irritation from the alimentary canal, which may be operating injuriously on the brain, the bowels should be cleared; and, in order that there may be as little effort as possible on the part of the patient in the expulsion of the contents of the bowels, it is expedient that this should be done by enema; but if this fail, and the vital power of the patient do not forbid it, you may give croton oil, a drop or two of which, placed on the tongue, will operate freely; or calomel, in powder, to the extent of five or ten grains, which may be similarly administered.

I would advise you to limit the further administration of drugs, to giving some slight corrective, as an alkali,—ammonia being, on the whole, the most appropriate—unless, indeed, you find the patient in an extremely prostrate condition, when it will be necessary to combine with it the cautious exhibition of other stimulants and restoratives, as chloric ether, brandy, &c.

The question of bleeding will arise; and, under the popular notion, that all head attacks are accompanied and caused by the rush of blood to the head, you will be pressed to have recourse to this expedient.

There are three objects to be attained by bleeding: first, to

diminish an undue amount of blood in the head; secondly, to check hæmorrhage or to prevent it; and lastly, to quiet the heart's action.

Let me briefly point out to you the circumstances under which bleeding is inadmissible.

If the patient be cold and collapsed, it is clear you should not take blood; nor should you have recourse to this practice if the heart's action be very feeble or intermittent; nor if there be an anæmic state; nor if the patient be of very advanced age; nor if the evidence of extensive disease of the arterial system, or of the heart, leave no doubt on that head; nor would it be desirable to bleed if it were clear that already a large amount of hæmorrhage had taken place into the brain.

Should none of these objections exist, then you will have to consider whether any or all of the indications above named need to be fulfilled, and whether bleeding (local or general) promises to fulfil them.

As to the first indication—namely, the diminution of an undue amount of blood in the brain, I think modern investigation of the actual state of that organ clearly points out, that the brain is not in a hyperæmic state, in the cases in which the form of hemiplegia under discussion is likely to occur.

Will taking blood check or prevent hæmorrhage? The sudden or rapid abstraction of a moderate quantity of blood, either from the arm or temple, or by skilful cupping from the nape of the neck, may, I can conceive, check hæmorrhage; and with this object it is, sometimes, a very justifiable practice, but the quantity taken should not be large. Now and then bleeding helps to diminish the frequency and force of the heart's action; but here, again, the quantity of blood withdrawn should be moderate, for the removal of much blood is apt to quicken the heart's action and render the blood poor.

I would have you to look upon this question, to bleed or not to bleed, as almost the most important one you will have to decide; and, judging from my own experience on this point, as well as from the results of the practice in a large number of cases collected from various sources, as I pointed out in a former lecture (Lecture XXXVIII, p. 680), I have come to the conclu-

sion that, in cases of white softening, with or without hæmorrhage, you are less likely to err by omitting rather than by adopting the practice.

It sometimes happens that in these cases a rigidity of the muscles comes on very early, which indicates an inflammatory process going on around the clot, which may end in the formation of pus and abscess, and is to be combated by the use of mercury. But you must be careful to distinguish this from the muscular rigidity which is of late occurrence, and results from a restorative effort of nature; and with which it is, therefore, not desirable that you should interfere.

You will often be consulted as to "some expedient for promoting the restoration of the paralysed limbs to their normal condition." To this question, after having given a fair trial to the various means which have been proposed for this purpose, I must reply, that I know of nothing which more decidedly benefits the paralysed limbs than a regulated system of exercise; active, when the patient is capable of it; passive, if otherwise. As to the use of electricity, which is now much in vogue, or the employment of strychnia, which has been strongly recommended, I feel satisfied, as the result of a large experience, that the former requires to be used with much caution, and that the latter is very apt to do mischief, and never does good. I have seen cases in which, after the employment of electricity for some time, that agent has apparently brought on pain in the head, and has excited something like an inflammatory process in the brain. And strychnia is also apt to induce an analogous condition of brain, and to increase the rigidity of the paralysed muscles.

Some good may occasionally be effected by the use of frictions, or cold water, or shampooing, all of which tend to improve the general nutrition of the nerves and muscles.

In my next lecture, I propose to speak of that form of hemiplegia which is associated with a more or less rigid condition of the paralysed muscles.

LECTURE XLIII.

ON PARALYSIS.

HEMIPLEGIA WITH RIGID MUSCLES.

You will remember that the first kind of hemiplegia of which I spoke was that which is accompanied with a relaxed state of the paralysed muscles, and that I described two forms of it—the one in which there is sudden paralysis, with a relaxed condition of the paralysed muscles, without loss of consciousness; the other in which these phenomena are associated with loss of consciousness, to a greater or less degree, at the moment of, or soon after, the palsy-stroke.

I mentioned, also, that the second kind of hemiplegia is where the muscles of the paralysed limbs are rigid, and where this rigidity comes on simultaneously with the paralysis, or very soon after it. You must bear in mind that a distinctive feature of this hemiplegia is the very early period at which the muscles assume this rigid condition. This is the more important, inasmuch as we meet with another form of hemiplegia with rigid muscles, in which the stiffness gradually supervenes a *long time* after the paralytic seizure, and may succeed to the relaxed condition of the paralysed muscles.

My purpose, to-day, is to bring before you examples in illustration of the clinical history and morbid anatomy of hemiplegia, *with early rigidity of the paralysed muscles,* and to make some remarks on the pathology of this form of paralysis.

Now of this kind of hemiplegic paralysis, you will in practice meet with two varieties; the one, in which the rigidity of the paralysed muscles is very slight, and confined to one or two muscles; the other, in which it is considerable, and affecting all, or nearly all, the muscles. The former of these is very apt

to occur in those cases of hemiplegia in which most of the paralysed muscles are flaccid, one or two only being in a rigid condition; and, as illustrations of this form, I adduced cases in my last lecture, in which there was a greater or less impairment of consciousness—where a clot had been formed with laceration of brain-substance, and where hemiplegia resulted, accompanied with a flaccid condition of all the muscles of the paralysed arm and forearm, except the biceps; other cases will occur in which there will be slight rigidity, not only of the biceps, but also of the triceps and the flexors of the fingers, and in a still less degree of the hamstring muscles and the biceps femoris. In many cases of this description, the rigidity of these muscles will not be apparent, unless they are thrown into action by exciting their antagonism. Thus, when you attempt to extend the forearm upon the arm, you will find that the biceps will become more or less stiff and rigid, and resist the extension; and so also will the triceps resist flexion; and, in like manner, will extension of the fingers be resisted by the flexores digitorum. In general the actual assumption of the rigid state, or the tendency to assume it, is more marked in the flexor muscles than in the extensor, and in the upper than in the lower extremities. It likewise very rarely affects the muscles of the face, or any other paralysed muscles than those of the limbs; but sometimes the muscles of mastication are involved, as the patient, although insensible, will resist powerfully any attempt to open the mouth.

This condition, of slight and partial rigidity of muscles, is that of most frequent occurrence in the hemiplegia caused by an apoplectic clot. My idea as to its cause is, that it depends upon a state of irritation, propagated from torn brain to the point of implantation of the nerves of the affected muscles. But, you will ask, why is it that in some cases of clot the hemiplegia will be accompanied with complete relaxation of muscles, while in other cases the rigidity of which I have spoken exists? The answer to this question is as follows: in the cases where there is no rigidity the clot lies in the midst of softened brain, and has not in any degree encroached upon sound brain; but when rigidity exists, the clot has extended beyond the bounds of the white softening, and has torn up to a greater or

less extent, or irritated, sound brain. I leave this explanation to be tested by further experience and observation.

I am anxious to relate to you one or two cases in addition to those which I have already detailed in my last lecture.

Case CLXXVIII.—Mrs. C—, aged fifty-seven, had been for some time in a depressed and nervous state, with failing memory. One day at noon, when walking into her drawing-room, she suddenly became paralysed on the right side, and would have fallen had not the servant caught her; she became speechless, and the only sign of consciousness she showed was evinced by her weeping at the sight of her daughters. She quickly passed into coma with collapse. I saw her soon after, with Mr. Dunn, of Norfolk Street.

We found her lying in a state of insensibility, from which, by very loud speaking, she could only be roused so as to open her eyes; she was breathing slowly, but without stertor. There was hemiplegia of the right side, with rigidity of the biceps, which resisted extension. She kept her mouth firmly closed; and when an attempt was made to open it, she resisted so powerfully that it was found impossible to introduce anything in the natural way. Two of her teeth on the right side were gone, and through the vacancy fluid could be introduced, which she swallowed with facility. On introducing the pipe of an enema-syringe, it was found that the sphincter offered no resistance, and was paralysed. She quickly showed marked indications of collapse, on taking away a few ounces of blood by cupping. Reflex actions could be produced by tickling the sole of the right foot. The pupils were much contracted.

These symptoms continued till the morning of the third day, when the coma became profound, and she died on that day. On examination, it was found that the left hemisphere, just external to the corpus striatum, contained a large clot, the size of a bantam's egg, dark and firm. This clot had torn up the surrounding brain substance, and had excavated a cavity for itself; the cerebral substance around this clot did not seem to be particularly softened, but presented a very peculiar appearance, in consequence of being studded over by numerous small coagula (capillary apoplexy). The corpus striatum was firm, and seemed

compressed by the clot. The optic thalamus was natural. The arteries of the brain, to their minutest ramifications, were studded with atheromatous deposits. The skull was very thick; in front, seven eighths of an inch.

It is most probable that in this case, owing to the disease of the arteries, there first occurred a softening of a considerable portion of the left hemisphere, just outside the corpus striatum. In this softened portion several small vessels gave way, and a large hæmorrhage broke down the softened cerebral matter and encroached upon the healthy brain, compressing the corpus striatum.

Here is another instance of a fatal case of this form of hemiplegia:

Case CLXXIX.—A man named Frost (vol. xxxi, B, p. 15), a water-gilder, of intemperate habits, was brought into the hospital in a state of coma. It was then found that his right side was paralysed; the right side of the face seemed motionless, and the right arm also, which exhibited rigidity of the biceps and triceps muscles; the leg was partially paralysed, and was frequently moved in a jerking manner. Pupils contracted. He was bathed in a profuse perspiration, and his heart was acting feebly; pulse 80. No satisfactory history of his attack could be obtained, beyond that it had occurred suddenly, and that he was immediately brought to the hospital.

On the following day (Feb. 14th) he became more conscious, but still remained very stupid; the right arm and leg paralysed, the arm being rigid, but only as regards the biceps and triceps; the leg twitched convulsively now and then; pupils contracted and equal; deglutition not impaired. On the 21st, there was no other change than an improved state of consciousness, and a slight increase of power in the leg. On the 25th, he was attacked with severe diarrhœa, under which he sank rapidly.

The *post-mortem* examination revealed softening of the white substance of the left hemisphere, outside of, and on a level with, the optic thalamus. Within this the optic thalamus was broken down, to the extent of its external two thirds, by a clot of quite recent black blood, which encroached on the corpus striatum, having slightly torn and compressed it. The nervous

matter immediately around the clot was discoloured by intermixed blood; that external to it was softened, and free from discoloration. In the latter the nerve-tubes were found to exhibit their natural structure; in the former there were numerous nerve-tubes, with blood-corpuscles, and numerous vesicular bodies filled with highly refracting fatty particles.

Now, in this case, the existence of the compound cells in the softened brain substance immediately around the clot seemed to me to indicate that a process of softening had been going on in the brain, anterior to the rupture of blood-vessels. This rupture was followed by laceration and compression of parts immediately influencing muscles, as the corpus striatum and optic thalamus; and hence, not only was the lesion paralysing, but likewise, to some extent, irritative, giving rise to the rigid state of the biceps and triceps muscles.

A much more interesting form of the second kind of hemiplegia is that in which there is considerable rigidity of all the muscles of the arm and forearm; where the arm is kept at an angle with the trunk (and sometimes these patients hold it across the chest), the forearm being flexed on the arm, and the fingers bent on the palm. In these cases the paralysed muscles appear to be firm and contracted, and sometimes in an almost tetanic state, and offer considerable resistance to extension or flexion, which frequently also excites a good deal of pain. When the rigidity is of this nature, the paralysis is generally not complete, a certain degree of the power of moving the whole limb or some part of it being still retained; and very frequently sensibility is affected, being sometimes obtuse, but oftener in an exalted condition, while it not uncommonly happens that reflex actions are also considerably exalted. In the latter cases, you will find the excitation of reflex action produce a considerable degree of pain. No doubt the same cause, which gives rise to the rigid condition of the paralysed muscles, contributes to produce the exaltation of the reflex actions. In hemiplegic paralysis reflex actions are usually most marked in the lower extremity; indeed, it is very rare to see them well developed in the upper; but in these cases the application of a stimulus to the palm will sometimes excite them in that limb, and in the lower extremity they will seldom

fail to be produced by stimulating the sole of the foot, or pulling a hair of the leg.

This form of hemiplegia sometimes occurs in surgical practice, in consequence of a blow on the head, with depression of bone, or from considerable hæmorrhage within the cranium, such as results from injury of the middle meningeal artery, or one or more of its branches. The hæmorrhage, thus produced, separates the dura mater from the adjacent bone, and exerts pressure on the corresponding surface of the hemisphere, and this, in turn, gives rise to paralysis of the opposite side of the body, accompanied, very often, with a rigid condition of the paralysed muscles. Indeed, when hemiplegia with a rigid state of the muscles supervenes soon upon some injury to the head, you may almost always make a certain diagnosis, that it is the result of irritant compression of the opposite hemisphere, by depressed bone or by hæmorrhage outside or inside the dura mater. A well-marked case of the kind occurred some time ago in Albert ward. I happened to come in as the patient was admitted, and found him lying in a comatose state, with great rigidity of one side of the body, while the other was in its normally flaccid condition. This immediately directed my attention to the side of the head opposite the rigid muscles, and, after death, we found a large effusion of blood on that side, between the dura mater and the bone, the result of injury to the middle meningeal artery.

Sometimes inflammation of the pia mater or arachnoid causes an accumulation of puriform fluid in the sub-arachnoid or arachnoid spaces, which, by compressing the corresponding surface of the hemisphere, gives rise to this form of paralysis on the opposite side of the body. A good example of this occurred in the following case:

Case CLXXX.—A woman named Wilson, aged thirty-five, (vol. xxxi, B, p. 51), was admitted with syphilis into the surgical wards; but symptoms very like those of typhus fever having manifested themselves, she was transferred into Lonsdale ward, where she came under my care. For a day or two, we were at a loss to determine whether she was suffering from arachnitis or typhus fever; for, as you know, it is often a matter of great difficulty,

and indeed impossible at first, to distinguish the effects of the typhus poison from those of meningeal inflammation upon the nervous system. When she was admitted into our ward, on the 8th of March, her symptoms were as follows: a flushed face, hot and dry skin, tongue dry and coated with a thick black fur, sordes on the teeth, intense thirst. She lay constantly on her back; the bowels were very much relaxed, and her pulse was small and frequent, 120, and compressible. She had vomited two days before admission into the medical wards, and had a shivering, and complained very much of headache, not referred to any particular spot. On the 9th there was a muttering delirium; she passed all her evacuations under her; the tongue was still very dry and black, with sordes on the teeth; pulse 112. Next day, the 10th, much more stupor; decubitus on the back. A new symptom now presented itself; this was hemiplegia of the right side; the palsy of the right upper and lower extremities, especially of the former, was accompanied *by a rigidity of the muscles,* described in my notes of the case as "almost tetanic." The tongue was protruded with difficulty to the right side, the *left* pupil was fully dilated, and the right was also dilated, but less so than the other. She died on the 11th, and after death we found that a copious effusion of pus had taken place in the arachnoid sac, over the surface of the left hemisphere of the brain, and that, being confined by certain adhesions, it had compressed and hollowed out a cavity on the corresponding surface of the convolutions. Lymph was effused over the whole surface of the hemisphere, inwards to the falx, and forwards to the fissura Sylvii.

This case well illustrates the mode of formation of this kind of hemiplegia, showing that it is due to a cause which exercises at once a paralysing and irritating influence on the brain. This influence is propagated to the spinal cord, and through the nerves implanted in that portion of the nervous centre, to the muscles of the paralysed limbs, in which it excites a state of contraction. This effect is analogous to that produced by the continued action of the electro-magnetic machine, which you sometimes see in use in the hospital; and just as a rapid succession of electric shocks may maintain a constantly rigid condition of muscles, so may continual shocks

of nervous force, due to irritative pressure, bring about a similar result.

An affection of the substance of the brain, of an irritative character, may give rise to this form of hemiplegia. Of this you will find a good instance, in a patient lately under our observation, of which Dr. Plowman has kept an excellent record.

Case CLXXXI.—The subject of the case was Mary Reeves, a cook, forty-nine years of age. She appears to have freely availed herself of the opportunities which the kitchen afforded to indulge in intemperate habits. She had suffered, for some time before her admission, from severe pain in the right arm and leg, which her medical man told her was gout. One evening, while out, she was seized with pain in the right foot, which was so severe as to make her limp in her gait, and oblige her to obtain assistance to walk home. The next day, however, the pain was better, but a paralytic state of the right arm and leg had come on. Notwithstanding this, she was able to walk without assistance; but she dragged her leg, and the arm hung useless by her side. She now put herself under medical treatment, and her health and strength improved, but the paralytic state remained the same.

On Monday, the 9th of November, she lost her way while endeavouring to walk to Chelsea, and had to come back a considerable distance, but did not seem the worse for the exertion. But on the following Saturday she became much worse, having had a fit, as her friend supposed. She fell to the ground insensible, but was not convulsed, nor did she bite her tongue; she recovered from this quickly, but a similar attack recurred daily. During all this time she faltered very much in her speech, and exhibited a considerable amount of drowsiness. Her bowels had been much confined; on the Tuesday and Wednesday she had no evacuation; but on Thursday, the 18th, she began to pass everything under her in bed. The drowsiness and paralysis increased until her admission on November 20th, and when brought into the hospital she was in a semi-comatose state, would answer questions only when loudly spoken to, complaining of headache, chiefly frontal (from which she had

suffered all along), and was paralysed on the right side of the body, the palsied muscles being extremely rigid. The arm was bent firmly across the chest, and she retained a little power of motion in it; reflex actions were very marked in the lower extremity; the tongue, when protruded, did not verge particularly to either side; the left pupil was dilated; and there was slight external strabismus, and also a slight amount of ptosis of the left lid. In this state she continued, and though various remedies were used, at first mercury and subsequently iodide of potassium, she gradually sunk, and died eighteen days after her admission.

Upon examining the body of this woman, we found the right hemisphere of the brain in a healthy condition. The surface of the left hemisphere was rather flattened, and its convolutions expanded. On slicing, it was found evidently much congested, as shown by the numerous bloody points from cut veins in the white substance, and more especially by the dark colour of the gray matter of the convolutions. The white matter of the anterior lobe of the left hemisphere, and the surface of the corpus callosum, were much softened, as was demonstrated by pouring a gentle stream of water upon it; and the softening extended from the convolutions to the corpus striatum, the anterior and inferior portions of which were completely broken down, leaving, however, many of its fibres intact and free. At the posterior part of the horizontal portion of the corpus striatum, there was a mass which presented the appearance, as far as regards colour and consistence, of a disintegrated blood-clot, consisting of a gelatinous-looking substance, which resembled fibrine in an imperfectly coagulated state, together with some colouring-matter, through which numerous nerve-fibres were found passing, but no blood-globules. The lesion extended backwards to the corpora quadrigemina, and downwards to the inferior layer of the crus cerebri; and thus arriving at the base of the brain, it could be traced to the fissure of Sylvius, where a considerable induration was found. I am inclined to believe that the disease consisted primarily in this induration, and that the lesion gradually spreading upwards from this point finally involved the anterior lobe. The posterior portion of the upper surface of the mesocephale towards its

continuation into the thalamus, and the posterior part of the thalamus itself, were in a very indurated condition.

Now, without attempting to determine whether this extensive brain-lesion was inflammatory or not (which from the want of a minute microscopical examination is impossible), there can be little doubt, that there was quite enough alteration in the induration and the extensive white softening to excite irritation and to cause paralysis. And it was to this *irritative* as well as *paralysing* lesion that the rigid state of the muscles of the paralysed limbs was to be attributed. It is probable, as I have already said, that the starting point of the disease was at the indurated portions. They, no doubt, had much to do with the early attacks of epileptic character, with the faltering speech and the semi-comatose state. To these symptoms were soon added the paralysis and the rigidity of muscles.

Rostan gives an interesting case of this form of hemiplegia, with a rigid state of the paralysed muscles, in which the symptoms depended on an exostosis, projecting from the parietal and the petrous portion of the temporal bones, and compressing the cerebellum, so as to form a deep hollow on its surface, thus causing paralysis with rigidity of the opposite side of the body.

In Lallemand's work, there are the narratives of several cases of this form of hemiplegia with early rigidity, in all of which the brain lesion was of an irritative or of an inflammatory character. I must limit myself to quoting one case. It is the first of his second letter. A man, aged seventy-six, was brought into the Hôtel-Dieu, on the 1st of April. He had been found lying insensible on the floor of his room. The limbs of the left side had lost sensation and motion, but they were semi-flexed, stiff, and contracted, especially when an attempt was made to extend them. He regained his intelligence slightly, but remained in a lethargic, almost comatose state, the palsy being unchanged, until the 6th, when he died at noon. A very thin layer of lymph covered the arachnoid on the right hemisphere. There was extensive softening of the middle and posterior lobes, and the softened matter had the colour and consistence of pus, and in the upper part of the posterior lobe a small deposit of pus was found. Towards the inferior part of the middle lobe, the softened gray substance had a brownish colour from the infiltration

of a small quantity of blood, and in this situation the vessels were dilated and filled with blood.*

The cases which I have detailed to you—and it would not be difficult to multiply them—will, I think, sufficiently illustrate the mode in which the hemiplegia with early rigidity is apt to come on. Let me point out to you, that it is not an inflammatory state of brain only which may excite this rigid palsy, but one of irritation, whether inflammatory or non-inflammatory. By an irritated state, I mean one of exalted polarity of the nervous tissue, or, as the physical philosophers would say, a state of high tension. Inflammation may exercise as depressing an influence as atrophy; in other words, it may as effectually destroy the conducting power of the nerve fibres or the generating power of the vesicular structure, as if the elements of those two kinds of nerve-matter were wasted or ruptured. And, as Lallemand has remarked, an early stage of the inflammatory process may be irritative and paralysing; a later stage paralysing simply.

You may fairly ask, is a paralysing lesion compatible with an irritative one? To this I think an affirmative answer may be readily given. A state of irritation interferes with the due conducting power of the nervous fibres, and with their prompt obedience to the influence of the will. The physical nervous action refuses either wholly or in part to obey the mental influence which usually controls and directs it; the disturbed nutrition of the nervous matter prevents the ready development of those

* In the 'Proceedings of the Pathological Society of London' for 1851-52, there is an account of a very well-observed case, in which red softening appears to have supervened upon white softening. The hemiplegia was on the left side, and the muscles of the arm, at first, seemed to have been flaccid, and to have subsequently become rigid. The case is recorded by Drs. Sibson and Handfield Jones.

Dr. Bennett has put on record a case in which there was twitching of the muscles of the right arm, followed by numbness of the fingers of the right hand, and diminished power of the arm on the subsidence of the twitchings. The right leg was only benumbed. This state was followed some weeks after by an attack of coma, with rigid flexion of the right arm, and great resistance when an effort was made to extend it. The lesion consisted of inflammatory induration surrounded by softening of a portion of the left cerebral hemisphere. —*Clinical Lectures*, No. IV, p. 152.

physical changes in the nerve-cells and nerve-fibres, without which the will cannot be freely obeyed. Moreover, in many cases, the lesion consists at once of ruptured fibre and compressed brain substance.

In further illustration of this form of hemiplegia, let me call your attention to a symptom which often occurs in cases of cerebral disease. It is this, that not only is hemiplegic paralysis present in a more or less complete form, with or without rigidity, but also frequently a convulsive condition, with or without loss of consciousness. Now and then, the patient will be seized with more or less active clonic, and sometimes choreic, convulsions of the paralysed limbs, and occasionally these convulsions will pass into the complete epileptic paroxysm. The convulsive attacks to which I allude, which may be appropriately called *epileptiform*, last sometimes as long as twenty minutes or half an hour, leaving the patient greatly exhausted, and the limb paralysed.

One of the best marked examples of this kind, which I have seen, was in the case of a little child, whom I attended some years ago with Mr. Dunn. In this case these convulsive movements were an early and a prominent symptom. The convulsions were of the clonic kind, and sometimes while the arm was jerked convulsively, the leg was affected with tonic spasm. The left side of the body was affected with the convulsions, and it was found, after death, that a considerable deposit of tubercle had taken place in the pia mater of the convolutions of the right hemisphere, around which an extensive inflammatory softening had occurred.*

Another interesting example of similar convulsions some of you may have witnessed in the hospital, in a man, who had often been the subject of our clinical observation. His name was Beglin; he was first admitted with a remarkable spasmodic rigidity of the right forearm, which was cured by iodide of potassium. He was a man of intemperate habits, and after every bout of drinking he would be liable to attacks of convulsions, sometimes with complete, at other times with partial, insensibility, the convulsions always affecting the right side. For these attacks he frequently applied at the hospital, and was

* See Mr. Dunn's account of this case, in 'Med.-Chir. Trans.,' vol. xxv.

an inmate of it on more than one occasion. With a rare gratitude for the interest which I had taken in his case, he left instructions before his death that I should be invited to examine his head. We found over the left hemisphere of the brain a thickened state of the dura mater, and marks of old inflammation of the other membranes.*

I have seen other similar hemiplegic convulsions in women during pregnancy and after parturition. In one of these cases, a lady whom I saw in October, 1852, with Mr. Muriel and Dr. Young of Kennington, there had been previous attacks of epilepsy. The second case was a lady whom I attended with Mr. Street and Dr. Hetley. This lady was a perfectly healthy person, and had gone through her confinement without the least untoward circumstance. Being one of those persons who get over confinements very readily, she was able to sit up at the end of a week. One day she seemed to have done too much; and that evening she was suddenly taken with a convulsive fit, which was strictly limited to one side of the body, and was accompanied with loss of consciousness. These convulsive attacks were repeated, and at such short intervals that it was evident, if something were not done to check them, she must soon sink exhausted. She had been bled early, and mercury was freely exhibited, but without affecting the severity or the frequency of the attacks. Under the use of chloroform the fits were very much modified and shortened, and I have no doubt her life was prolonged four or five days. On one occasion Dr. Hetley, who watched the case with the most praiseworthy assiduity, allowed the convulsions to go on unchecked, and they then lasted a quarter of an hour; but when the chloroform was inhaled, they generally stopped in from two to four minutes. In neither of these cases, unhappily, could we obtain a post-mortem inspection, as is too often the case in private practice, unwisely and unfairly for medical science.

A similar hemiplegic convulsive state is met with after injuries to the brain. "We find it," says Sir Benjamin Brodie, "occur in cases of punctured and wounded brain, where there

* See the full particulars of this case in Lecture XLIX, and the remarks in hat lecture generally.

is no pressure; and it so happens, when it has fallen under my observation in cases of depression of bone or extravasated blood, and where the exact nature of the injury has been afterwards ascertained, that the pressure has been always found to be complicated with wound or laceration of the substance of the brain."*

Convulsive movements of a less violent kind, more like the movements of chorea, occur in connexion with softening of one hemisphere of the brain. Thus, a man of fifty years of age, who had suffered from gout very much, became subject to fits of wandering; he would talk incoherently and seem lost, but after a little time recover; there were also chorea-like movements of the arm and leg of the right side. There were numerous spots of ossification in his radial arteries. After a free bleeding he became comatose and sank. On examination, the gray and white matter of several convolutions of the left hemisphere were in the state of colourless softening, easily broken up by a stream of water. There were several osseous spots in the arteries of the brain, especially in the middle cerebral.

Not long ago, we had, in the Rose ward, a man named Fleming, aged twenty-nine, in whom these choreic movements of the left arm, with an imperfectly paralysed state of the upper and lower limbs on that side, accompanied a very weakened and deranged intellect. Any one who simply watched him as he lay in bed, his arm moving unceasingly, would have set the case down as one of chorea. To add to the resemblance, the symptoms originated in fright, caused by his house taking fire. Three days after the fire his illness began with these movements; he remained a long time in the hospital, becoming only more and more imbecile and deranged, and he was ultimately sent to St. Luke's.

In cases like these, as doubtless also in chorea, the perverted nutrition of the brain occasions irregular developments of the nervous force, which are tantamount to a state of irritation, and give rise to the convulsive twitchings. In chorea, the alteration is not so profound as to be beyond repair; in the softening of adults, unhappily, the lesion is too often beyond recovery.

* 'Med.-Chir. Trans.,' vol. xiv, p. 352.

There is one point with regard to this rigid condition of the paralysed muscles in this form of hemiplegia, to which I must refer before I conclude. It is this—that the rigidity is always, so far as my experience goes, much greater in the upper than in the lower extremities; indicating that the irritation is propagated from the brain to the spinal cord, and that the upper limb exhibits a greater degree of the muscular affection, because it is nearer the seat of irritation.

I have yet to consider the pathology of those cases of hemiplegia with rigidity of the paralysed muscles, in which that phenomenon comes on gradually, and some time after the paralytic seizure.

LECTURE XLIV.

ON PARALYSIS.

ON HEMIPLEGIA WITH LATE RIGIDITY OF THE PARALYSED MUSCLES.

In my last lecture I described to you the phenomena of hemiplegia with early rigidity of the paralysed muscles.

I showed you that in the first form of this kind of hemiplegia in which the rigidity is slight, and confined to the biceps of the arm, or at most to the biceps and triceps, and in a very trifling degree to the flexor muscles of the fingers, the paralysis is generally dependent on a clot which has lacerated the brain, and which is frequently preceded by white softening. The second form, in which the rigidity is considerable, and sometimes tetanic, is due to a more decidedly irritative lesion of the brain, or of its membranes, which is often inflammatory. The various cases which I cited showed, that although the lesions capable of producing such a hemiplegia differed, they agreed in one point—that they excited and maintained an irritated state of brain.

Now the prognosis in this form of hemiplegia is, on the whole, unfavorable: many of the cases die pretty soon after the

attack, especially when the brain-lesion is complicated with a sanguineous apoplectic effusion. As a general rule, the larger the clot the shorter will be the duration of life. Patients will survive an attack of this kind from a few hours to two or three weeks. *Perfect* recovery is, I suspect, extremely rare; indeed, I doubt if it ever occurs, excepting after inflammatory softening of very limited extent. In a few cases the state of rigidity gives place to that of relaxation, and the limbs remain relaxed and paralysed for the remainder of life—the muscles becoming wasted to the last degree. In one patient of mine (Cochrane, vol. xii), in whom the paralysis and rigidity depended on inflammatory softening, the deltoid muscle and the scapular muscles of the shoulder-joint became so relaxed and wasted, that the head of the humerus dropped away from the glenoid cavity.

Many cases recover a slight amount of power in the paralysed limbs, after the shock of the palsy-stroke has passed off. But this improvement is not progressive, and, after the lapse of time, the muscles waste, the rigidity remains or increases, and the limb is permanently more or less flexed. In short, the paralysed limbs pass into the condition of what I would call *late rigidity*.

This leads me to bring before you the phenomena of hemiplegia with late rigidity of the paralysed muscles—to give you examples of it, and offer some explanation of its pathology. It may follow the hemiplegia with relaxed muscles, as well as that with early rigid muscles.

I have already described a well-marked example of this form of hemiplegia in the case of Shea (Case CLXXVII, p. 731). In this man, who frequently presents himself at the hospital, you have a favorable opportunity of observing the peculiar condition of the paralysed limbs. In the first place, you will perceive that the muscles are wasted; next, that the limb is in flexion, and even in extreme flexion; the arm is strongly adducted to the side, the forearm bent upon the arm, and the fingers bent into the palm of the hand. In extreme cases this state of the fingers is often attended with great inconvenience, from the irritation to the skin of the palm caused by the growth of the nails. Lastly, the muscles are tense like cords.

It is remarkable that in this, as in the palsy with early rigidity, the rigidity is most marked in the upper extremity, which is nearest the seat of the paralysing lesion.

In the history of Shea, you have that of nearly all the examples of this form of hemiplegia in its highest degree. He, you remember, fell in an apoplectic fit, and came out of it with hemiplegia of his right side, with all the muscles relaxed except the biceps. He regained power slightly, but after some time the process of wasting and of contraction showed itself in the muscles, and now they are not only attenuated but stiffened, as you may often see them in the dead body during the continuance of the rigor mortis. The attempt to extend the flexed joints is encountered by a powerful resistance, which can be only partially overcome, and always excites pain. In its lowest degree, the rigidity in this form of hemiplegia is limited to the flexors of the fingers. There are few cases, indeed, of long-standing paralysis which do not exhibit some degree of flexion of the fingers, resembling very much that caused by rigor mortis in the dead body. In the lower extremity the stiffness is most in the hamstring muscles, and in those of the calf, and the tibialis posticus and the flexors of the toes, and the biceps femoris. In its lowest degree it will affect the flexors of the toes or the hamstring muscles, or both.

As the process of contraction shows itself, in general, most in the upper extremity, so also it usually commences there; but now and then it will begin in the lower extremity; not unfrequently it will be met with in the upper extremity only.

The view which I have always taken (and which many of you have often heard me express in passing through the wards) of the manner in which this contraction is produced, is as follows: at the seat of the original lesion, whether it be simply a white softening, or an apoplectic clot, or a red softening, with more or less destruction of the brain-substance, there takes place an attempt at cicatrization, more or less perfect. Attendant on this, there is a gradual shrinking or contraction of the cerebral matter, which, acting on the neighbouring healthy tissue, keeps up a slow and lingering irritation, which is propagated to the muscles and excites in them a corresponding gradual contraction, while at the same time their nutrition be-

comes seriously impaired by the want of proper exercise and the general depressing influence of the lesion.

Case CLXXXII.—Many years ago I watched the case of a young girl (A. M. Dawson), aged twenty, who had been the subject of rheumatic fever and extensive endocarditis affecting the mitral valve. This girl became suddenly hemiplegic on the right side, with loss of consciousness and relaxed muscles. She recovered from the attack, and regained a very slight power over the leg. The muscles of the paralysed limbs wasted very much, and in the course of a little time they began to contract; the fingers were bent into the palm, and the forearm bent upon the arm; the leg, likewise, became rigid. Soon after this she began to have some epileptic fits at short intervals, the convulsions being confined to the right side. These fits were of the most fearful kind, consisting of very sharp clonic spasms, succeeding each other with extraordinary rapidity, and leaving the patient in an exhausted condition. After repeated attacks of this nature she died. On examination there was found a small cyst, containing serous fluid. This cyst was situate in the left hemisphere, just outside the optic thalamus. Its interior was lined by a yellowish matter, outside which, for some distance, the brain-substance was very much indurated. This was, no doubt, the contracted cyst and cicatrix of an apoplectic clot, the gradual shrinking of which operated as an irritant foreign body on the brain. Hæsit lateri lethalis arundo.

I shall give you a case similar to that of Shea, and also, no doubt, the same as to the nature of the lesion.

Case CLXXXIII.—Mr. H—, aged fifty-nine, a healthy man, had been latterly working harder than usual, having unexpectedly become head of a large mercantile firm. On the 9th of June, 1852, he dined out, but as he felt unwell he ate and drank very sparingly; in the middle of the night he got out of bed to pass water, and suddenly became weak on the right side, and giddy; there was no loss of consciousness, and but slight impairment of speech. He was immediately attended by Mr. Robert Brown, of Brixton Hill, who cupped him on the back of the neck and purged him. After this he quickly recovered;

but at two o'clock, p.m., on the 10th, he became completely hemiplegic on the right side, including the face and tongue, and lost entirely the power of speech, retaining that of deglutition, without any loss of consciousness. At six p.m. I saw him, and found him hemiplegic on the right side, with relaxed muscles, except slight resistance of the biceps; there were good reflex actions of the leg; he was quite speechless, and protruded the tongue with deviation to the right; the sphincter ani was partially paralysed.

It was evident that a very grave lesion had occurred in this case, sufficient to inflict so severe a shock on the brain as to destroy the power of speech, and to impair the power of the sphincter ani. Consciousness was retained, although the patient was lethargic. I think there must have been a good deal of white softening of the fibres connecting the convolutions and the corpus striatum; it is probable, too, that there were clots near the surface of the brain, but too small to produce such compression of the brain as would generate loss of consciousness.

On the 13th of June, at noon, just after passing water, this patient had a general convulsion, after which he remained for some hours in a lethargic, soporose condition. From this time he began to recover very slowly; he became more lively, and regained a very slight degree of power in the leg and arm; the face-palsy also diminished. On the 16th of June he was still speechless, but understood all that was said to him, and made an attempt to write with his left hand. On the 4th of September his general health was much improved; he was wheeled out in a chair every day, and remained very much in the open air. The paralysed limbs had become much wasted, and now we observed considerable shortening and stiffening of the biceps. On the 16th of March, 1853, intelligence was good; general health improved; and he made a fair attempt to sign his name. The paralytic limbs were much wasted; both *arm and leg had become semiflexed and rigid; the fingers were bent into the palm, and could not be perfectly extended.*

This case affords a striking instance of the progressive change in the paralysed limbs, proceeding probably *pari passú* with a cicatrizing or contracting process in the brain, and which is, therefore, slow and very gradual in its development.

Let me place in juxtaposition with this case another very interesting one, in which I had the opportunity of watching the phenomena during life, and of ascertaining the condition of the brain after death.

Case CLXXXIV.—A lady, aged sixty, was seized in the night of October 6th, 1844, with hemiplegia of the right side, without muscular rigidity, with coma and stertorous breathing. She was quickly attended by Mr. Dunn, who bled her and gave purgatives. In a few months she recovered completely, regaining the full use of the arm and leg, but retaining the single peculiarity of using one word for another, and of not applying appropriate names to the things she intended to signify; she never afterwards called even her own daughters by their right names.

On the 17th of May, 1847, she had a second attack. She was found in the morning lying insensible on the floor of her bedroom. She was again, and for the rest of her life, hemiplegic on the right side, completely as to motion, but some sensation remained; *there was no muscular rigidity whatever.* She was likewise speechless.

This patient recovered her general health slowly. And as she improved in this respect, the paralytic limbs wasted; the arm was kept in a semiflexed position, the muscles slightly rigid; the fingers were bent inwards upon the palm, *and it was with some difficulty they could be straightened.*

In this state she remained until the 14th of April, 1850, when a third sudden seizure occurred, and for a short time *the paralysed arm and leg* shook violently, and the left side now became paralysed as well. From this attack she never rallied, and died on the fourth day.

The cause of the fatal attack and the left hemiplegia was found to be a recent clot, with softening of the right corpus striatum. And there was likewise abundant evidence of old disease of the left side of the brain, upon which the original hemiplegia of the right side depended. Here were shrinking and wasting of the optic thalamus; this body had shrunk to less than half its natural size, its upper surface being greatly wasted. There was likewise considerable softening (colourless) of the

white substance of the hemisphere, of the corpus callosum, part of the corpus striatum, and of the fornix. In the softened brain-substance there were numerous small vessels in a state of fatty degeneration, and also abundance of compound cells.

In this case the contracted state of the muscles of the paralysed limbs on the right side was no doubt associated with the shrunken condition of the left optic thalamus; and it is not improbable that, had a similar state of the corpus striatum existed, there would have been still more rigidity of the muscles. The softening of the hemisphere sufficiently explained the loss of speech; but I think that a large portion of the softening on this side must have occurred shortly before death, as was indicated by the violent agitation of the paralysed limbs, which occurred at the time of the last seizure.*

In further evidence of the connexion between a cicatrizing process in the brain and this state of late contraction of the muscles, let me refer you to a case recorded by Andral, of which the leading points are these:

Case CLXXXV.—A man, aged seventy-one, suddenly lost his consciousness on the 15th of May, 1820. This loss of consciousness lasted only a few hours, but, on his recovery, he found himself paralysed on the whole of the left side. On the 28th of June he was admitted into La Charité. He was still paralysed on the left side; there was "complete *immobilité*" of the left upper and lower extremities, with very obtuse sensibility of those limbs. About the middle of August, it was observed that the paralysed limbs were not only much wasted, but contracted; the forearm was strongly flexed upon the arm, and the leg upon the thigh. Towards the end of October, the upper extremity, remaining paralysed, ceased to be contracted, but the lower extremity still remained contracted. He died on the 28th of November.

In this case a cavity large enough to hold an apple of moderate size was found. It was situated outside, above and behind the

* I saw this patient several times with Mr. Dunn, who has published an excellent narrative of the case in the 'Lancet' of October 26 and November 2, 1850. From that narrative my account has been condensed.

corpus striatum, midway between the two extremities of the hemisphere. Its walls were lined by a cellular, dense, and resisting membrane, in the substance of which numerous vessels ramified. This membrane adhered closely to the substance of the brain, which surrounded the cavity. The cerebral substance around, for an extent of four or five lines, was softened.

It is evident that a distinct process of cicatrization had been going on in this man's brain. Latterly this had been arrested, and a softening took place, which was, no doubt, the cause of the resumption of the state of relaxation by the extremity.

I find in Romberg's book a highly corroborative case.

Case CLXXXVI.—A woman, aged seventy-three, applied for paralysis of the right arm and leg. The muscles were at first flaccid; subsequently they contracted so forcibly, especially at the hand and forearm, that the fingers could only be opened by using considerable violence; nor did they remain extended, but instantly returned to their former position, like elastic springs. Owing to the long continuance of the disease, depressions had formed in the palm of the hand, which were covered with a mucous membrane, and secreted a thick fluid of an offensive odour. The contraction of the foot was less forcible. Death ensued four years and a half after the supervention of the hemiplegia, which had become associated with dementia; and six months before the fatal issue, paralysis of the left leg occurred, with contraction of the muscles. The left arm retained its mobility to the last. The lesion connected with the paralysis of the right side, where the late rigidity had appeared, consisted of an old apoplectic cyst, of the size of a cherry, near the surface of the posterior lobe of the left hemisphere, surrounded by indurated brain and lined by a membrane, and in the same hemisphere, near to the corpus callosum, a brown spot of indurated medullary substance, extending into the left half of the corpus callosum to the septum lucidum. The left thalamus was flattened and wasted, and half an inch smaller than the right one.*

* Romberg, on 'Diseases of the Nervous System' (Sydenham Soc. ed.), vol. ii, p. 424.

I am far from asserting that the evidence which I have now brought forward is sufficient to *prove* that late rigidity is due to a cicatrizing process in the brain.* Many further observations are needed before this view can be considered finally confirmed. There are no doubt many more cases which might have been adduced, had I been able to ascertain certainly the early condition of the paralytic limbs. Enough has been said to show you that the distinction between *early* and *late* rigidity is well based, and to direct your attention to the different circumstances under which each form occurs. Opportunities do not frequently offer to a hospital physician of watching cases of paralysis of very long standing. Patients afflicted with palsy are admitted in the early period of the attack, and are discharged as soon as it becomes chronic; and then, in too many cases, the further progress of the case is lost sight of. In workhouses and other institutions, where the disabled poor are received, opportunities often occur of completing the histories of chronic paralytic cases, and of determining the precise nature of the lesion. To these institutions many of you may hereafter be attached as medical officers; and I trust you will not neglect to investigate this point. It is only by the well-directed industry of many labourers that we can expect to bring so wild and rugged a field as that of cerebral pathology into a more productive cultivation.†

With reference to the actual condition of the muscles in the paralytic limbs with late rigidity, I think it must be admitted, as I have already intimated, that the rigid state of the muscles is due primarily to an irritated or excited condition of the nerves; and that on the cessation of that irritation, the muscles might resume their relaxed condition, or that a similar result would follow the severance of all connexion between the muscles and the seat of cerebral lesion by section of the nerves. It seems to me, however, that after a long continuance

* Dr. Abercrombie's case, CXXII, appears to have been one of this kind.

† Observations pursued like those of Turck will, no doubt, throw great light on the changes which take place throughout the cerebro-spinal axis in connexion with chronic lesion of the brain. I have not yet seen more than a brief abstract of his book, entitled, 'Uber secundäre Erkrankung einzelner Ruckenmark-stränge.' Vienna. 1851.

of this rigid and shortened state, the muscles would become permanently shortened, and would assume a condition similar to that into which those about ankylosed joints are apt to fall—a condition from which they would recover very slowly, or not at all.

I must now pass on to consider some other forms of hemiplegia; and this will afford abundant material for another lecture.

LECTURE XLV.

ON PERIPHERAL AND ON HYSTERICAL HEMIPLEGIA.

The next form of hemiplegia which I shall notice is that which I have termed "peripheral hemiplegia." This name, which is not a correct designation when the whole of the pathology of the affection is taken into consideration, is, nevertheless, a convenient one, inasmuch as it indicates the most prominent feature of the malady, namely, the mode of access of the paralysis. The palsy is at first of the hemiplegic character, but after a time both sides of the body become involved, and it seems to travel from periphery to centre. At the onset of the disease, the patient complains of a feeling of numbness in the arm or leg affected, although, usually, touch remains perfect for some time, and he can distinguish two points, as such, at very slight intervals from each other. There is probably some altered condition of the nerves of sensation; and, perhaps, of the sentient nerves of the muscles, rather than of those of the skin. After a time the patient finds that he cannot perform small actions with the diseased arm; he cannot button his clothes, nor pick up a pin with the one hand, as well as with the other; then he notices that he cannot write so well as formerly; he has not the same command over his pen which he previously possessed, and his handwriting becomes tremulous.

A failure in the temperature of the extremity next manifests itself; it feels colder than the healthy limb, and sometimes the thermometer indicates that it really is colder. If the weather be cold, the affected limb will suffer more than the sound one.

For some time the patient continues to be able to grasp with one hand as well as with the other; the disorder at this period seeming to consist chiefly in the want of power to adjust the muscles for the finer movements, but not for the coarser actions; after a time more or less inability to accomplish the latter also comes on. After a longer or shorter period, the leg goes through a train of symptoms similar to that which the arm has previously undergone; the patient drags it after him in walking; his movements become stumbling; and he speaks slowly and hesitatingly, but yet his mental faculties do not seem to suffer much. This may go on for many months, or even years, for these cases are exceedingly chronic; the other side of the body, in some instances, begins to be affected almost simultaneously with, or very soon after, the first; but in others it may be a considerable time before it suffers. Whenever it may begin, it passes through a train of symptoms similar to that which I have described. The patient now begins to stoop; he finds he cannot hold himself erect; and in some instances his gait is apt to pass into that which is known as symptomatic of the disease termed "paralysis agitans," all the limbs shaking more or less, the trunk being bent forward, and, at every attempt at walking, there begins an irresistible tendency to assume a running gait. Sometimes the few closing months of life are disturbed by occasional slight attacks of epilepsy, and the memory and other mental powers participate in the general bodily decay.

In some persons this affection seems to limit itself to one leg only, or one arm—more frequently, I think, the latter; and in these cases, it is accompanied by a trembling or shaking movement, always aggravated by emotion of any kind, or by depressing influences, such as overwork, trouble, or anxiety.

In some cases the trembling movement can be controlled by a strong effort of the will; in others, such an exertion increases the trembling, as it does in chorea. In the case of a hard-working lawyer, in very extensive practice, this symptom affect-

ing the arm existed for a period of five years, the patient having been all that time under my observation. It never extended to any other limb, and the patient died of the effects of cancer in the right kidney.

The peculiar affection of the hand which prevents a patient from using his pen, while he can perform any other action perfectly, seems allied to this. In one case of this disease I have seen both hands attacked. The patient had acquired the power of writing in an exquisitely beautiful style with the left hand, when it became affected in the same way as the right hand. In some cases the sentient nerves alone seem affected for a considerable period prior to the motor derangement.

Case CLXXXVII.—I have here the notes of one of these cases, in which the early symptoms affected both sides equally, although one side was ultimately more weakened than the other. The patient was a man of the name of Barber, thirty-eight years of age (vol. xxvi, A); and I may here remark that, so far as I have seen, this malady generally comes on at a more advanced period of life than in this case, the patients being almost always above the age of fifty. This man had always enjoyed good health; he had lived well, and had never had syphilis. The disease commenced six months before admission, with a sensation of tingling and pricking in both hands (what is commonly described as "*pins and needles*"), with a considerable amount of numbness about both elbows, just as if the ulnar nerve had been jarred, or, in the patient's own words, "as if he had struck his funny-bone." With all this, there was a sensation of heat in the forearms; and he soon became unable to button his clothes or pick up any small object with the right hand. Under treatment, the tingling and numbness became diminished, but the loss of power continued, affecting the right side chiefly: this increased to such an extent, that he could not raise the right arm above the shoulder. When he came to the hospital, he could scarcely grasp with the right hand, and that with difficulty; he could only raise the arm to the level of the shoulder, and dragged the right leg considerably in walking. There was some deficiency of power in the left hand also, but not so much as in the right, and he could

raise that arm perfectly; the power of the left leg was not impaired. The morbid sensations which I have described existed on both sides. There were no pains in the head, nor giddiness; but he experienced a certain feeling of insecurity in walking.

He remained in the hospital some time, and was subjected to various plans of treatment, among which were free purging and mercurializing; but without any other benefit than that he could walk more securely than on his admission.

Case CLXXXVIII.—I shall refer you to a second example of this affection, which occurred to me in private practice. The subject of it was a medical man, aged fifty-two, who consulted me early in 1849. He had noticed for some months before, gradually coming on, a sense of numbness in the right arm—a sort of feeling as if it were not in its right state, while the faculty of touch was not impaired. Then he found himself unable to execute the finer movements, his handwriting became very shaky, and he was often compelled to employ an amanuensis. Hurry, excitement, or emotion always increased his difficulty, as regards these actions. After this, being engaged in the practice of midwifery, he found he could not manipulate with his right hand with the same facility as formerly; and this was, of course, a source of great inconvenience to him. All this time he could grasp very well, and with considerable power. Soon afterwards, the right leg became similarly affected, and it was then that I first saw him, now upwards of four years ago. There was no symptom immediately referable to the head; no pain, nor giddiness; memory good. This spring (1853), happening to be in that part of the country in which he lives, I visited him, and found that the disease had spread to the left arm and side, and that a train of symptoms had occurred on this side, precisely similar to that which had previously taken place on the right. His articulation was affected, having become thick and rapid in the mode of utterance. His body was now beginning to be bent forward, and he had found it necessary to retire from practice; but his mental faculties were so far quite unimpaired.

Case CLXXXIX.—A very similar case occurred to me also in

the previous year (1848), in the person of a country gentleman, aged sixty-four. He came to me one day from his club, complaining that he had, for some time, experienced increasing difficulty in buttoning his waistcoat or his trousers, and consequently he used sometimes to leave parts of the latter garment open, in a not very seemly way. He complained of pain and a numb sensation in the right arm, but for a considerable time his sense of touch remained unimpaired, and he was able to grasp with considerable power. After some time the leg became similarly affected; when I last saw him the disease had spread to the opposite side of the body, and he is at present almost bent double, and has for some time been obliged to give up writing. A great variety of remedies was tried both in this and the preceding cases—among them galvanism, without any effect.

The pathology of this affection is exceedingly obscure. I have never had an opportunity of making a post-mortem examination, as the disease generally goes on, for many years, without proving fatal to life. Both the patients, whose cases I have just related, are still living; but the man, who was under treatment in the hospital, we lost sight of soon after his discharge. Nor do I know of any published account of a post-mortem examination of a case of exactly this kind, and I am therefore unable to offer any satisfactory hypothesis as to the pathology of this affection. I am inclined to think that it consists in some degree of atrophy of the nerves of the extremities, with a similar condition of some portion of the brain, either of which may stand to the other as cause to effect. But from the mode of access of the disease, from the first symptoms showing themselves in the extremities, without the occurrence of any head-symptoms, I think we may, for the present at least, conveniently mark this disease *clinically* by designating it *peripheral hemiplegia*.*

* The late Dr. Cheyne describes, under the name of Creeping Palsy, a disease which I believe to be the same as this. He also gives the dissection of one case. I quote the whole of his description.

"First, there is observed numbness in the course of a nerve, often in the sciatic or in the ulnar nerve. This may exist for some months without any other symptom of disease, and, indeed, I believe the disease sometimes goes no

Hysterical Hemiplegia.—Let us now inquire into the nature of the hysterical hemiplegia, or that form of hemiplegia which occurs in hysterical patients, without any apparent lesion in the brain. Hysterical paralysis is by no means of uncommon occurrence, the variety most frequently met with being that which is called "hysterical aphonia," wrongly termed *aphonia*, because the subjects of it are almost always able to speak in a whisper. This may be considered the type of this kind of paralysis. It frequently comes on very suddenly, and goes off just as suddenly as it came. A young lady, for instance, will go to bed quite well, but, on rising the next morning, she is unable to speak in her natural tone of voice. After a time her voice comes back suddenly, and it often returns under the influence of strong emotion. The loss of voice may go on for many weeks; indeed, in the last case of the kind that I have seen, it continued upwards of three months. In some of these cases the patients are in a very weak state of health, but in others there is apparently no great deviation in this respect from the normal condition.

Let me mention to you here an instance of the sudden manner in which the lost voice will return in cases of this kind. The

further; but frequently, after some months, a slight defect—a drag, as it is called—is observable in one of the legs, which renders the patient liable to trip; next an inability to use one of the hands in such ways as require combinations of the muscles of a more complicated nature; for example, the patient cannot guide his hand into his coat-pocket. The pulse will be found slow, the circulation languid, the expression inanimate, together with restlessness. Some defect of mental power is discoverable; the apprehension is tardy, the speech less articulate, the words inappropriate, and the recollection of recent events not distinct. Then the disease proceeds more rapidly, the sphincters begin to fail, slight convulsions occur, the individual becomes hemiplegic, or complete paraplegia takes place, with imbecility of mind, the convulsions become stronger and more frequent, and in one of these the scene closes."

Dr. Cheyne gives one dissection of a case in which the paralysis was on the left side. "The medullary substance of both hemispheres of the cerebrum was melted down into a soft mass of the consistence of thick cream. The cortical part of the brain surrounding this substance was firm, and seemed condensed, so that the softened medullary mass seemed confined in a kind of cyst. The corpus striatum of the right side was softened like the medullary matter of the hemispheres; that of the left side was natural."—*Dublin Hospital Reports*, vol. iv, p. 269.

patient was a young lady who had lost her voice. She came to see me at my house several times; after having thoroughly satisfied myself as to the nature of the affection, I assured her that her voice would come back quite as suddenly as it went away,—some day, perhaps, when she least expected it. One morning she came to see me, as usual, and, having been reminded of what I had so often told her, she left the house; she had hardly walked ten yards from the street door, when she recovered her voice. Her sister wrote to me immediately, to acquaint me with this happy fulfilment of my prediction, and added, that on returning home her relatives would not believe but that I had been electrifying her, or performing some conjuring upon her; and, no doubt, had I shortly before tried some new remedy, or made some mysterious passes before her, one or other of these expedients would have had the credit of the cure. What you have to do, in such cases, is to satisfy yourselves that there is no morbid condition of the laryngeal mucous membrane, whether tubercular or otherwise; and if no seriously disturbed state of the system should arise, you may prognosticate, with certainty, that the voice will in due time come back. The subject of "hysterical aphonia" I have brought under your notice as exemplifying the typical form of *hysterical paralysis*, which, in these particular cases, consists in a weakened state of the nerves and muscles of the larynx. But *hysterical paralysis* sometimes affects one limb only, sometimes both lower extremities, constituting "*hysterical paraplegia;*" and sometimes, though certainly least commonly, the upper and lower extremities on one side, and then it will constitute "*hysterical hemiplegia,*" a condition which, although its existence is denied by some authorities, still is sometimes, though rarely, met with. Indeed, if a patient lose the power over one arm, there is no reason why she may not lose that over the leg of the same side, and then you at once have a hemiplegic case.

Hysterical hemiplegia occurs in the same class of persons, under similar circumstances, as other forms of hysterical paralysis. The period which immediately follows that of puberty, and that which precedes the change of life, seem to me most liable to hysterical affections. They are brought on by exhaust-

ing causes, such as excessive menstruation, leucorrhœa, overwork, anxiety, or excitement, or indeed by any debilitating influences.

In the hysterical hemiplegia, neither the face nor the tongue is affected; the palsy is limited to the upper and lower extremity, and is often not complete; the muscles are generally relaxed, but do not suffer much in their nutrition, as compared with those of the other side; now and then one or both limbs may be affected with spasm of some of the muscles, or may have a tendency to pass into cataleptic rigidity. In walking, when the palsy is pretty complete, the leg is drawn along, as if lifeless, sweeping the ground.

Let me direct your attention to some examples of this affection. One occurred in the hospital some years ago, and I then gave a clinical lecture upon it, to which I must refer you for the details of the case, and my remarks on it. The patient's name was Leigh; she was at the later of the two periods of life which I have mentioned. (*Vide* Lecture XXXIII, p. 618.) There were in this case the peculiar mode of progression as regards the lower limb—the sweeping movement, so to express it—and the absence of face and tongue palsy, which, I think, characterise the hysterical affection. There were, likewise, no reflex actions.

Case CXC.—A second example of this affection was in the case of E. Somers (vol. xii, p. 66), at the early age of twenty-one. This patient was brought into a state of great debility by typhus fever. The catamenia were very defective; she had not been unwell more than twice in the last four years. After an accidental fall, in which she struck her head violently, the paralytic state first showed itself. The fall happened six months before her admission, and the paralysis occurred a month afterwards. The left arm and leg were affected, from which she recovered nearly completely, when another fall aggravated the evil, and she came into the hospital.

The peculiar sweeping movement in the leg was less marked in this case than usual, because there was a spasmodic state of the muscles which rotate the thigh inwards, and in consequence the patient kept her foot in an almost constant state of inversion. She nevertheless dragged it along as if it were lifeless,

without raising it from the ground, or resting upon it in the least; nor was she able to walk without the aid of another.

The hysterical nature of this case was indicated by the frequent occurrence of paroxysms of hysteria, in which the patient would pass quickly into a state of nearly complete coma. In these attacks the paralysed limbs would be affected with spasms. It was certain that they were not epileptic, for they were not attended with the complete loss of consciousness which is characteristic of epilepsy. There was no face or tongue palsy; but while she was in the hospital the right side was, for a day or two, affected like the left.

This patient was subjected to a tonic treatment. She was made to walk at regular periods daily, with the help of another, along the wards and corridors, and regained very much the power of the leg, the arm having very soon recovered itself. The hysteric paroxysms ceased, and she left the hospital in three weeks improved, having refused to submit to the application of galvanism to the leg.

Case CXCI.—Let me quote another example of hysteric palsy, to illustrate how the hemiplegic state must be regarded as a simultaneous loss of power in the upper and lower limbs on one side, rather than as a crossed influence acting from the brain. This case affords an instance of the palsy affecting one leg. The patient, Sarah Best, aged twenty-seven, was married between three and four years before her admission (August, 1843). Since her marriage the catamenia had become very irregular and painful, and her health, previously good, had suffered very much. She became subject to flying pains in various parts of her body, and lost strength and flesh. She suffered, likewise, from frequent paroxysms of hysteria, and passed large quantities of pale, limpid urine. A month before admission she began to complain of pain across the upper part of her abdomen, which the least pressure greatly increased. It was treated by leeches, purgatives, and fomentations, and subsided. A week before she entered the hospital she experienced a dull, heavy pain in the left leg, which impeded her walking. Next day the pain increased, and with it came a sense of numbness in the leg; the power of motion decreased very rapidly, and she

was unable to use the leg in walking, dragging it along as if it were dead, being unable to move it in any other way. The catamenia had ceased for six months. There was no indication of brain or spinal disease, nor was there any palsy of any other part of the body. The treatment was directed to the restoration of the catamenia, and she was made to exercise the weakened limb. In a week she was able to walk about the ward, and was well enough to return home in a little more than a fortnight.

Another case similar to the preceding was that of E. Goldsworthy.

Case CXCII.—This young woman was aged twenty-eight, admitted March 6th, 1850. She had lately come off a journey from India, where she had had the cholera; she was a person of distinctly hysterical temperament, and suffered much from leucorrhœa. For two years she had suffered from pain in the left hip, and during all that time she was unable to use the left leg as freely as the right. Shortly before her admission the left leg became weak, and she had occasional numbness and twitchings in it; the leg, on her admission, was so weak, that she could not walk without help. In progression the leg was drawn lifelessly after her, sweeping the floor; sensation was not impaired. The arm experienced the same kind of numbness and weakness as the leg, but to a much less degree. There was no face palsy.

This woman remained in the hospital upwards of three months, under a treatment by tonics, shower-baths, and galvanism. She left it much improved, but not quite well.

As it is important to establish the fact of the occurrence of such cases as this, let me add another example.

Case CXCIII.—M. Holdup, aged thirty-five. This woman has been a cook. Two of her sisters are nervous, the elder being affected similarly to herself; admitted March 19th, 1850. Three years before her admission she had a similar affection, and was cured by a change of air.

Three weeks before she came in, on returning home one evening, she suddenly felt great feebleness in walking, but con-

trived to get home, and had to be carried upstairs; she was purged, leeched freely, and blistered behind the ears, but without benefit.

On admission, the left arm and left leg were found very weak, the arm less so than the leg. She could move the arm, but with diminished power and some tremulousness of motion; the grasping power was also enfeebled. The leg was moved with feebleness and difficulty, and she swept it after her in walking; her gait was very unsteady; the muscles of both limbs flaccid. There was in this woman a decidedly hysterical constitution, and the catamenia were scanty and irregular.

This patient benefited very much, and quickly, by tonic treatment, shower-baths, &c., and at the end of ten days her power of walking was much improved, and she dragged the left leg much less. She was obliged to leave the hospital at this time.

Case CXCIV.—Early in the present year (1853) I was consulted in the case of a lady about thirty-five years of age, who had well-marked paralysis of the hysterical kind. In this case there was excessive leucorrhœa and amenorrhœa, with occasional attacks of hysteria. In September, 1851, she caught a severe cold. Some days after this, on getting out of bed, she fell forwards on the floor, having lost the use of both her legs. She recovered the use of the left leg, but the right continued paralysed up to the time I saw her. At first there was a total inability to stand; she gradually recovered from this, and was able to use the leg in walking, but with the peculiar sweeping movement. It was a curious feature of this case, that at one time the *left* arm was paralysed, and the power of the hand was so affected, that she could not hold small articles, such as a pin.

Under treatment by Mr. Morley, of Barton-on-Humber, the uterine function was restored and the leucorrhœa reduced; and although a long time elapsed, she had regained all power but that of the leg, which still, in May, 1853, remained paralysed. Galvanism had been only partially used, and I recommended its continuance in a more effective form. This lady, I am informed, has been progressing favorably, under a course of muscular exercises, directed by Mr. Amesbury.

I have thought it important to bring before you this form of

one-sided or hemiplegic paralysis, because it may so far simulate that from cerebral lesion, as to lead to serious mistakes in practice. A very serious mistake would be to pronounce the paralysis from brain lesion to be hysterical; and this is the more possible, as a state of hysteria may co-exist with brain lesion, which may give to the paralysed limbs much of the hysterical apparent lifelessness.

The two following cases of hemiplegia are examples of its occurrence in young women at an age when hysteria is most developed, and most apt to simulate organic disease. One recovered, the other died. Both resembled the hysterical affection in many points, and for this reason I trouble you with the details of them.

Case CXCV.—Caroline Willis, aged twenty-five (vol. xviii, p. 25), employed in bookbinding; always a resident of London; unmarried. The following is the abstract of her case from the case-book; date, June 2d, 1846:—For the last three or four months she has had general weakness, headache, and drowsiness, for which, one month ago, she was bled to one pint. The weakness increased, and two weeks afterwards she had an hysterical fit during the night. She jumped out of bed, threw her arms about, and laughed very much. This lasted about half an hour, when she became perfectly rational. After this, her mother noticed some impediment in her speech, which increased up to May 29th, when she quite lost her speech. Ten days before admission her right arm and leg became quite paralysed, with relaxation of the muscles, and without loss of consciousness. The arm was completely paralysed; the leg not so much so; she could use it in walking. The right side of the face was paralysed, and the tongue deviated to the right; speech much affected, and deglutition impaired; manner childish. It was ascertained that she was affected with gonorrhœa at the time of her admission. She complained of pain in the head, chiefly in the forehead. The right pupil was larger than the left.

In consequence of her great weakness, when admitted, she was put on quinine, after a slight purgation. In seven days she had improved considerably. She could walk better and swallow more easily. As it was feared there might have been some

syphilitic taint, she was ordered calomel and opium. Under this treatment she continued to improve; she soon gained the complete use of the leg; that of the arm came much more slowly. This mercurial treatment was continued for a week only, and she then took ammonia and bitter infusion.

She remained in the hospital till the end of July, and was then discharged very much better in all respects.

In this case there was evidently either cerebral or meningeal lesion, which it is not improbable may have had its origin in syphilis.

Case CXCVI.—The second case is of recent occurrence, and excited great interest from the early age of the patient, and from the many points of resemblance to the hysterical affection which we observed in it. Some of you will recollect how closely we watched the case, hoping to find some sign which would unequivocally indicate its real nature, and especially as to the more favorable view of the case. The patient, Mary Ann Hopkins (vol. xxxix, p. 130), was nineteen years of age; she came into the hospital the 28th January, with incomplete paralysis of the right arm and leg, also of the right side of the face —the muscles being flaccid. The tongue did not exhibit any signs of paralysis. The pupils were equal. Her mode of progression resembled that which I have described in the hysterical palsy, as she seemed to sweep the foot along the floor as she walked. The power of the arm and hand was not so much weakened as to prevent her doing many useful things. Thus, on the 29th of January it was reported: "She can dress herself, and fasten her gown without any difficulty, and she was observed yesterday arranging her back hair, but she complains that she cannot do any neat work, her fingers feel numbed, and she can scarcely feel the needle which she holds. She complains of numbness and weakness of the right side, and a sense of weight in the arm. The grasping power is much impaired, and the sense of touch is less acute than on the left side; she has no headache, and cannot assign any cause for her illness; no evidence of heart disease; her appetite is very good, almost ravenous. Bowels regular."

Now the history of this girl was as follows: She was of

healthy parentage; was born and lived all her life in a village in Kent, not far from London. Her general health had never been very good. Three years before her admission she had had typhus fever. She was naturally of a nervous disposition, subject to great variation in her flow of spirits, and suffered from frequent attacks of giddiness. She had menstruated regularly for the last three years, at intervals of three weeks, and lately the discharge had been very profuse, continuing for about four or five days. There was no leucorrhœa.

Two months before her admission she suddenly felt her right foot, while walking, become numb and weak, and she began to halt a little on that side. The palsy and numbness gradually extended up the whole of the leg and thigh. The right arm now became similarly affected, and felt cold and heavy; the features became at the same time slightly distorted, and the speech a little affected.

You will remember that in discussing the point, as we went round the wards, as to whether the hemiplegia was, or was not, of the hysterical kind, I stated that the existence of facial paralysis went very far against the diagnosis that it was of that character. For a time I was much tempted to take the latter, more hopeful, view of the case, and to this I was led by the resemblance of the mode of progression to that which I had already previously noticed in hysterical cases; and also by the early age of the patient, a period when so extensive hemiplegia does not often exist, and by her catamenia being irregular; also by her having latterly become subject to hysterical fits of a very severe kind, and her sisters having been likewise subject to similar attacks. Another feature noticed in this case, and also in hysterical palsy, was the inability to excite reflex actions, but no reliance could be placed on this as a diagnostic sign. After a time, symptoms occurred which rather militated against this diagnosis; the patient became subject to attacks of violent sickness, which would last for two or three days, and were so severe that, during this time, everything was rejected by the stomach. I may remark here, that nothing seemed to relieve these attacks of sickness so much as free purgation by large doses of calomel. At the same time the hysterical paroxysms increased in frequency and severity, the

attacks were so severe as to border on the epileptic, but we tested her sensibility on several occasions, and found that she always evinced sufficient consciousness to show that they were not of the latter kind. I will read to you the description of one of these attacks, by Mr. Plowman, my clinical clerk. It occurred on the 8th of February. "At the visit she was found in a very severe and continuous fit, in which she had been, the nurse said, for upwards of an hour; she had passed her water under her, and was rolling about, and appeared suffering violent contortions of the left side of her body; but she was evidently conscious, as when the bed-clothes were turned down, she repeatedly endeavoured to replace them, though with uncertain jactatory movements; and when her legs were pinched, her countenance gave marked indication of feeling, which was equally expressive, on whatever side she was tried."

After she had been a fortnight in the hospital, the paralysis increased, and at length became complete, the relaxed condition still remaining. At length she died in one of the hysterical paroxysms, eight months after the attack of paralysis. On examination of the brain after death, the left hemisphere was found to be the seat of considerable lesion, which appeared to be due to the deposition of tubercular matter about midway between the convolutions on the outside and the corpus striatum and optic thalamus within. There was a large amount of softening of the brain-substance all around the deposit, so that the white matter seemed diffluent, and fell away, leaving a large cavity, just outside the lateral ventricle. The disease, no doubt, consisted in the gradual deposit of this scrofulous matter. So long as this was small, and the brain-substance around it healthy, the palsy was slight; but as soon as the cerebral fibres, especially those going to the corpora striata, became softened, and altered in their nutrition (under the influence of the pressure of the scrofulous matter), the paralysis became complete.

In this case we had an example of the hysterical diathesis highly developed, and complicating the effects of cerebral lesion. The difficulty of the diagnosis was at one time much enhanced by the exquisite character of the hysterical phenomena, and to a degree which cannot be fully appreciated by

those who have only heard or read the details of the case, and did not see the patient. The result confirmed the value of face-palsy, as a distinguishing symptom of the paralysis of brain-lesion from that of hysteria.

The diagnosis of *hysterical paralysis*, whether it be of the hemiplegic or paraplegic form, or whether one limb only be affected, depends on these points:

1st. The hysterical constitution of the patient herself and of her family; and there are certain signs which, as you know, are held to be indicative of the *hysterical diathesis*, such as a lax condition of the tissues generally, a peculiar fulness of the upper lip, drooping of the upper eyelids, &c.

2d. The absence of signs of lesions of the nervous centres.

3d. The characters of the paralysis itself; the absence of palsy of the face and tongue; the peculiar movement of the leg in progression; the fact of the paralysis not being complete, the muscles not being so much wasted, and the fact of the patient being sometimes able, under the influence of strong emotion, to use the paralysed limb as well as the sound one, or nearly so. But you will not fail to recollect that, even in decided lesion, the paralysed arm is sometimes moved in yawning or sighing, or under strong emotion.

In concluding this already too long lecture, I shall refer briefly to a kind of hemiplegia of which I have seen but little, but which is sufficiently marked to demand special notice. It occurs in men of hypochondriacal habits, and in women too. In its mildest form, it may be distinguished as "emotional paralysis," or when it affects one side of the body, "emotional hemiplegia." It most commonly consists in a simple loss of speech, occurring under some strong excitement, the power of speaking returning usually in a few days, and, indeed, generally very rapidly after the patient has regained the ability to pronounce one or two words, such as "yes" and "no."

The following case affords a good example of this affection in both forms.

Case CXCVII.—The patient was a man between fifty and sixty years of age, of irritable temper and hypochondriacal

habit. A question, respecting some very trifling matter, happening to arise one evening in his family-party, some one present held out too strongly against his view, and this led to a vehement contradiction on his part, which was met by a counter-statement and a rejoinder, and thus he became excited to such a degree, that his power of speech completely abandoned him.

Every one knows how mental emotion and excitement tend to choke the power of utterance; you will remember how well Virgil makes Æneas express the combined influence of grief and terror in the line—

"Obstupui: steteruntque comæ; et *vox faucibus hæsit.*"

Men of irritable and excitable temper, under the influence of strong passion, are apt to stammer and stutter, and find it difficult to give expression to what they mean. It is just this that occurs in the cases to which I am referring, only in a much greater degree. But the fact, that recovery takes place speedily, indicates that no considerable amount of lesion can have occurred. Whatever, indeed, that may have been, it must be looked at as a lesion, caused by powerful mental influence, and partaking very much of the nature of *shock* affecting some part of the brain.

This view of the case was corroborated by other features of it. The patient had full use of his muscles; he had full power over his hands and feet; he could sign a cheque, and his mental faculties seemed unaffected; only he could not speak, and whenever he tried to do so, the attempt would end in a fit of crying. He continued in this speechless state for about a week, when he recovered; and when once he began, the power of speech returned fully in a very short time. Two years after this occurrence, the same gentleman got into a similar argument, and difference of opinion, upon a matter equally trivial, and became again strongly excited; but this time, instead of becoming speechless, he became hemiplegic on the left side, without mental affection, but with decided palsy of the left side of the face. The paralysis was not complete, for he could move the fingers and leg very slightly. After a little time, without any other treatment than that of removing as far as possible all exciting causes, he recovered to a great extent the

power over the arm and leg; but, although the principal recovery took place about six weeks after the attack, he is now, four months after the occurrence of the hemiplegia, by no means quite well. To what extent lesion has taken place in the brain, in this case, I cannot take upon myself to say, having never had an opportunity of examining, after death, the brain of a person who had suffered from this affection; but I am disposed to think that, if there be any lesion, it must be slight and, probably, allied, in its nature, to that which occurs in those transient attacks of hemiplegia which occur after epileptic seizures. Of these I shall speak in my next lecture.

LECTURE XLVI.

ON PARALYSIS.

EPILEPTIC HEMIPLEGIA.

There are yet other forms of hemiplegia, which clinical observation will teach you to recognise, and which, for the sake of prognosis and treatment, it is highly important you should clearly distinguish from those which I have described in the preceding lectures.

The most common of these is that which follows, and is caused by the epileptic paroxysm, and which, to mark its relation to the fit, I would designate "*Epileptic Hemiplegia.*"

The history of the more simple cases of this kind is just this: A patient has a fit, distinctly of the epileptic kind; he comes out of it paralysed in one half of the body; generally that side is paralysed which has been more convulsed than the other, or which has been alone convulsed; but the paralysis may occur where both sides have been convulsed equally. The paralytic state remains for a longer or shorter time, varying perhaps from a few minutes or a few hours to three or four days, or even much

longer. It then goes off, or improves, until the next epileptic fit, when a train of phenomena, precisely similar, recurs with like result.

Let me give you some illustrations of this epileptic hemiplegia.

Case CXCVIII.—A good example of this occurred in Sutherland ward in February last (1853). The following are the chief points of the case: Jonathan Woolley, aged ten (vol. xxxviii, p. 188), had convulsions when teething; since that time he continued healthy, till the end of 1852, four months before admission. He then had his first fit, which came on without apparent cause: he seemed equally convulsed on both sides of his body. In all the subsequent fits the convulsion has been confined to the right side, and after each, the patient was distinctly paralysed on that side, with relaxed muscles. The paralysis was of motion only, and was not complete, a slight amount of power remaining. This boy's fits were of short duration, not lasting above four or five minutes, and the remaining coma was also very short. The intervals between the fits varied very much; sometimes he would have several at once, as many as eight in a day, and this was more likely to occur after a long interval of freedom. The boy remained three weeks under treatment by good food, cold water splashing, and occasional aperients, and he continued free from fits during the last fortnight of his stay in the hospital.

CXCIX.—Thomas Orton, aged thirty-four (vol. xxx, p. 80). This man had been healthy up to fifteen years before his admission in March, 1850. In the year 1835 he was thrown out of a chair, and pitched violently on his head. He was stunned, and his left arm contused, but recovered perfectly. A year after this he was suddenly seized with a sensation as of an electric shock traversing the whole of his left side, and he fell down insensible, foamed at the mouth, bit his tongue, and was convulsed. From this he speedily recovered, but with *his left arm and leg paralysed;* the face was paralysed, and the tongue greatly lacerated, on the left side. These fits recurred daily for about ten minutes and were always ushered in by twitchings in the fingers of the

left hand. The paralysis had never perfectly recovered since the first attack; but after each fit it became worse for a few days, and then recovered to the point at which it existed before the fit. There were also in this case minor fits, the patient experiencing a sudden start and falling down for an instant, with a vacant look, but not insensible. The weakness in the left side is not at all increased by these smaller attacks. This patient has been treated by nitrate of silver, and his skin was discoloured in consequence. He remained in the hospital two months without any material change in his symptoms, excepting that the intervals between the attacks seemed to be prolonged. He was treated with the tincture of sumbul, and afterwards with valerianate of zinc.

Case CC.—H. Pitt, aged twenty-six, a labourer (vol. xxxiv, p. 84), had fits at irregular intervals for eight years, from which he recovered very quickly, so as to be enabled to resume his work on the same day. In each fit he became paralysed on the right side with relaxed muscles. The face was paralysed on that side, and his speech was slightly impaired. The paralysis was never complete. It did not improve for eighteen months previous to his admission, December 9th, 1851, but became worse after each fit, and recovered to a certain point before the next. It was ascertained that the fits were the effect of intemperate habits; and evidence was obtained of a syphilitic taint. He was subjected to a mild course of blue pill for twenty-eight days, and left the hospital very much recovered.

Case CCI.—Mary A. Godfrey, aged twenty-nine (vol. xxxi, B, p. 147). No positive evidence of syphilitic taint could be obtained, although upon that point I had strong suspicions. Her first epileptic fit was in January, 1850; the second, early in March. She came out of this fit with paralysis of the left arm and leg and side of the face. This attack of paralysis lasted only half an hour. In three weeks afterwards she had another fit with like results. In the night of the 16th of May she had a fit in her sleep, and awoke up in the morning with the left side paralysed, including the face and tongue. The paralysed parts had their sentient power very much diminished,

and there was ptosis of the left upper lid. She was admitted into the hospital on the 21st of May. Under the use of quinine, iron, and iodide of potassium, and occasional purgatives, she quickly regained the power of the paralysed limbs, both as to sensation and motion, and left the hospital quite well in seven weeks, notwithstanding an attack of pericarditis, which came on after she had been three weeks in the house.*

The next case affords an instance of paralysis of one arm, consequent upon the epileptic fit.

Case CCII.—Ellen Biddlecomb, aged twenty-four (vol. xxx, p. 130), admitted April 3d, 1850. Catamenia disappeared three years ago, and soon after she had her first fit. She suddenly fell, and was convulsed and insensible for half an hour. She came out of the fit paralysed as to her left arm, but recovered it the next day. The fits, since this first one, have recurred generally twice a week, although there has been as long an intermission as three weeks. After every fit she was found paralysed in the left arm, and she suffered excessive pain in it. Both the paralysis and the pain always subsided in one or two days.

On the 6th of April, three days after her admission, she had a fit, and the following note was made: "Last night she had a fit, which appears to have been less severe than any previous one, having lasted only for ten minutes. This morning she can scarcely move her left arm, which is likewise very painful." Similar reports were made on May 11th and June 11th. The arm recovered in two days, and the pain was relieved by chloroform applied locally. This girl was of a decided strumous diathesis, and had enlarged cervical glands; she derived benefit from the treatment in the hospital. She had shower-baths, cod-liver oil, and sumbul. The fits were suspended for a whole month.

Case CCIII.—Some of you will remember the case of an old soldier, Arnold Young, aged fifty-eight (vol. xxxv, p. 83), who was admitted on the 29th of October, 1851. This man's

* *Vide* p. 722.

first fit was in February, 1851; of this we have only a very imperfect account. Of the subsequent fits, each was ushered in by a numbness and twitching of the right great toe, extending gradually up the leg and side to the arm, which was also affected. Both leg and arm would then be seized with convulsive twitchings. Very often the attack would end there; and he stated that, sometimes, by having the arm and leg diligently rubbed, or, less effectually, by tying a tight ligature around the arm and thigh, the fit of general convulsion would be prevented from coming on; and, in fact, this happened several times while he was in the hospital; when he felt the warning he would throw himself on the bed, and rub the arm and leg, and employ others to do so, or apply the ligatures, and frequently with success. If, however, these preventive means failed, the fit would come on, with convulsions and coma, the former affecting chiefly the right side, and leaving the right arm and leg paralysed, and also the right side of the face and tongue. He had not had a fit for between four and five weeks prior to his admission into the hospital; the right arm and leg, which were rendered utterly powerless by the last fit, had, on his admission, regained a considerable amount of power. He could raise his hand to his head, but the power of grasping was very feeble, and he could not use the hand to cut his meat; he walked with a limp. The recovery of the leg was more rapid and complete than of the arm.

From this man's admission to the 30th of November he had three times a threatening of a fit, which was stopped each time by friction. On the 30th he had a complete attack, notwithstanding the application of ligatures. He was very much convulsed; and, on recovering, the right side, which, in the absence of the attacks, had regained power very much, was greatly weakened. From this time till the 12th of January he had two threatenings, and one fit.

The treatment consists in free counter-irritation of the scalp, on the left side, by tartar emetic ointment, and the use of iodide of potassium. Further than that he gained power in the paralysed limbs, he did not seem to derive marked benefit.

Case CCIV.—A very good example of this form of hemiplegia

is afforded by the following case:—Sarah Bone, aged sixty-three, admitted September 17th, 1853. I shall quote the notes of the case made by Mr. Liveing:—"She is a charwoman, of tolerably healthy appearance for her age, thin, and rather pale. She has always enjoyed good health, excepting that she had been subject to severe headaches, as she says, 'seldom getting up in the morning, or going to bed, without one, since her childhood,' until she ceased menstruating, since which time they have disappeared. The headaches used to be always most severe on the right side of the head.

"Last November (1852), after having been heavy and sleepy for some days, she had a fit; her daughter states that she foamed at the mouth slightly, and that her left hand was convulsively clenched. From this fit she recovered with paralysis of the left side of her body; but in a short time she was able to walk about again, only experiencing a numbness and sensation of coldness in the left side.

"In July last, while getting out of bed one morning, she had another fit, in which she became quite insensible, foamed at the mouth, and slightly clenched her hands. She remained insensible for three or four hours; and recovered with decided paralysis of the left side, inability to protrude her tongue straight, and her face drawn to the right side. On this occasion it was three or four weeks before she recovered sufficient power to enable her to walk about.

"Last Tuesday (September 14th), as she was engaged in cleaning a room, she fell down suddenly, the left leg and foot seeming to give way under her; she was carried up to bed, and there she became insensible for an hour; and when she came to herself again she found she had almost completely lost the use of her left arm and leg, and that sensation was much impaired.

"Since Tuesday she has been gradually recovering the power and sensation of the left side. It is still, however, very numb, and much colder to the touch than the opposite side. There is no rigidity of the muscles, and she can distinguish two points, at half an inch apart, on the back of her left hand."

This patient remained in the hospital till the 15th of November, and left it much recovered in power, but still feeble on the left side.

In this case, no one can doubt the epileptic nature of the first and second attacks. The third seems less of the epileptic character; it rather indicates a solution of continuity of brain-fibres, and possibly a slight effusion of blood; I say a slight effusion, because, under a large one, she would scarcely have recovered her consciousness in so short a time as one hour.

Case CCV.—I have seen a few times lately a gentleman, a patient of my friend Mr. Lynch, of Sudbury, who has distinct epileptic attacks, immediately following the cessation of frequent periodical attacks of gout, to which he had been for many years subject. The first epileptic attack was in April, 1852, and, since that time, he has had them at intervals of from ten or fourteen days to six weeks. Usually before the fit he has a slight warning of its approach, in the shape of a little giddiness in the head and sinking in the stomach; he then falls unconscious, the muscles of the mouth, left arm and leg, working spasmodically. The fit lasts two or three minutes, sometimes a little longer, and it leaves him with temporary paralysis of the left side, particularly of the leg, which disappears in from fifteen to twenty minutes. The next day he appears quite well. In this case there was a small quantity of sugar in the urine.

I am informed that latterly this patient has had the fits less severely, and that the paralysis is less marked, and not so extensive as it used to be.

Case CCVI.—I am tempted to add another case, which affords a good example of an important class, namely, syphilitic epilepsy with hemiplegia, occurring in an exhausted state of the system. A gentleman, aged twenty-five, had had chancres a few months before, followed by a slight sore throat and syphilitic lepra. These symptoms had disappeared. For some weeks he had given himself up to the hard labour of a gay life in London. On Saturday, September 24th, 1853, he went to bed in his usual health; in the course of the night he evidently had a fit, as indicated by the disturbed bedclothes, and by his exhausted state next morning, and by hemiplegia of the left side. This recovered slightly without any medical treatment; I saw him on the Wednesday, and found that he had still imperfect

hemiplegia of the left side with relaxed muscles, deviation of the tongue to the left, face-palsy on the left, and dilated pupils. There was some periosteal tenderness of the head, and the patient was evidently much exhausted, partly by the attack and partly by previous dissipation. He was put on a treatment to regulate his digestive organs; and he took the nitro-muriatic acid, and afterwards iron and quinine. Subsequently the iodide of potassium and citrate of iron were prescribed. This treatment was pursued in the country, under the superintendence of Mr. Bennett, of Gateshead.

At the end of January, 1854, this gentleman presented himself to me perfectly recovered, and I have seen him also in the course of the year 1855 in excellent health. The paralysis had been of only a few days' duration.

Through the kindness of my friend and former pupil, Mr. John K. Spender, of Bath, I am enabled to add a case in which very complete anæsthesia followed the epileptic attack, the voluntary power being affected only to a slight extent. I shall give the case in Mr. Spender's words.

Case CCVII.—"Thomas Nowell, aged forty-two, an apparently healthy, well-built man, lost a leg from an accident when a boy, and suffered from repeated attacks of acute disease (the nature of which I could not clearly make out) in the earlier part of his life.

"Last winter, he had what he calls a fit, for which he was bled largely. He quite recovered from this, and had nothing to complain of till the day on which I first saw him, June 6th, 1854. He had been tolerably well that day, with the exception of a slight headache, when, at about nine o'clock at night, without previous warning, he was seized with trembling and almost immediate insensibility, which lasted nearly an hour. On awaking from this state, in which he was reported by the attendants to have been convulsed, and to have foamed at the mouth, he was found to be labouring under almost complete anæsthesia of the whole of the left side of the body. Volition was very little injured. When I saw him, which was almost immediately after recovering from the fit, I noticed that the arm appeared swollen and that the superficial veins were exceedingly prominent; and he was rubbing the limb very violently with the right hand. On

asking him why he did this, he replied, "To get away the deadness." He could move the left arm perfectly well, and could grasp the objects with the hand almost equally as well as with the other. The speech was decidedly affected; but he had perfectly regained the possession of his senses, and there was no mental hallucination, nor anything approaching to coma. Reflex action could be induced in the lower extremity. The bowels had not been moved for two or three days; the face was hot and flushed, partly, perhaps, from his violent exertions. On persuading him to remain still for a few minutes, I counted the pulse, which numbered 100. The urine was normal in quantity, but rather high-coloured. I could obtain no evidence of either rheumatism or syphilis; *possibly* some gout, but this was uncertain. The nature of his employments precluded the hypothesis of mineral poisoning.

"I moved the bowels with a brisk cathartic, applied a small blister to the back of the neck, and in forty-eight hours he was apparently well.

"He had another attack in October, very similar to the former. Since that time he has not been to the dispensary at all, nor have my colleagues seen anything of him."

I have thus given you examples sufficient to show that, in a clinical point of view, the distinction of a form of hemiplegia in connexion with the epileptic paroxysm is well founded. The paralysis, you observe, in all these cases, follows the epileptic paroxysm. In two instances I have known the paralysis precede the fit; and this may have been the case in the last attack of the woman Bone (Case CCIV), if that were really epileptic in its nature.

I cannot doubt that many of the cases of sudden loss of consciousness, followed by hemiplegia, which are popularly and even medically described as "apoplectic fits," are of the epileptic nature. It is the success of cases of this kind under bleeding, and purging, and mercury, that has emboldened so many practitioners to adopt this treatment in all paralytic cases. I think the examples quoted show that the natural tendency is for the paralysis to recover without such interference.

What is the paralysing cause in such cases as these, especially when the paralysis is so transient as to pass off in a few minutes

or even hours? Those who are so ready to explain all brain symptoms by a reference to congestion will, of course, find no difficulty in discovering a local congestion, which occurs at the time of the fit, and remains for a longer or shorter time after it, and this congestion compresses some part of the brain, and causes paralysis. But I cannot too strongly impress upon you, that you must not rest satisfied with such a clumsy explanation as this. It is unsatisfactory in a scientific, and dangerous in a practical, point of view. The vessels of a part, all-important as they are to its nutritive and other vital actions, are nevertheless only secondary elements in the construction of the organ; and unless in themselves diseased, they can play only a secondary part in the production of organic or functional derangement. Congestion of blood-vessels, or hyperæmia of a part, must be an *effect* either of some disordered state of the intrinsic elements of the tissue, or of the blood, or of the forces by which the blood circulates. And a sound pathology ought to receive no other explanation of morbid phenomena, or of congestion, if it exist, but that which traces the real state of these.

If any one ascribe these hemiplegic phenomena, consequent on epilepsy, to congestion, you must then ask, first, what causes the congestion on which the fit depends. A man is apparently in good health at one moment, and the next he falls in an epileptic fit. This, you say, is due to congestion. What causes that congestion? why does it come on so instantaneously? But this not all. A has a fit from which he recovers speedily and apparently unscathed; B has a fit of the same kind, or even lighter, and recovers from it paralysed on one side. What is the difference between the congestion of A (supposing it to exist) and that of B? Why is the latter determined to one side more than to the other?

To enter fully into this discussion would prolong this lecture to an inconvenient length, nor would it be quite consistent with its practical character. I must be content to have given you the clue to the argument on this point, and to state briefly my own view of the case.

The phenomena of the epileptic fit depend upon a disturbed state of the nervous force in certain parts of the brain—a morbidly disturbed polarity. This may take place, under the

influence of some poison which may have an affinity for those parts, such as prussic acid, in the same way as strychnine induces an exalted polar state of the spinal cord, or from some disturbance of nutrition, which may be strictly local or sympathetic. This undue exaltation of the polar force induces, subsequently, a state of depression or exhaustion, not only in the parts primarily affected, but in parts of the brain connected with them, according to the degree of the primitive disturbance; just as undue muscular action exhausts the muscular force. The disturbing cause may operate primarily upon parts of the brain more directly concerned with the phenomena of consciousness, as the hemispheres; or upon parts which, when excited, may cause convulsions, as the mesocephale, the region of the tubercula quadrigemina. If the former be chiefly affected, and the latter slightly or not at all, convulsions are either very slight or do not constitute a part of the epileptic fit. If, on the other hand, the latter are chiefly and primarily disturbed, convulsions form the prominent part of the fit. Now, the exciting cause of all this disturbance generally operates equally on both sides of the brain. But it may operate more on one side than the other. It leaves behind it a more or less exhausted state of brain; which, again, will be most upon that side upon which there has been the greatest previous excitement. This state of exhaustion is very apt to continue as one of weakened nutrition, in which the brain-tissue is more or less in the condition of white softening. If the parts involved in this be the convolutions, mental power, memory, perception, suffer; if deeper parts, as the deeper parts of the white matter of the hemisphere, and the corpora striata and optic thalami, then we have hemiplegic paralysis.

Let me relate to you two cases in illustration of the state of brain which is apt to coexist with the tendency to repeated attacks of epilepsy. They show how an atrophic state of brain may be produced by the same cause which excites the epileptic paroxysm, followed by a paralytic state.

Case CCVIII.—Elizabeth Tribett, aged thirty-eight (vol. xvi, p. 164), a married woman, of temperate habits; admitted January 27th, 1846; had always enjoyed good health. Twenty-

one months ago, without warning or assignable cause, while working at a mangle, she fell down in a fit, with complete loss of consciousness, without stertor, and apparently with but little convulsion. She did not recover from this for twenty-four hours, and then she was found to be paralysed on the left side and speechless. In a fortnight she recovered from this attack, and was able to resume her work.

Six months afterwards she had another fit, the insensibility lasting only a few hours; but speech was again lost, and the left side was paralysed. From the effects of this attack she recovered only very partially and slowly, although she had been sent into the country for change of air.

She now gradually fell into a state of general paralysis, with feebleness of mental powers, and great imperfection of speech. The left side was distinctly weaker than the right. All the functions became feeble and sluggish, and bed-sores formed on the nates. The sphincters did not fulfil their office, and she sunk by slow degrees. Just previously to her death she became comatose, and was slightly convulsed. She died three months after her admission into the hospital.

The inspection of the body revealed the following appearances, indicative of a chronic enfeebled nutrition of the brain and its membranes.

The Pacchionian bodies were very large, and the arachnoid membrane was extensively opaque. The hemispheres of the brain had a flaccid, flabby appearance; the sulci between the convolutions were wide, and occupied by a considerable amount of sub-arachnoid effusion. Atheromatous spots were found in the arteries at the base of the brain, especially in the anterior parts of the basilar and middle cerebral artery. The right lateral ventricle was dilated, and contained an ounce of fluid; the foramen of Monro was large, but the left ventricle did not much exceed its natural size. The septum lucidum was remarkably thin, but firm. The corpus striatum of the right side was distinctly smaller and less prominent than that of the left; it was also softer; its gray matter was especially soft. There was a slight degree of softening of the cerebral matter of both hemispheres, and great paleness of the gray matter of the convolutions; these states were especially marked on the right side.

The other organs were healthy, but exhibited a good deal of congestion in their most dependent parts.

The effusion into the right ventricle in this case was due to the shrinking of the corpus striatum, and also, perhaps, of the whole of the right hemisphere. It seems to me that effusions into the ventricles, when not caused by venous obstruction or inflammation, obey the same laws as those in the sub-arachnoid space. The corpus striatum and optic thalamus are internal convolutions, and when they shrink fluid occupies the space which they abandon, just as fluid accumulates over the site of a shrunk external convolution.

Case CCIX.—The second case many of you will remember, as it is not long since the patient was in the hospital. Abraham Metcalfe, aged thirty-nine, a carpenter, admitted November 22d, 1852. Nothing could be elicited from this patient or his friends, as regards his own antecedents or his family history, to account for the occurrence of his malady. He had been always healthy till last Christmas (1851), when, while engaged at his work, he was seized with "queerness" and inability to stand, and was obliged to sit down till some one came to help him into the house. He did not lose his senses, nor was he ever unconscious, but found that he had lost the use of his right arm and leg, and the sight of his right eye, and also his speech.

He recovered from this attack so perfectly in ten or eleven weeks, as to be able to go to work again.

In April, as he was walking home, he was suddenly seized as before, with precisely the same symptoms. He recovered in a great degree from the paralysis, but never so completely as to be able to resume work.

He had a similar, although slight, attack on the 14th November, with increased paralysis of the right side, and his speech was much impaired.

On his admission he was found to be generally feeble in body and sluggish in mind; the right arm and leg were decidedly weaker than the left; and a feebleness of gait, with trembling of the limbs, indicated a general weakness of his nervous system. His speech was slow and indistinct, and he seemed to collect his thoughts with difficulty. For the last three or four months he

had been in the habit of passing a large quantity of urine. Shortly before his admission he passed, according to his wife's statement, as much as eight pints in one night. The specific gravity of the urine was 1012; it contained neither albumen nor sugar.

After he had been a week in the hospital, the arm and leg recovered very much; the grasping power seemed quite to return, and he could hold things out from him steadily, and button his shirt, which he could not do before.

He continued to improve until the 31st of December, when he suddenly lost the power of the right side as before, the arm being chiefly affected, and the speech almost entirely lost for a few minutes. He went to bed, and in a few hours was as well as before the attack.

On the 8th of January he had another attack, accompanied by loss of speech and hemiplegia of the right side. From this attack he did not seem to rally; the paralysis, which was more complete than on any previous occasion, remained; he passed his evacuations under him, and became more and more lethargic. On the 13th of January he was seized with a slight convulsive fit, and died in a few hours afterwards. The pupils were throughout large and sluggish, and the left a little larger than the right. The daily quantity of urine fell considerably after he had been a short time in the hospital, but still exceeded the normal amount.

On examining the head there was found an abundant subarachnoid fluid, with some opacity of the arachnoid membrane, that on the left side exhibiting the greatest amount of opacity. The convolutions were somewhat wasted, and the sulci large. The substance of the cerebral hemispheres was softish, and seemed soaked with fluid; and this was more marked in the left than in the right hemisphere. Both ventricles were large, and contained a considerable quantity of fluid. The left optic thalamus was larger and more flaccid than the right; nothing abnormal was discovered in the corpora striata or the other parts of the brain, excepting that they all participated in the diminished consistency and soaked appearance. The basilar and vertebral arteries were large, but free from deposit. There was no indication of either local or general congestion.

In this case the attacks, although not strictly epileptic in

their nature, were undoubtedly of that character. Consciousness was impaired, though not suspended. Whatever may have been the immediate exciting cause of these paroxysms, it or they seemed to increase, for a time, the damage done to the general nutrition of the brain, and after each succeeding attack the patient showed less rallying power.

This case resembles the preceding one in the general atrophic state of the brain, and the consequent increase of the sub-arachnoid and intra-ventricular fluids. I regret much that the brain was not microscopically examined, as we should probably have obtained more decisive evidence of the exact nature of its impaired nutrition. The greater degree of softness of the left hemisphere than of the right, and the more lax state of the left optic thalamus, sufficiently indicated that the nutrition of the left side of the brain had suffered the most, and accounted for the hemiplegia being on the right side of the body. It is very desirable that in all cases of this kind in future, not only should the two sides of the brain be compared as regards their minute structure, but also as to their specific gravity.

One word more I must add. The effusion of fluid in the ventricles in this case, and the increased quantity of that in the sub-arachnoid space, were due simply to the general wasting of the brain, and had no influence in producing the paralysis. They should be regarded, in a case like this, as the consequence of that general wasting; and, indeed, it is only when such wasting occurs that water in the ventricles exists at the same time with an increased quantity of sub-arachnoid fluid.*

* I have elsewhere stated that intra-ventricular effusions and sub-arachnoid fluid do not generally coexist. A large effusion into the lateral ventricles pushes away the sub-arachnoid fluid, doubtless into the spinal canal, or prevents its secretion. So also does any enlargement or great congestion of brain, or a tumour, or a large apoplectic clot. This statement holds good in all cases in which the brain is firm and not wasted, and the exception to it is found in cases like those above detailed (CCVIII and CCIX), where there have been during life more or less of general paralytic symptoms and failure of cerebral function, associated with impaired cerebral nutrition. My friend, Dr. Parsey, of the Warwickshire Lunatic Asylum, has communicated to me the details of several cases of general paralysis, such as are of common occurrence in every asylum, in which fluid has existed simultaneously, within and outside the brain, along with a more or less atrophic state of the whole organ.

I must here conclude this lecture, reserving the consideration of choreic and of spinal hemiplegia until another occasion.

LECTURE XLVII.

ON PARALYSIS.

EPILEPTIC HEMIPLEGIA—CHOREIC HEMIPLEGIA—REMARKS ON THE DIAGNOSIS OF THE VARIOUS FORMS OF HEMIPLEGIA NOTICED IN THE PRECEDING LECTURES—SPINAL HEMIPLEGIA.

The cases related at the conclusion of my last lecture serve to show what kind of mischief may be done to the brain, through the disturbance caused by the epileptic paroxysm. They are examples of an aggravated amount of disturbed nutrition in the brain, ending in general softening, and a corresponding extensive weakness of the physical and mental nervous actions.

In Cases CXLVI and CXLVII, of which I have given a detailed account in former lectures,* the attack, which ultimately terminated fatally by a considerable effusion of blood, began with an epileptic fit. In the first of these cases the early epileptic seizures were followed by hemiplegic paralysis, from which the patient partially recovered, in the intervals between the attacks. In both, the last attack ushered in the fatal rupture of blood-vessels.

The early attacks, in these cases, many would call apoplexy, *i. e.* coma from compressed brain. But I have shown that there is no good reason for supposing that the brain is, either in whole or part, subjected to compression in such attacks. And I have called them attacks of *epileptic coma*, which are often associated with diseased kidney, which may or may not

* Vide Lecture XXXVIII, p. 672, and Lecture XXXIX, p. 682.

be accompanied by convulsions. It is very possible, and even likely, that the fatal rupture of blood-vessels and effusion of blood may be caused by the congestion which *follows* the impeded breathing and the struggle of the epileptic paroxysm; and these accidents are the more likely to occur when the capillary blood-vessels are in a state of fatty degeneration.

Epilepsy in its most acute form, with one convulsive fit after another, and a comatose state prolonged for many hours, will occur without any anatomical indication of compressed brain. I have related, in my lectures on Delirium and Coma, a remarkable case, in which not only coma, but paralysis of the right half of the body, ensued without any compression of the brain. The brain was not healthy, it had shrunk somewhat, and there were congestion of its surface and of the convolutions, and a large sub-arachnoid fluid, with a small quantity of fluid in the ventricles; and the only morbid condition which seemed to explain the hemiplegia was slight softening of the left corpus striatum. There was chronic disease of the kidneys, and in this, no doubt, as well as in some chronic disease of the arteries, originated the depraved and imperfect nutrition of the brain, and the fatal attack of paralysis and coma.

Patients suffering from an enfeebled condition of the heart, especially that from fatty degeneration, are liable to sudden attacks of coma. These cannot certainly be apoplectic in their nature, *i. e.* from too much blood compressing the brain, or from an effusion of blood. The examination of many of these cases shows that the latter did not exist, notwithstanding that the comatose attacks had been frequent; and there are strong reasons for believing that the want of a proper supply of blood to the brain will induce a comatose state. The erect posture, for example, is favorable to these attacks, and they are sometimes relieved by the assumption of the horizontal position, or by hanging down the head. A patient here, two or three years ago, was seized with attacks of this kind whenever he sat up in bed; and Dr. Stokes relates, that a patient of his learned to ward off such attacks by placing himself on his hands and knees, with his head dependent. They occur likewise in cases of diseased heart, in which, from regurgitant vulvular disease or from some other cause, the blood is not expelled

from that organ, either in proper quantity or with adequate force.

A very striking case is related by Dr. Stokes, as observed by Dr. Fleming, in which the patient was liable to these pseudo-apoplectic paroxysms, or epileptic fits, as I would call them, which came on generally at night or during sleep. The patient came out of each of these attacks perfectly paralysed on the left side, and also jaundiced. Both the hemiplegia and the jaundice would subside in a very short time, the former within a few hours after the attack, and on the following day scarcely a trace of jaundice could be seen.*

"It was found," says Dr. Stokes, "that these attacks were only to be treated by the use of stimulants. During one of them, owing to a different course having been adopted, in the absence of Dr. Fleming, the patient was brought into the most extreme state of collapse. The stimulants had been withheld and the head blistered; but even under these circumstances, so decided was the effect of stimulants, that the patient, who in the morning was hemiplegic, was within six hours completely restored to the use of his limbs."

The lesion in this case consisted in an enormous dilatation of the left ventricle, with great enlargement of the mitral orifice, which must have admitted a very free regurgitation. It was unfortunate that permission was refused to examine the brain; but from the absence of any permanent brain symptoms, it seems certain that no decided lesion could have existed. The state of the brain, in all probability, corresponded with that of the early periods of the cases of epileptic hemiplegia which I have described.

Many of the epileptic seizures which take place only or chiefly in the night or during sleep, in elderly persons, I have reason to believe, have some intimate connexion with a morbid condition of heart—an altered nutrition of its muscles rather that of its valves.

* *Vide* p. 206 of Dr. Stokes' recent important and interesting work on 'Diseases of the Heart and Aorta,' Dublin, 1853. I would suggest that the jaundice in this case was a paralytic symptom, due to a temporary paralysis of the biliary ducts, analogous to the instances of jaundice from strong mental emotion or shock to the nervous system.

Sleep exercises an important influence on the respiratory function—on the generation and exhalation of carbonic acid. Thus an altered state of blood, and a modified action of the heart, are apt to occur in sleep, and these cannot fail to affect the brain in a way which may be favorable or unfavorable, according to the previous condition of that organ.

Choreic hemiplegia.—I shall now say a very few words on the hemiplegia which is associated with chorea. In the large proportion of cases of chorea, as I have often remarked to you, the choreic movements occur more on one side than on the other, and sometimes they will be altogether confined to one side—the child being hemiplegically affected in a very exact manner. When, in such a case, the choreic movements have wholly or in great measure subsided, the patient remains paralysed in the limbs, which were before the seat of convulsive movements. The phenomena now resemble, in many points, hemiplegia, from a decided lesion of the brain. But you will, I think, generally observe the following points of difference. First, you will often find the face not affected, or if it be, only slightly so; secondly, there will not be any paralysis of the tongue, but more or less of the peculiar mode of protrusion which characterises chorea; thirdly, the paralysed limbs will exhibit, even in a very slight degree, the choreic movements.

Case CCX.—A boy, nine years of age, was brought to me in December, 1848, with well-marked signs of hemiplegia of the left side; he dragged the leg, and had but very feeble power of the arm; the muscles were quite lax. The face was slightly paralysed. He protruded the tongue with the choreic thrust. I learned that he had been the subject of chorea, affecting chiefly the left side, for a few weeks. The choreic movements still existed, but to a very slight degree.

The patient was treated by citrate of iron, shower-baths, and exercise of the limbs, and in eight days he returned nearly well. He recovered perfectly.

Case CCXI.—Charlotte Plater, aged eighteen (vol. xlii, p. 239), a sallow-looking maid-servant, was admitted into

Augusta ward on November 8th, 1854, with highly developed symptoms of chorea, affecting the right side of the body only. Neither the patient nor her friends could assign any cause for the attack, except, perhaps, that her daily work had been very hard, and that her food had been poor and scanty. The catamenia appeared regularly, but she had been suffering from leucorrhœa for six months prior to her admission into the hospital. After being in the hospital a day or two, the choreic symptoms almost disappeared, but left behind a paralysed condition of the muscles of the right side of the body. The account of this, given by my clinical clerk, Mr. Teale, is as follows:—"The chorea seems in great measure to have taken its departure, but has left behind it hemiplegia of the right side; there is scarcely any jerking of the muscles of the face or of the tongue, but, in walking, she appears *uneasy* in her right foot; her right arm is paralysed, her grasp with the right hand is very feeble, and she has great difficulty in picking up a pin; she complains of numbness of the right arm, hand, and shoulder, but not of the right leg or foot."

The treatment to which this patient was subjected was of the supporting kind, consisting of a generous diet, together with small doses of steel three times daily, under which her general health gradually improved, and the hemiplegia pretty rapidly disappeared; so that, on December 16th, *i. e.* about five weeks after her admission into the hospital, she was discharged with the full use of all her limbs.

I may mention, in connexion with this case, that the patient was, at the commencement of the treatment, ordered a *splash-bath* every morning; but, as indeed in practice you will find every now and then happens, the first application of the cold water produced so much fright that it was considered inexpedient to repeat it.

Case CCXII.—Another case, illustrative of this form of hemiplegia, is that of a little boy, named Henry Windred, who was admitted into the hospital on October 8th, 1853 (vol. xliii, p. 82). This child, who was five years of age, had always been healthy up to twelve months before his admission, when he was taken, in his mother's words, "with a trembling motion of both

arms," but whether the legs were affected in the same way or not there is no evidence to show. These trembling movements continued three months, without getting materially better or worse, when, one morning, whilst being washed, he became suddenly hemiplegic on the right side, the palsy being unaccompanied by any loss of consciousness. Two or three days subsequently to this attack, the muscles of the right side of the body became affected with the irregular jerking movements of chorea, and in this state he came under our observation. He was at once treated by upholding the general nutrition of the body in every way, and he gradually improved, the choreic movements became less and less, he slowly regained the use of his right side, and was discharged from the hospital on November 17th, about five weeks after his admission.

It is needless to multiply cases of this kind. They are of daily occurrence in hospital practice in a more or less perfect form. The case which I have related may be regarded as a more exquisite example of the paralysis.

This form of hemiplegia is liable to be confounded with one which sometimes occurs in children from tubercular disease of the brain, and the more so as the latter form is very apt to commence with jerking movements of the arm or leg, or of both, on the side opposite to the seat of the tubercular deposit. The two conditions are to be distinguished by the absence, in the tubercular form, of the more decided chorea symptoms, and of the peculiar thrust of the tongue; by the existence, generally, of pain in the head, and of a scrofulous diathesis; by more or less of a constitutional disturbance, vomiting, fever, and general debility; and by the occurrence sometimes of general convulsions.

Now you will ask what is the actual condition of the nerves and nervous centres upon which choreic hemiplegia depends? I answer, that it is very analogous to that which I have already explained in speaking of epileptic hemiplegia. Chorea being due to a disturbed nutrition of some part of the brain in intimate connexion with the centre of volition, the disturbing cause may act exclusively on one side of the brain, or it may operate more on one side than the other. The effect of this disturbance is first manifested in an irritative state, creating the

choreic movements, and this passes sooner or later into an exhausted or paralytic state.

Opportunities for post-mortem examination after chorea, and especially the partial form to which I am now referring, are extremely rare. I have not had one of this latter form in my own experience. When such occur, the various parts of the brain constituting the centre of volition and the centre of emotion, the corpora quadrigemina and the superior part of the mesocephale, should be most diligently searched, and it is very desirable that the specific gravity of corresponding parts on opposite sides should be ascertained.

An interesting investigation of the brain in a case of general chorea has been recorded by Dr. Aitken, in the 'Glasgow Medical Journal.'* He noted the important fact, that the specific gravity of the corpus striatum and optic thalamus on the right side was 1·025, and that of the same parts on the left side was 1·031.† Further observations on this subject are greatly needed, and if made with the same minuteness and exactness as those of Dr. Aitken, will no doubt throw great light on the pathology of chorea and other allied affections.

I must bring my remarks on these forms of hemiplegia to a close, with some allusion to their diagnosis, prognosis, and treatment.

To distinguish the different forms of hemiplegia which I have brought before you from each other is not a matter of much difficulty. Having carefully noted the actual phenomena presented by the patient, you will derive great aid in determining the particular form to which to refer his case, from the previous history of the mode of access of the present attack. You should also investigate the patient's constitution, and inquire into his family history. It is important to learn as much as possible respecting his habits. Fail not, likewise, to ascertain the state of his kidneys, by a careful examination of the urine.

* "Contributions to Pathology," by William Aitken, M.D., 'Glasgow Medical Journal,' No. I.

† According to Dr. Bucknill's observations, the average specific gravity of healthy brain is 1·036; according to those of Dr. Sankey, the gray matter afforded a specific gravity of 1·034, the white matter 1·041.

I cannot too strongly impress upon you the importance of this latter point. In every case of nervous affection, this should be among your first inquiries. How many cases, formerly supposed to be anomalous, are now readily understood by reference to uræmic poisoning, through inefficient kidneys! Moreover, there are many other points of interest in connexion with the state of the urine in brain disease which can only be settled by many observers; such, for instance, as the presence of sugar,—not uncommon in epilepsy,—the variations of the phosphates, the quantity of the sulphates and the chlorides; and whether, in the marked increase or decrease of these salts as elements of the urinary excretion, we can derive trustworthy aid to determine the inflammatory or the non-inflammatory nature of the brain-lesion.

A case of hemiplegia presenting itself, you may ascribe it to a special lesion of the brain, in a large proportion of cases, provided the face and tongue be affected in the usual way, on the same side as the limbs. This opinion will be confirmed if you find sensibility impaired on that side—if there be or have been pain in the head, as a marked symptom, on the side of the head (or towards it) opposite to the paralysis; and if there have been premonitory symptoms prior to the paralytic attack, such as pain, cramps, numbness, or convulsive jerks in either of the affected limbs. A dilated state of one pupil, generally that on the opposite side to the palsy, is also a symptom which should excite your fear for cerebral lesion. The sudden invasion of the paralysis would be, on the whole, confirmatory of this view, although, in certain forms of brain-lesion, the palsy may come on gradually. In most instances, the suddenly developed palsy indicates, as I think you will find, solution of continuity of brain-fibres, either alone or caused by rupture of blood-vessels. The completeness of the paralysis is in favour of the existence of brain-lesion, which is irritative when you find the muscles contracted and rigid from the time of the seizure, atrophic or non-irritative if they be flaccid. A suspension of consciousness may or may not occur in connexion with the paralysis of brain-lesion, as is abundantly exemplified in the cases detailed in the preceding lectures. But a sudden suspension of consciousness, whether with or without paralysis, should be always

regarded by you as among the gravest of symptoms, as certainly indicating brain disturbance, either primary or secondary.

When after a sudden attack of suspended consciousness, with or without convulsions, sometimes indeed with no more evidence of the latter than a bitten tongue, the patient comes to himself pretty soon, with a hemiplegic paralysis, which goes off in no very long time, to be succeeded, after an interval of complete or nearly complete recovery, by an attack precisely similar; such a case is a typical instance of what I have designated the Epileptic Hemiplegia.* In investigating the history of cases of this kind, it is important that you should inquire

* The following remarkable case has occurred to me while the pages of the first edition of this work were passing through the press:

Case CCXIII.—A gentleman, aged sixty-seven, of very full plethoric habit, was sitting at his club, writing letters, and had nearly completed a fourth letter, when he fell from his chair to the left side. He was taken up semi-comatose and sleepy, and was immediately attended by the late Mr. Warren Fincham. I saw him within half an hour of the attack, and found complete paralysis, with relaxed muscles, on the left side, and well-marked face and tongue palsy on the same side. He continued in a sleepy state for two hours, but would answer questions when addressed loudly. For three quarters of an hour after the attack there was difficulty of deglutition, and imperfect ptosis of the left upper lid. The pulse was slow and irregular, and the heart's action weak. Neither Mr. Fincham nor I thought that the state of the heart's action warranted depletion.

He was subjected, therefore, to no other medical treatment than placing him nearly horizontally, and giving occasionally small quantities of water, and now and then a little brandy and water.

After two hours the sleepiness and stupor began to pass off, and he showed signs of recovering the power of moving the leg and arm; and in another hour he was able to grasp well, and to move about his leg freely, and to stand. In half an hour more he walked out of the club to a cab, and after he had got into bed he grasped my hand with nearly as much power as he could exert with the right hand.

He was now freely purged, and took small doses of ammonia through the night. The next day there was no remnant of paralysis, except in the left cheek, and slight external strabismus of the left eye. This disappeared on the following day. On the third day he seemed quite well, the face and tongue palsy having disappeared likewise; and he walked about his room with his usual strength and gait.

The phenomena of this case accord best with those of Epileptic Hemiplegia. The attack was epileptic, and probably associated with incipient white softening

particularly respecting the previous existence of syphilis.* Contamination by this poison is very frequently at the root of the epileptic condition, and of the cerebral disease which attends it, either as cause or effect. (Case CCXIX.)

To determine the nature of the cerebral lesion involves the discussion of the following questions: 1. Is the cause of the palsy a cerebral softening? and, if so, is this inflammatory or non-inflammatory? does it depend on some previous chronic disease of the brain, as a tumour, which may be benign or malignant, or a scrofulous deposit, or meningeal disease? 2. Is the lesion an apoplectic clot, which has torn up more or less of brain-substance? 3. Is the paralysis due to a compression of the brain, by serous or exudation deposit, or effusion of blood on its surface? 4. Are the symptoms in any way dependent on disease of the kidneys?

I cannot attempt to answer these questions fully in the present lecture, and must limit myself to directing your attention to one or two points, which will materially aid you in the solution of them.

First, the co-existence of disease of the heart, especially an enfeebled state of the muscular structure or of valvular disease, whether obstructive or regurgitant, with hemiplegia, is very favorable to the diagnosis of non-inflammatory softening, and, if signs of compression exist, of apoplectic effusion. Secondly, in the vast majority of cases of hemiplegia occurring after fifty years of age, which are accompanied by evidence of diseased arteries and an arcus senilis, you will have strong reason to suspect white softening, and (if other symptoms tally) consequent apoplectic effusion. Thirdly, an early impairment of consciousness will, in a large number of instances, indicate sanguineous effusion, and the existence of slight early rigidity of some of the paralysed muscles will confirm this. The comatose

of the right hemisphere, from fatty degeneration of the capillaries. A premonitory symptom of this condition had occurred ten or twelve days before, in a violent cramp of the left gastrocnemius muscle, which caused considerable subcutaneous ecchymosis.

* There is a peculiar class of cases of epileptic hemiplegia, in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power and speech. These I propose to discuss in a future lecture.

affection will be the greater the nearer the clot is to the pons Varolii and medulla oblongata, and the more the brain is compressed and the greater the quantity of blood effused. Fourthly, the sudden or rapid development of coma is in general due to an apoplectic effusion. The more gradual development of it is dependent on an inflammatory softening, or an intraventricular, serous, or sero-purulent effusion, or on an exudative effusion on the surface of the brain, or on renal disease. But when you have to deal with a case of coma, you will not lose sight of the fact that it may be epileptic; and in adopting or rejecting such a view, you must be guided by the mode of access of the attack, and the previous history of the patient.

With reference to the prognosis, I would give you this advice: —"In all cases of paralysis, but especially in cases of hemiplegia, be particularly cautious in giving a prognosis. First, because it is often very difficult to form an accurate opinion as to the precise nature of the lesion, although we can generally tell whether there be lesion or not. Secondly, because few persons who are hemiplegic ever completely recover; for, if once the brain be so damaged that hemiplegia results, it is very uncertain whether the powers of life are sufficient to restore or repair the injured parts; and a fresh attack, or consequent epileptic fits, are to be apprehended. The form of hemiplegia, dependent on lesion of the brain, which offers the best prospects for recovery, is that kind of white softening (if not of great extent) which occurs in young and otherwise tolerably healthy subjects, if the brain recover its nutrition by the restoration of the circulation, the obstacle to the free flow of blood having been overcome, or a sufficient collateral circulation having been established.

The *hysterical hemiplegia* also promises ultimate recovery, but this is often very slow; and the same may be said of the *emotional hemiplegia*. In that form of hemiplegia which is associated with *epileptic fits*, the prognosis is not in general satisfactory. The fact of the occurrence of hemiplegia shows that the brain has suffered more from the fit than is usual. It is, in my experience, very unfavorable in hemiplegia generally, when total loss of speech precedes, or occurs simultaneously with, the attack of paralysis. These cases, although the disease

may not immediately prove fatal and destructive to life, seldom perfectly recover the paralysis, and still more rarely regain the power of speech. That fine old man, Ryan, now in Fisk ward, is an instance of this nature. He had an attack of hemiplegia of a slight kind, but with complete loss of speech; the hemiplegia has recovered, but, although his intelligence appears good, he makes no progress in speaking. If time allowed, I could adduce several other cases demonstrating the accuracy of this statement.

Now as to TREATMENT.—Looking over all the forms of hemiplegic paralysis which I have described, the antiphlogistic plan of treatment is strictly and fairly applicable only to that in which the rigidity of the paralysed muscles occurs early, and it must then be employed proportionately to the strength and age of the patient; but recourse to *large* bleedings is certainly not justifiable in any case with which I am acquainted. By a large bleeding I mean such as exceeds ten or twelve ounces taken at once. The remedies to be used in such a case are mercury, free purging, and general or topical bleeding; but, in the other forms of hemiplegia, no extensive antiphlogistic treatment should be adopted, and especially in the simple hemiplegia without loss of consciousness, which is purely an atrophic disease. You should adjust the diet to the powers of the stomach, keep the patient in the horizontal position, as quiet as possible; and carefully guard against all causes of mental agitation or excitement.

Many advocate very much the use of mercury in hemiplegic cases. It is a valuable purgative, perhaps the most valuable we possess, and as such it may be used in nearly all the forms. But, for its specific influence, it ought only to be employed where cerebral inflammation exists, either primarily or around a clot, or where there has been a syphilitic taint. To use it with this object in the atrophic hemiplegia, or in apoplexy, appears to me to be worse than useless. Its employment in this way is also to be especially avoided when there is renal disease and albuminous urine.

Then, with respect to the treatment of the paralysed limbs, some have strongly recommended the use of *strychnia*, while others have as strongly advocated the employment of *electri-*

city; but when the paralysis is the result of cerebral lesion, neither of these remedies promises much good, and they very frequently do harm. In the administration of strychnia, the greatest caution must be used; and if electricity be employed, it should be of feeble intensity.

There is one curious fact with respect to the exhibition of strychnia in these cases, which was first noticed by Fouquier; it is, that this agent first shows its effects on the paralysed limbs. This fact was some time ago brought forward by Dr. Hall, to show that in paralysis dependent on cerebral lesion the irritability of the paralysed muscles is augmented. Sufficient facts have, however, now been collected to prove that this statement is not correct—that the muscular irritability in such cases is not increased, though in certain instances the paralysed muscles may be more excited by a galvanic stimulus than the sound ones. The reason why strychnia first manifests its action on the paralysed limbs is, because it is attracted in greater quantity to the diseased side of the brain than to the healthy side, and it there excites an irritative condition, which is propagated to the paralysed muscles; and this ought to point out that the use of strychnia in these cases is by no means devoid of danger, as it tends to produce at least an irritated, if not an inflammatory, condition of brain around the seat of lesion.

The chief points, then, to be attended to in the treatment of hemiplegia are the careful employment of ordinary hygienic measures, and the promoting the nutrition of the paralysed limbs, by moving them by mechanical means.

An affection of the bladder sometimes occurs in cases of hemiplegia, which causes much trouble to the patient and anxiety to the physician. I have seen it most developed in men of gouty constitution, and have thought it in some degree influenced by gout. It consists mainly in an urgent desire to pass water without a corresponding readiness of expulsive power. As far as I can determine, it is not influenced materially by the quality of the urine, excepting so far as that a highly acid or a highly alkaline state of the urine augments the distress. It will occur with urine of the highest and the lowest density.

The best remedies for this state are the tincture of cantha-

rides, the cold *douche* over the region of the bladder, the dilute nitric acid carried to a high dose, and narcotics introduced by the rectum.

I shall now call your attention to Spinal Hemiplegia, a rare form of hemiplegic paralysis.

It needs but a very elementary knowledge of the anatomy of the spinal cord to show that hemiplegia from disease of that organ can only occur when the paralysing lesion is seated high up in the cord, just below the decussation of the anterior pyramids, and where it is very exactly limited to one half the cord, —*i. e.*, to one side of the median fissure.

The following case will afford you a very perfect example of the affection, and explain to you, better than any general description, the clinical history of this form of hemiplegia.

Case CCXIV.—Marianne Catlin, aged sixteen, admitted November 18th, 1849, had been always a delicate child, and suffered from headaches; she was pale and thin. On her admission, she complained of pains in the head, not fixed, and some stiffness of the neck. There was complete paralysis of the left arm, with flaccidity, and some wasting of the muscles. In walking, she dragged her left leg with a sweeping movement. The case had at first very much the appearance of hysterical paralysis; and this view of its nature was favoured by the absence of all palsy of the face and tongue. She was of delicate frame, and the catamenial function had been suspended for six months. She complained of a pain at the vertex, which was just that to which hysterical women are very subject. In organic disease, in women, symptoms purely hysterical are often coincident even in a prominent form, and embarrass the practitioner not a little. Although the palsy of the arm was complete, as regards motion, she retained some power of the leg, and could move it slowly up and down, as she lay in bed. But she was unable to hold it up from the bed. Sensibility was impaired, although she retained a considerable amount of feeling in the paralysed side.

On further examination of this patient, our attention was arrested by a marked stiffness of her neck, in consequence of

which the head was drawn to the right side, and the face looked forward, and rather to the left. There was a very rigid state of the right sterno-mastoid muscle, such as one commonly sees in ordinary cases of wry-neck. On the left side the neck presented considerable deep-seated swelling in the region of the uppermost cervical vertebræ. It was evident to the touch that this swelling was not due to any accumulation of fluid, nor were the integuments and muscles in any way diseased. They were stretched over the swelling, and a thickened state of bone and ligamentous tissue could be felt through them. The rotatory and other movements of the head were much impeded; those to the right existing only to a slight degree; those to the left being limited by some mechanical hitch connected with the vertebral articulations. The patient complained very much of pain in this part of the neck, both when pressure was made upon it and at other times; it was especially painful at night, and she found it difficult to obtain a position of ease in which to lay her head. Her nights were consequently much disturbed.

On being questioned as to the origin of this affection of the neck, she stated that it had commenced about six months before her admission by stiffness and pain, the head being drawn to the right side. Soon afterwards it began to swell, and as the swelling increased, she suffered much more pain. The palsy of the arm appears to have come on gradually, as she was unable to fix the precise date of its occurrence; but it was certain that it had come on subsequently to the appearance of the swelling. She first noticed weakness of the leg on the 2d of November, a fortnight before her admission, and this had gradually increased ever since. She attributed the pain and swelling of the neck to a cold, and denied that she had ever received a blow there.

The distinct limitation of the paralysis in this case to one side excited our interest very much; and we discussed the various causes which could have produced it. Was it cerebral? Was it hysterical? Could any connexion exist between it and the cervical swelling? Excepting the pains in the head, which seemed very much of the same nature as those in the neck, there was no other cerebral symptom but the palsy, and this

differed from that usually dependent on brain lesion, in the absence of any affection of the face and tongue. The hysterical constitution was scarcely sufficiently developed to lead us to regard it as hysterical hemiplegia. But we found, in the pain and swelling, and the impeded motion of the neck, and the paralysis coming on subsequently to, and increasing *pari passû* with, the cervical enlargement, quite enough to explain the phenomena. I, therefore, expressed the opinion that the paralytic symptoms were due to a disease of the spinal column; that an enlarged odontoid process, or some other swelling at the upper part of the spine, had been for some time slowly compressing one half of the spinal cord just below the decussation of the anterior pyramids; and that if the disease were not arrested, death would result from a gradual process of "pithing" by the extension of the compression to the opposite side, and by the extinction of the respiratory process. By the compression, the connexion between the medulla oblongata and the spinal cord (the seat of implantation of the phrenic and other nerves, which influence the breathing movements) would be cut off, and the mechanical acts of respiration would be rendered impossible.

From her admission to the 11th of December, our patient manifested no additional symptom of importance. She suffered much from pain in the neck and loss of rest. She was treated by iron and iodide of potassium; opiates at night; aperients when necessary; and counter-irritation by iodine paint, and afterwards opiate applications to the neck. A generous diet was allowed her.

On the 11th of December, my clinical clerk, Mr. T. C. Dickinson, on visiting her in the morning, found that she had lost the use of the left leg completely, and that she was totally unable to do anything for herself. She complained also of twitching on the left side of her body. At three o'clock, p.m., on the same day, I saw her, and found complete paralysis, not only of the left lower extremity, but also of the intercostal and abdominal muscles of the left side, which remained perfectly motionless during respiration.* A remarkable sign indicated

* I am disposed to regard this latter paralysis as specially diagnostic of

the want of equilibrium between the right and left abdominal muscles in expiration; the umbilicus at each expiration was distinctly drawn to the right side. Sensibility was not more impaired in the paralysed limbs than on her admission. No difference could be observed between the two sides as regards temperature. The pulse, which hitherto had not exceed 100, now rose to 120.

On the 12th, after a restless night, the right arm was found to be partially paralysed; she was just able to flex and extend the fingers slowly and feebly. The power of the sphincters was lost, and the fæces and urine were passed involuntarily. On examining the respiratory movements, it was found that the action of the diaphragm on inspiration was so feeble, that it could scarcely be felt in its descent. No contraction could be detected in the intercostal muscles of the left side, and the very slight action of that side of the chest seemed to depend on the movements of the right side. The respiratory murmur in the left lung was distinctly more audible than that in the right, especially in front. The heart's sounds were quite normal. Pulse 120, respirations 32.

On the 13th she appeared to suffer more pain in the neck, and the least movement increased it so much as to make her cry out. Sensation was more impaired in the left arm and leg, and she complained of a want of power over the right leg. Pulse 112, respirations 32.

On the 14th the following note was made:

9¼ p.m.—The paralysis continues to increase. It is complete of the whole of the left side, below the neck, and of the right arm. Voluntary power over the right leg is rapidly failing; she can but just move the toes and contract the muscles of the calf, but not sufficiently to move the foot. No reflex movements can be excited. Respiratory movement seems to be performed exclusively by the right side, and feebly by the diaphragm. Complains of great pain in the neck when it is moved. Pulse 120, respirations 32.

A few minutes after this report was made, Mr. Steele, the

spinal hemiplegia, In the most complete cerebral hemiplegia these muscles are rarely affected. *Vide* p. 711.

house-physician, was called to her, and found her speechless, with livid face and purple lips, breathing in gasps at intervals of twenty seconds. The only muscle which could be observed acting was the sterno-mastoid of the right side; there was no perceptible motion of the ribs of either side; no abdominal movement; pulse full, 90; heart's action good at first; it afterwards however, gradually became slower and more feeble; the pulse ceased at the wrist, and in twenty minutes the beating and sounds of the heart could no longer be distinguished. Some breathing was perceptible for a minute or two afterwards, and she ceased to exist. It is impossible to conceive a more easy or gradual mode of cutting the thread of life.

It was not a little remarkable how small an amount of disease was capable of producing such dire results. The disease consisted chiefly in an enlargement of the odontoid process of the second vertebra. This extended backwards, wearing through the dura mater, and it was covered at its upper part, and on the left side, by a fibro-cartilaginous growth, which compressed and flattened the spinal cord on the left of the median fissure. The compression of the cord was so great, that it seemed as if a large portion of the nervous matter had been pushed from the left to the right side and partly upwards; and the cord was swollen both above and to the right of the compressed part. The nervous matter on the left side was soft and slightly discoloured, as from small ecchymoses; that on the right of the fissure was very soft and diffluent. The pia mater of this portion of the cord was red and congested. No other disease was found in any part of the body. It was plain, then, that the left hemiplegia, which first arrested attention in this most interesting case, was caused by the compression of the left half of the spinal cord, just below the decussation of the anterior pyramids, as the pressure became gradually more and more complete. The extension of the paralysis to the right side was due to the softened and diffluent condition of the right half of the cord, a state of imperfect nutrition which, no doubt, was likewise dependent on the pressure exercised by the enlarged odontoid process.

Did time permit, I might occupy hours in discussing the many interesting points in physiology suggested by this case. The striking illustration which it affords by contrast to the law

of crossed influence in brain disease; the interesting natural experiment, which it showed, to prove how the association of brain and cord is necessary to voluntary actions; and how the severance of but a small link in the chain, notwithstanding that nearly the whole spinal cord was intact, is sufficient to destroy these, and ultimately other actions; the proof which it yields that but a small bond of union is necessary to preserve sensibility, if not intact, at least in a very good state.*

I shall add the particulars of a patient who recovered very much under treatment.

Case CCXV.—Daniel Bryant, aged twenty-four (vol. xliii, p. 130), a bricklayer's labourer, admitted November 14th. This man appears to have been healthy all his life, and free from syphilitic taint as far as could be discovered.

For five or six weeks prior to his admission into the hospital he had suffered from a pain in the back of the neck, which prevented him from throwing his head back, and greatly impeded its motion from side to side. For this he was treated as an out-patient, without improvement. On the 4th of November, after a sudden feeling of weakness, which led him to sit down for a minute or two, he found a partial loss of sensation and motion in the left arm and leg; he dragged the leg, and both it and the arm felt colder than those of the right side.

On his admission, there was partial paralysis of motion in the left arm and leg; he dragged the leg in walking; he did not admit any defect of sensibility in the leg, although there still existed some in the arm. Reflex action good. There was no symptom of brain affection, except a slight difference in the size of the pupils.

The motions of the neck were very limited, and the patient always presented an appearance of great stiffness in that region. The lateral motions of the neck were much impeded; and a decided fulness and thickening were felt in the upper cervical vertebræ.

* A case resembling this in several particulars has been recorded by Dr. Bright—viz., Case CXCVII, vol. ii, Part 1, of 'Bright's Reports.' I find a more perfect example, as regards the hemiplegic paralysis, recorded by Dr. Cathcart Lees, in the "Transactions of the Pathological Society of Dublin," December 1843.—'Dublin Journal,' February 1846, p. 221.

This swelling and enlargement were most conspicuous on the left side, as if in that region the greatest alteration in the condition of the bones or ligamentary textures had taken place.

The relative sensibility of the two sides was tested by points of compasses, and it was found that two points were much more readily appreciated on the right than on the left side.

On passing the galvanic current through both arms, due attention being paid to the direction of the current, it was found that the muscles of the left arm responded less readily to the galvanic stimulus than those of the right.

Our attention was also directed to the movements of the chest, as it was reasonable to expect that a compression of the cervical regions of the spine, sufficient to cause weakness of the leg, would affect these movements more or less. It was found that the breathing was less loud on the left side; and that the movements of the ribs were less marked on that side.

The diagnosis in this case was between cerebral and spinal hemiplegia; and, in adopting the latter, we were influenced mainly by the absence of face palsy, and the existence of what seemed to be a considerable thickening (probably rheumatic) of the ligamentous structures of the upper cervical vertebræ, especially of the left side. Nor was there any symptom, except the palsy, indicative of brain disease.

The patient had a long sojourn in the hospital, during which he was treated chiefly by iodide of potassium. Four grains of this salt dissolved in an ounce and a half of distilled water were given three times a day; after a few days the dose was increased to ten grains, and the back of the neck and head was ordered to be painted with iodine paint.

This treatment was begun on the 13th of November. On the 28th of November it was reported that the fulness of the neck was very much diminished, and the movements much more free. A corresponding improvement took place in the condition of the limbs, especially of the leg.

The change for the better continued until the 19th of December, when, from some cause (perhaps an increase in the rheumatic condition consequent on change of diet, as he had been allowed full diet ten days before), increased pain and stiffness, with swelling of the neck, came on, and some diminution of power in

the leg and arm. A blister was now ordered to the nape of the neck, and five grains of blue pill to be taken night and morning; the iodide of potassium was omitted; and fifteen grains of the mercurial ointment were to be rubbed in over the back, night and morning.

On the 3d of January free salivation had been established; the neck was much better, it could be moved without pain. All medicines were discontinued for some days. On the 18th, as no further diminution had taken place in the cervical swelling, a seton was introduced at the back of the neck, and the syrup of the iodide of iron was directed to be taken in doses of one drachm three times a day. Under this treatment a gradual improvement took place; and on the 21st of February he left the hospital much better in every respect.

It is worth recording, in connexion with this case, that on three different occasions the urine was found to contain sugar in notable quantity by the test of liquor potassæ and sulphate of copper.*

I know of no form of spinal hemiplegia so perfect as that which these cases illustrate. A tubercular or other deposit, occupying with great exactness one half of the cord; or an apoplectic clot in a similar situation, or a softening strictly limited to one side (a very unlikely occurrence), would produce a hemiplegic state on the same side of the body; but I have not met with any such cases.

It is not uncommon to meet with cases in which, from a lesion affecting one side of the spinal cord more than the other, the paralysis is greater in one limb than the other.

* I regret much that more minute observations were not made in the case of Catlin (CCXIV), as to the state of sensibility of the paralysed limbs. It was certainly diminished, and not augmented, as Brown-Séquard's most interesting experiments would lead us to expect. In the case of Bryant, the reader will have remarked that the sensibility was diminished.

ADDENDUM TO LECTURE XLVII.

I THINK it worth while to subjoin to the case detailed in the preceding lecture the particulars of a very remarkable case of temporary hemiplegia, which seemed to me to be of spinal origin. I give them as an extract from a clinical lecture on that, among other cases, in December, 1850.

CASE CCXVI.—The third case on my list is that of Benjamin Mattey (vol. xxxi, p. 136), now in Fisk ward; it is very deserving of your attention in some points. He is twenty-five years of age. He comes from Woolwich, where he is a workman in the Arsenal. With the exception of an attack of ague four years ago, when he first went to Woolwich, he has enjoyed good health. Ten months ago, a plank fell on his left side, and struck him in the interval between the last rib and the crest of the ilium. He suffered severe pain in the lumbar region immediately after the accident, and although he was enabled to return to his work in the course of a few weeks, the pain returned, and gradually increased, and was accompanied with imperfect *paralysis of motion of the arm and leg of the left side*, with impairment of sensation. He was now received into one of the metropolitan hospitals, where he received considerable benefit from the treatment to which he was subjected. He was freely salivated, and twice cupped. In a short time the power of sensation returned nearly completely, and he lost the pain in his back; he likewise recovered the power of his limbs in some degree. After this he was enabled to resume his work; but in nine days he was again obliged to leave off, in consequence of a recurrence of the pain. He was now placed under the care of my friend, Mr. Gallwey, surgeon in the Royal Artillery, who was then stationed at Woolwich. Mr. Gallwey established an issue in the

side, and gave him iron and quinine, and cod's liver oil. Under this treatment he improved slightly, but not permanently. He was admitted into the hospital on the 14th of November, and at this time the pain had assumed to a remarkable degree a periodical character. It commenced at night when he went to bed, and lasted for three or four hours, subsiding gradually. On awaking in the morning, he would feel a soreness in the region, where the pain had existed the previous night. It was a relief to get out of bed, and assume the erect posture. But this, as all other movements of the trunk, was done with great care and caution. Any sudden contraction of the muscles of the left side caused severe pain—pain somewhat of the same description as that which a man would suffer from sudden muscular action, when labouring under a fit of lumbago.

A careful examination of the spine discovered nothing wrong there; but a decided pain was produced by pressure over the region of the quadratus lumborum muscle, and the act of coughing or sneezing was extremely painful.

From the fact that the pain had its seat where the injury occurred, and also because it was excited by muscular exertion, when the lumbar muscles on the left side were in action, it seemed to me clear that great part of the pain was due to mischief going on in the muscles and fasciæ of the left lumbar region—the result of the injury inflicted by the fall of the plank. Some of the muscular fibres had been probably ruptured, and perhaps also some of the nerves themselves had suffered. The periodical nature of the severe pain led to a suspicion that it might be regarded as neuralgic (the position being determined by the injury, and the periodicity by some remnant of the marsh poison, which may have remained in his system since the attack of ague), and Mr. Gallwey seems, judging by his treatment, to have taken much the same view.

But there are other symptoms of the case which do not admit of so easy an explanation; the paralysis, namely, of sensation and of motion. The parts affected are the left upper and lower extremities. I read the following account from the case book: —"The sensibility of the left arm is decidedly diminished, so that he cannot distinguish two points of a compass placed on it or the fore-arm, an inch apart. He is unable to grasp

firmly with, or completely to extend the fingers of the left hand. The sensibility of the leg is not so much diminished, but he is not able to bear much of his weight upon it, and he walks as if his legs were tied together. There was no tenderness of the spine, and he has full power over the sphincters."

Now, the hemiplegic character of the paralytic affection would certainly seem to indicate, *primâ facie*, that it had its origin in the brain. But this view is quite untenable, seeing that he is perfectly free from every other symptom of disease of that organ. It seems to me that we can only explain his symptoms by supposing an extension of some inflammatory condition from the lumbar nerves and muscles to the dura mater of the spinal cord. The chief difficulty in the way of adopting this view arises out of the extent of the paralysis, which involves the supposition of a corresponding extent of affection of the dura mater; and it is difficult to conceive the existence of so extensive disease of the dura mater with pain so completely limited to one spot. But if we consider the intimacy of the connexion of the dura mater of the spine with the spinal nerves, we need not scruple much to admit that a slight affection of that membrane would impair the power of the nerves sufficiently to explain the existing degree of paralysis. Or it might be supposed that the injured nerves exercise some depressing influence on the region of the cord, in which they are implanted, and this, by its reflection, may cause a similar state of depression of nerves higher up. On the whole, the more probable view seems to me to be, that the injury has created an inflammatory condition of the fibrous tissues external to the spine, and that this is extending to the meninges, and perhaps even to the bones.

At first we treated this man on the supposition that the pain was purely neuralgic, and he had large doses of carbonate of iron. After three days' trial of that plan, I resolved to deal with it as a case of inflammation of a fibrous tissue, and then gave him iodide of potassium in ten-grain doses thrice a day.

The results of this treatment were extremely interesting, and very deserving of your attention.

On the 20th of November, he began to take the large doses

of the iodide. A decided iodism made its appearance in a very short time. After the first dose he felt an uneasiness in his head, and after the third dose he began to feel as if he were tipsy; and a profuse discharge flowed from the nose, and the eyelids became œdematous, with a slight blush of redness; at the same time there was a flow of saliva as copious as if he had been freely salivated. In consequence of these symptoms, he was not permitted to take a fourth dose of the medicine. During the latter part of the 21st and the 22d, he took no medicine. On the 22d there was evidently a considerable improvement as regards the pain; most of you will remember how distinctly the expression of his countenance indicated relief from pain. On the 23d the iodism had completely subsided, and I now ordered the iodide of potassium to be resumed, in the same doses as before. Immediately after taking the first dose on the second occasion, there was a return of the flow of saliva to the amount of half a pint; but on continuing the use of the medicine, the ptyalism subsided, and a tolerance was established. Under this treatment he improved greatly—the severe periodical pain subsided—he is able to move about much better, and is regaining the power and sensibility of his limbs.

It is worth while to notice here a curious change in the characters of the urine, which occurred while he was under the influence of the first doses of iodine. On the 20th November the urine was pale, and the sp. gr. 1006. On the 23d, the quantity was much as before, that is, normal, but the sp. gr. had increased to 1030, and there was a copious deposit of the lithic acid; and on the 25th, the sp. gr. was 1035; there was an abundant precipitate of lithate of ammonia, and there was evidence of the presence of bile in the urine from the play of colours caused by the addition of nitric acid.

I presume no one can doubt that, in this case, the peculiar group of symptoms which we call iodism, the diminished pain, the increased power in the limbs, and the remarkable changes in the urine, bore, to the iodide of potassium as administered to him, the relation of effect to cause. Doubtless the iodide has promoted elimination through the kidneys and the salivary glands of some material which at least was maintaining a febrile state of his system. Can it be that the man was of a rheu-

matic or gouty diathesis, increased by a sedentary life since the accident, and that this kept up and increased the pain?

I fear that the favorable change will not be permanent, and is only due to the temporary clearing out he has experienced from the iodide; and I am led to this opinion by the fact, that on a former occasion he experienced nearly as great improvement from the use of mercury, but speedily relapsed.*

* The further history of this case was as follows:—The patient continued to improve up to the 11th December, having taken the iodide (to each dose of which, on the 29th of November, five grains of citrate of iron were added) during the whole time. He then left the hospital, stating that he could walk better than ever he did since the accident. The day after he left the hospital the pain returned, he became gradually worse, and was re-admitted January 1st, 1851. The pain was now continuous; there was spinal tenderness over the last lumbar vertebra, the pain was much increased by turning in bed or stooping; he moved with extreme caution: a sudden jar or false step was exquisitely painful. The weakness of leg and arm had returned. He was told that his symptoms indicated disease of the vertebra, that great quiet would be necessary, and that for a long time. He then proposed to leave the hospital, preferring to remain at home. He was discharged on the 10th of January, and since then it has been ascertained, through the kindness of my friend, Mr. Fogo, of the Royal Artillery, that he was discharged from the dockyard, having shown unequivocal signs of caries of some of the lumbar vertebræ, with paralysis of the lower extremities.

LECTURE XLVIII.

ON COMA AND PARALYSIS.

ON A CASE OF EPILEPTIC COMA AND LEAD PALSY.

In Fisk ward we find a man of the name of J. Clarke, aged thirty (vol. xxxii, p. 21), who was admitted here about two months ago, Oct. 1850.

Case CCXVII.—This case exhibits an example of two very interesting and highly important classes of disease—epilepsy, and the effects of lead poisoning. He was admitted in a state of profound epileptic coma; and from October the 18th to the 20th, he had five convulsive fits, accompanied and followed by coma, which continued for two days, when he began to recover.

The long continuance of a state of profound coma is always calculated to excite anxiety in the friends and attendants of a patient, and to create fear lest some extensive mischief shall have been done to the brain. In this case there were two circumstances among others which greatly encouraged me to believe that no serious lesion existed in that organ. The first of these was, that the coma was accompanied with epileptic convulsions. Now you will not understand me to say that I do not apprehend danger from such paroxysms of epilepsy as this man has had; but that, when coma is followed by a succession of attacks of epileptic convulsions, and when there is no hemiplegic paralysis, we have a good deal of presumptive evidence that there is no apoplectic clot, or other organic lesion likely to damage the brain permanently. Thus we were led to ascribe both the coma and epilepsy, not to the pressure of a clot upon or within the

brain, but probably to one and the same cause, which cause was suggested by the second circumstance to which I have referred —namely, that his urine was scanty in quantity, and highly impregnated with albumen. I viewed the case, then, as one of those in which the cerebral affection was due to the presence of some irritating matter in the blood which ought to be eliminated by the kidneys. There are very good grounds for believing that when urea is retained in the blood, the brain is very likely to be affected so as to cause coma and convulsions. Other substances retained in undue quantity may produce the same effect, for aught we know; and certainly, coma and convulsions may occur in cases where we have no evidence of the presence of urea in the blood; but it is quite as certain that when the kidneys fail in their action and secrete only an ounce or two of urine in a day, instead of thirty or forty ounces (whether the poisonous agent be urea or something else), coma and convulsions are very apt to ensue.

And in many such cases we get proof of the presence of urea in the blood, as we have done in this case. The following is the method which was pursued for this purpose:—A blister was applied to the back of the neck; and when it rose, the serum was carefully collected, and tested for urea. The whole quantity of serum was evaporated to dryness over a water-bath, and the residue was extracted with alcohol, which is a ready solvent of urea. This alcoholic extract was then evaporated to dryness, and a little water added so as to make a syrupy mass, which was plunged into a freezing mixture, and a few drops of pure nitric acid were added. If urea be present, the characteristic crystals of nitrate of urea are soon formed in the solution, and may be recognised either by the naked eye or by the microscope.

Our patient is an intemperate man, and filled the place of a waiter at a low tavern. We have not been able to trace any immediate exciting cause of his present attack, except exposure to cold shortly before. It is probable that, under the influence of his habits of intemperance, renal disease had been making insidious progress for some time; but, on exposure to cold, the action of the skin having been checked, an acute affection of the kidneys was induced, these organs became highly congested;

their tubes being filled with epithelium, allowed but a small flow of urine charged with serum. This defective secretion of urine causes urea or some other poisonous material to accumulate in the blood, which, passing to the brain, so far disturbs the nutrition of that organ as to excite convulsions, or coma, or both.

As soon as recovery from the state of coma took place, we observed a paralytic affection of the upper extremities, and of those alone; the legs were in no way involved. Nor did the paralysis involve all the muscles of the upper extremities: those of the arm and shoulder were only very slightly affected; the muscles of the forearm were chiefly engaged, and of these the extensors were most distinctly paralysed. All were wasted; but the extensors more so, as was obvious from the hollow which existed over those muscles on the back of the forearm. His power to flex the wrist and grasp with his fingers was very feeble, but he was wholly unable to extend the wrist or the fingers. When the arm was stretched out from the trunk, the hand hung, as if lifeless, from its articulation at the wrist. Both upper extremities were affected in precisely the same way, although not precisely to the same degree; for the right forearm was evidently weaker than the left. The muscles which form the prominence of the ball of the thumb (the *thenar* eminence of surgical anatomists) were also paralysed and greatly wasted, and the power of flexion, or extension, or adduction of the thumb, was almost entirely destroyed.

It is plain that in this case there were two classes of symptoms with which we had to deal—an acute class, which yielded pretty readily to treatment, and which must have destroyed the patient sooner or later if they had not given way; and a chronic, which remained after the first had been removed, and which seemed to have no tendency to shorten life. The first were the coma and convulsions—the second, the palsy.

Viewing the first and acute class of symptoms as indicative of the state to which I have given the name *renal epileptic coma*,* I adopted a treatment actively eliminatory, with a view to remove by other channels, as much as possible, the material

* Lectures on Delirium and Coma. 'London Medical Gazette,' 1850.

which was disturbing the brain. The channels through which I endeavoured to conduct this noxious matter, were the intestinal mucous membrane and the skin.

His head was shaved, and he was freely blistered over the scalp; mustard cataplasms were applied to the back of the neck, and he was very freely purged. The best purgative to use in such cases is elaterium, because its acts promptly, and produces liquid stools, carrying off large quantities of water from the system, which, no doubt, like that obtained from the blister, contained urea. Warm baths, or hot-air baths, may often be used with great advantage in these cases; but I did not allow Clarke to take more than one of each, as I thought him too weak to be subjected to remedies which, especially the latter, have a decidedly depressing influence.

He was admitted on the 18th of October; on the 22d he was quite free from any comatose symptom, and there now remained to be dealt with the condition of the kidney (the *fons et origo* of the cerebral disturbance), and the paralytic state.

The urine had increased considerably in quantity; it was still, however, very highly albuminous, becoming almost solid by heat, and of low specific gravity; and, under the microscope, contained casts of tubes and epithelium, and some blood-corpuscles. It was plain that, whatever might have been the former state of the kidney, it was *now* very much irritated, and that the defective secretion and albuminous impregnation were due to this.

The condition of the kidneys has improved greatly in this case under the further use of purgatives, at first elaterium, and afterwards compound gamboge pills, and also of warm baths; so that now the urine contains very little albumen; but the paralytic state has remained very little changed.

It exhibits precisely the character of that form of palsy which results from lead-poisoning, more commonly known as the "painter's wrist-drop." All the characteristic signs of this form of palsy were as well marked in this case as in any case I have ever seen. When you make him hold out his arms, you see both hands hang down, and he has no power to bring them to the state of extension.

A practical man could not see such a case without asking if

the patient was a house-painter. Yet we found, on inquiry, that not only was he not of that trade, but that his proper vocation, that of a waiter, did not particularly expose him to the lead contamination.

Can the palsy be due to any particular lesion of the nervous centres, independently of lead? Or is it the result of the renal disease? It is not likely to be the latter, for it existed before the attack of coma, and its origin dates as far back as four or five years, and was preceded by two attacks of which he gives but a very imperfect account, but which were attended with obstructed bowels and severe pain in the belly—attacks resembling lead colic. He was at this time in Devonshire; but he states that he did not drink cider.

I do not think that the palsy can be attributed to any special lesion of the nervous centres. I know of none which would produce exactly this form of paralysis—so symmetrical—affecting particular classes of muscles in preference to others, and those of the forearm especially, and without any damage to sensation.

We are led, then, to attribute the palsy to contamination by lead; and, in confirmation of this, we have found the blue margin to the gums. The blue line is not uninterrupted, for he has lost several teeth, and at these points it ceases; but wherever there is a tooth, or a stump projecting above the gum, there the blue line is distinct.

But the difficulty in this case has been to explain how the lead came to be introduced into the system. It is true we are exposed to this contamination from the water we use, and we shall be so, as long as there are leaden cisterns and leaden pipes to convey the water. In time it may be hoped that glass may be substituted, or some other substance not likely to yield up poisonous matter to the water. The wonder is, not that an occasional case of this kind occurs, but that they are not infinitely more frequent. Some people, no doubt, exhibit the idiosyncrasy of being strongly affected by very small quantities of particular poisons, which it is generally necessary to administer in much larger doses to produce their specific effects. We see this often in the use of mercury and of iodide of potassium. It is possible that this man may have this idiosyncrasy as regards lead.

I think we have a better explanation than this. It appears

that a part of our patient's duty was to clean and keep bright the pewter pots belonging to the public-house to which he was attached. This he did by friction with his hands. Now pewter very commonly contains lead in considerable quantity; and no doubt the frequent contact of this with the hands would lead to a gradual absorption of a sufficient quantity of the metal to produce the poisonous effects; or the repeated frictions might cause the separation of minute metallic particles, which might be inhaled.

The palsy from lead is most probably due primarily to the contamination of the muscles by the lead: these structures become impaired in their nutrition, and the nerves, especially the motor nerves, suffer in consequence of their comparative inaction. Doubtless, after long exposure to the contaminating influence, the nervous matter itself will become poisoned, and thus in such cases brain symptoms ultimately show themselves. But the palsy may be regarded as a form in which the loss of motion is in the first instance due to a diseased state of the *muscles* themselves,—not, as is generally the case, to disease of the nerves, or of the nervous centres.

The theory of lead palsy, which refers it to lesion of the spinal cord or brain, evidently will not account for the phenomena. It will not account for the muscles of the forearms being chiefly affected; nor will it explain the symmetry of the affection; nor the greater palsy and wasting of extensors than flexors; nor the special affection of the muscles of the thumb; nor, in fine, the almost total exemption of the nerves of sensation amid so much injury to the motor function. Moreover, I think it may be affirmed with perfect truth, that a lesion of the spinal cord sufficient to create so much palsy of the upper extremities, as we often see in such cases as these, would necessarily affect other parts also; whereas this man Clarke has full power of his lower limbs, his mind and senses are perfectly clear, and he has no symptom of any nervous affection besides the palsy of the upper extremities.

I have already explained to you the treatment adopted in this case for the renal disease. Fortunately, that necessary to remedy the effects of the lead poisoning is much of the same kind. He has had frequent sulphur baths, with evident advantage to his ge-

neral health. I have seen many instances of great improvement to the state of lead cachexia following the long-continued use of sulphur baths.* He has also gone through a long course of galvanism with very little benefit. One arm has been fixed upon a splint, according to Dr. Pemberton's plan, while, for the sake of comparison, the other arm was left free. It was evident after this treatment that the arm which had been bound up was worse than before. On the whole, the lead palsy is very little better than on his admission; nor is it to be expected that it should be, in so short a space of time. The poison of lead damages the muscles so much, that it requires a very long time before any marked change takes place: and I do not know that any one remedy exercises a specific influence, unless it be iodide of potassium; but there can be no doubt that all those hygienic means, which contribute to promote a sound general nutrition, are the most useful in aiding the recovery of the patient.

The sequel of this case is very interesting.

The patient, Clarke (vol. xxxvii), left the hospital in January, 1851, after a sojourn there of three or four months, during which time all his symptoms improved very much, the albumen having disappeared almost completely from the urine, and the arms having become stronger; but a certain amount of weakness of the extensor muscles still remained. He did not return to his former employment, but went into the country, and he continued well until the summer of 1852, when he again applied for admission into the hospital, in consequence of increased weakness of the arms. Under the sulphur treatment he again improved, and he remained well until the 22d November of the same year. He now began to feel very unwell, and on the 24th was seized with violent pains in the belly, and cramps, and pains in the thighs and legs, for which he was admitted a third time on the 26th November.

These symptoms, which seemed to arise from either gouty or lead colic, were quickly subdued by free purging, warm bath, and counter-irritation by turpentine stupes applied to the surface of the belly, and a single dose of morphia, and on the 27th he

* *Vide* Lecture XXXIII.

was very much better. But his urine was found to be highly albuminous.

On the morning of the 29th he complained very much of swimming in the head, which rapidly got worse, and he went off into a fit of epilepsy, which lasted but a short time; during the fit the muscles of his face and limbs were slightly convulsed, the fists were clenched, the eyes were fixed in the upward direction, and he foamed at the mouth; and was quite unconscious. The urine was of sp. gr. 1015, and contained abundance of albumen. The quantity of urine passed could not be ascertained, but there was reason to believe it was below the normal amount.

The following note of the condition of the urine on the 1st of December was made by Dr. Evans. Its quantity, in the last twenty-four hours, was two pints and a half, sp. gr. 1016, pale, clear, acid, deposits a slight densish sediment, and contains a considerable amount of albumen. Its microscopic appearances are as follows:—The whole field is covered with pus-cells chiefly agglomerated in masses; a mass of smooth, oval, flat, epithelial particles (probably from the urethra); a granular cast with one epithelial cell upon it; a large waxy cast (from a tube probably deprived of its epithelial lining); a granular cast about $\frac{1}{700}$ inch in diameter; there is no appearance of the pus being moulded.

Thus we had quite sufficient evidence in all these symptoms to indicate that, while our patient was out of the hospital, whether from exposure to cold, or intemperate and irregular living, or all combined, fresh irritation and inflammation of the kidney had come on. He was ordered the use of the hot-air bath thrice a week; his diet was regulated, and purgatives occasionally administered.

Under this treatment he improved steadily in general health and strength; the quantity of urine increased considerably, averaging three pints a day, sp. gr. 1012, and presenting much the same characters as those already described, with a very gradual diminution in the quantity of albumen. On the 31st of December, the quantity of albumen was about one eighth of the portion examined; no cast could be seen, but there were still several pus-cells. He was now ordered small doses of the tincture of the sesquichloride of iron.

On the 15th of January, the bowels having been constipated, probably by the iron, he had a fresh attack of colic, accompanied with violent vomiting and retching, which lasted for three days. On the 20th, another epileptic fit occurred, just like that on the 29th November, which left him very debilitated and giddy, with dim vision. From the commencement of the attack of colic, the quantity of albumen in the urine increased considerably. On the 19th, the urine became almost solid by heat, and numerous pus-cells were seen in the sediment.

On the 24th, he had a fit of the gout in both great toes; he stated that he had frequently had similar attacks of gout before, preceded by colic. Since the appearance of gout in his toes, the abdominal pain had completely ceased. The quantity of urine increased; on the 25th it was five pints and a half, the albumen still very considerable, but diminishing, On the 22d he was ordered the bromide of potassium, in doses of three grains thrice a day.

He now gradually passed into his former state. All traces of gout disappeared. He passed urine largely, averaging four pints daily, sp. gr. 1011, its sediment containing pus-cells, some uric acid, and casts occasionally; and on the 3d of February the quantity of albumen had diminished to the same point as before the fit, namely, one eighth the quantity examined.

On the 13th, however, a very marked increase having taken place in the quantity of albumen, I ordered him to have the hot-air bath twice a day, hoping by promoting increased action of the skin, to forestal any further attack of epilepsy.

This treatment apparently answered; the quantity of urine was now the same as the quantity of liquid drunk; and the albumen gradually diminished. On the 19th, the hot-air bath was given up, and he was ordered fifteen minims of dilute nitric acid thrice a day.

On the 23d, a fresh attack of gout appeared, without any colic. This speedily gave way without any special treatment, and he steadily improved. On the 1st of March, 1853, he left the hospital, having gained strength, the paralytic state being much the same, and the kidneys acting as before.

I had repeatedly called the attention of the Class to this case,

as affording illustration of so many morbid processes. We had, in this one patient, an example of lead-poisoning causing the peculiar palsy; we also had that which frequently accompanies the lead-contamination, namely, gout; there was also colic, which probably was gouty; there were diseased kidneys, likewise gouty; and although last, not least, there was epilepsy.

In June of 1853, Clarke was again admitted into the hospital, having been for some time treated as an out-patient, for rheumatic pains. He now resumed the use of the hot-air baths, and improved slightly under them. After having been a fortnight in the hospital, he was seized with seven epileptic fits in rapid succession, from the exhaustion consequent upon which he died.

The post-mortem examination took place on the 18th.

The kidneys weighed each three ounces and three quarters; their surfaces were slightly uneven and granular. Numerous cysts existed in them, most of which were very small, but one was as large as a walnut. Many of the tubes were atrophied and denuded; many of them were opaque with granular disintegrated epithelium. The small arteries were thickened, and their coats hypertrophied.

No pus could be discovered in the tubes or in any part of the kidney. It seems most likely, therefore, that the numerous pus-cells which were so often found in the urine, were derived from the mucous membrane of the pelvis of the kidney and of the ureter and bladder (gouty irritation). That mucous membrane, however, after death, exhibited no marks of disease.

The brain was exceedingly pale and free from blood; but in other respects, as far as the eye could determine, its characters were those of health.

In consequence of the prominence of the nervous symptoms, and the evidence of that contamination previously alluded to, I thought it desirable that the specific gravity of different parts of the brain should be carefully ascertained, and that both it and the paralysed muscles should be examined as to their physical state, and also with reference to the presence or absence of lead. This inquiry has been most ably conducted by my friend, Dr. Conway Evans, whose report is as follows:—

"The experiments, by which the following results with

respect to the specific gravity of the brain were obtained, were performed on the evening of June 18th, 1853, sixteen hours after death.

"The specific gravity of the various parts of the brain, given in the annexed table, was obtained in the usual way for determining the specific gravity of a solid, heavier than water, and insoluble in, and not chemically acted on by that liquid. A piece of brain substance was first weighed in air, and then in distilled water; and by a comparison of these weights the density of the portion of brain experimented on, in relation to water as 1000, was determined; or, in the words of the school-books, 'the weight in air was divided by the loss in water,' the quotient so obtained being the specific gravity.

"The weights of the various portions examined were in every case very accurately taken, the greatest care being used that any mistake which might unavoidably occur should be less than the one-hundredth part of a grain.

"The weights of the several portions of which the specific gravity was ascertained, varied from 56·09 to 248·8 grains, the average of the whole sixteen experiments being about 115 grains; and great pains were taken in estimating the density of similar parts on opposite sides, to use, as nearly as conveniently could be done, equal weights of corresponding parts (although in several cases the weights differed considerably), and also to remove those parts from exactly similar situations on each side of the brain.

"*The following Table shows the results obtained in this manner:—*

Part of Brain	Sp. Gr. Water being 1000.
White substance of right hemisphere	1040·8
White substance of left hemisphere	1039·5
Grey matter of convolutions of right hemisphere	1035·8
Grey matter of convolutions of left hemisphere	1036·8
Right Corpus Striatum	1040·0
Left Corpus Striatum	1039·6
Right Optic Thalamus	1040·6
Left Optic Thalamus	1041·1

Part of Brain.	Sp. Gr. Water being 1000.
White and gray matter of right hemisphere of Cerebellum	1042·9
White and gray matter of left hemisphere of Cerebellum	1043·2
Right side of Pons Varolii	1039·7
Left side of Pons Varolii	1039·5
Right Crus Cerebri	1038·0
Left Crus Cerebri	1039·0
Right half of Medulla Oblongata	1037·9
Left half of Medulla Oblongata	1037·9

"The extensor muscles of both forearms, but especially those of the right, were of a very pale, almost fawn colour, and had a very wasted appearance; and on tearing, they were found to be much tougher than healthy muscle. Under the microscope they appeared to have become converted almost wholly into wavy bands of 'white-fibrous' tissue. Here and there a muscular fibre could be seen running amongst the fibrous bands, but in these the transverse striæ were very ill-defined; indeed there seemed to be hardly any transverse markings at all, and the whole fibre had a peculiar pale, faintly granular appearance. There were not the least indications of fatty degeneration. When treated with acetic acid, the whole specimen displayed vast numbers of nuclei, chiefly elongated and fusiform, their long diameter being, for the most part, in the direction of that of the fibres.

"The red, healthy muscle, when submitted to microscopic examination, exhibited no departure from the normal structure.

"A portion of the diseased and healthy muscles, and also of the brain, were subjected to chemical analysis with the view of detecting the presence of lead in those tissues.

"The portion of healthy muscle experimented on weighed 774 grains, and the diseased muscle 736 grains; and the process employed, which was precisely alike for each, was as follows:— The muscular tissue, having been thoroughly dried over a water-bath, was incinerated, the temperature being gradually raised to a very high degree. By this means a carbonaceous residue, containing all the fixed salts, was obtained, and this was carefully examined throughout to ascertain the existence of any minute metallic globules, which would probably have been

formed, had the muscle contained lead, in consequence of the reduction of the metal by the carbon of the organic matters. No metallic globules, however, having been discovered, the carbonaceous residue was boiled with dilute nitric acid and filtered; and through the clear solution a stream of hydrosulphuric acid was passed, with the view of precipitating, in the form of sulphide, any lead which might exist in solution.

"In the case of the healthy and of the diseased muscles, the results obtained were precisely similar; for in neither case was there the slightest trace of a metallic sulphide thrown down, the only precipitate being some sulphur, resulting from the decomposition of some of the hydrosulphuric acid.

"Portions of various parts of the cerebrum and cerebellum (chiefly the white substance), weighing in all 4000 grains, were also analysed; and the process adopted was exactly similar to that employed in the examination of the muscles, except that the carbonaceous residue, obtained by incineration, was boiled with a solution of carbonate of soda and filtered, previous to its being treated with dilute nitric acid. The object of this was to remove as much as possible of the fixed salts before getting the lead, if any were present, into solution. When the clear acid solution, obtained from the carbonaceous residue, was subjected to a stream of hydrosulphuric acid, a clotty, orange-yellow precipitate, very like in appearance the tersulphuret of antimony, was thrown down. This precipitate, when thoroughly dried, weighed 0·33 grain; but it was, most probably, nothing more than sulphur in combination with organic matter; for, when heated, globules which, when examined under the microscope, were seen to be evidently those of sulphur, sublimed, while a carbonaceous residue remained behind."

"So far, then, as the process employed in these cases is accurate, it appears, from these analyses, that the portions neither of the healthy nor of the diseased muscles, nor of the brain, which were examined, contained lead in appreciable quantity; and inasmuch as this method is an exceedingly delicate one, it seems fair to infer that had lead, even in minute amount, been present in these tissues, it would have been detected as the black sulphide, when its solution in dilute nitric acid was subjected to a stream of hydrosulphuric acid."

We are not yet sufficiently certain as to the normal specific gravity of the brain to justify any inference being drawn from the facts above collected on that point. What I have now recorded must remain, as a contribution to that large series, which has yet to be accumulated, before we can legitimately begin to reason upon this subject.

As to the absence of lead, this is not to be wondered at, notwithstanding the still wasted state of the muscles, when we consider how long a time had elapsed since the lead poisoning (between four and five years), during which time he had been frequently under medical treatment. Viewing the palsy as due to lead, this inference may be drawn, that the impaired nutrition, previously induced by the lead contamination, may not become restored even after some years. By an unfortunate oversight, the nerves of the diseased muscles were not examined.

In juxtaposition with this case of Clarke, I shall place another—in many respects very similar—illustrating how the contamination of gout generating diseased kidney may give rise to further contamination of the blood, out of which shall arise the most formidable and ultimately fatal disturbance of the brain's function. The following account of the case is taken from notes kept by my clinical clerk, Mr. C. Macnamara.

Case CCXVIII.—Mary A. Parry, aged forty-four (vol. xlii, p. 5), admitted May 19th; a married woman, and mother of twelve children, the youngest eighteen months old, has menstruated very profusely within the last three days. Her father died of gout and dropsy; mother died young.

This woman had, on admission, a remarkably dirty, sallow complexion (very common in cases of chronic renal disease); she had been losing flesh very much for some time, and was in a state of great debility. Ever since her twenty-fourth year she had been subject to attacks of gout. Almost every year she has had an attack, which sometimes lasted only a week, sometimes five or six weeks. It affected both large and small joints of the extremities, and has left deposits in the tendons and ligaments of the finger-joints. For some time past her breathing has been getting short, especially upon exertion.

The illness, on account of which she sought admission into the hospital, came on five weeks before with increased dyspnœa, and an attack of gout in her ankles. The gouty attack yielded, but the ankles remained swelled, and from that time her lower extremities and abdomen quickly became dropsical; and the quantity of urine passed, which previously had been considerable, gradually dwindled down to only a pint in the twenty-four hours. The whole of the lower extremities were œdematous, and the integuments of the abdomen and of the loins were in a similar state; and a considerable quantity of water had accumulated in the peritoneum, causing great distension of the abdomen. The urine was loaded with albumen. Pulse very small and weak; heart's sounds feeble and distant, with a very distinct mitral systolic bellows-sound. The superficial jugular veins were large and distended, and when compressed in the neck, did not empty themselves readily on the cardiac side of the point of pressure. She had cough, with free mucous expectoration, and a good deal of crepitation was heard throughout both lungs.

Upon microscopical examination by Mr. (now Dr.) Conway Evans, the urine was found to contain crystals in such abundance, that everything else which composed the sediment was almost totally obscured; here and there, however, a large and a small waxy cast were seen. The crystals were chiefly thick, yellowish lozenges (uric acid), and large, irregular-shaped masses, apparently consisting of the above-mentioned lozenge-shaped crystals aggregated together. With these there were many of the masses provided with projecting needle-like processes, generally regarded as urate of soda, and also many colourless, rectangular prisms; also a good deal of broad pavement-epithelium, probably from the vagina.

The case was regarded as one of gout, with gouty kidney, upon which acute inflammation had supervened. To this was added disease of the heart consisting of imperfection of the mitral valve, probably from shrinking of the chordæ tendineæ and the fibrous basis of the valve, with dilatation of the ventricles, especially that of the right side; œdema of the lungs; congestion of the liver, consequent on the feeble action of the heart. As a distinct grating was felt on moving many of the small

joints of the fingers and toes, it was inferred that their cartilages were roughened by gouty deposit, and it was conjectured that a deposit of the same nature damaged the valves of the heart.

Under the use of diuretics and sudorifics, the quantity of urine increased to upwards of a quart for the first ten days, the albumen being still very abundant, and the belly diminished in size. On the 30th, a diminution took place to a pint and a half; the pulse rose from 100 to 120, she had passed a bad night, and she complained of being much troubled with convulsive movements of the arms and legs.

On the 31st, at six in the morning, she got out of bed and sat for a short time before the fire, as if she had been chilly. Soon after getting into bed, she was suddenly seized with a fit, in which the whole body was convulsed, she foamed at the mouth, and the tongue was protruded between the teeth and bitten. This was followed by several other fits, each lasting about ten minutes, leaving her in a state of extreme prostration and exhaustion, and apparently insensible, until another fit was about to come on, when she would get up in bed and answer questions; and expressed herself as feeling very well and free from pain. Between the fits the pupils remained very much contracted, but during them they became greatly dilated, and the eyeballs were drawn upwards and inwards. In twelve hours she had eight fits, and as each lasted about ten minutes, there was an average interval of about an hour and twenty minutes between them.

The head was shaved, and a large blister placed over the occiput and down the back of the neck, and a quarter of a grain of extract of elaterium was administered every three hours. She was allowed small quantities of beef-tea and a little wine.

She was soon freely purged by the elaterium, and the fits ceased on the evening of the 31st. On the 1st of June she had quite recovered her consciousness, and from this time she gradually improved, still under the influence of elaterium. The quantity of urine increased considerably, and at the same time free watery discharges were passed five or six times a day from the bowels. The dropsy disappeared, and the heart's action im-

proved in force. On the 29th of June, the elaterium was discontinued altogether.

On the 3d of July the urine diminished in quantity from a quart and six ounces, to a pint and a half, and a slight fit occurred in the night; the elaterium was resumed on the 4th. From this time the urine increased rapidly, and the quantity of albumen in it became much diminished. Although weak, she left the hospital on the 20th, being anxious to return home.

This patient continued pretty well until the beginning of October. On the evening of the 6th of that month, she was brought to the hospital quite insensible, and suffering from a succession of epileptic fits, just of the same nature, and as violent as before, but with such rapidity, that the intervals between them did not allow of anything being given, either food or medicine. They continued throughout the greatest part of the night, and she died early the next morning, not a gleam of consciousness having shown itself.

The examination of the body took place on the following day.

The kidneys were small, contracted, and seemed much wasted at the expense of the cortical substance, the cones in many instances reaching almost the very surface of the organ; in some of the cones there were opaque streaks of deposit of urate of soda, taking the direction of the tubes, and probably occupying the canals of some of them. There was no healthy epithelium in any part of the cortical substance; in some situations the cells were filled with oil, in other places they were opaque. The walls of the minute arteries were thickened and hypertrophied. Each kidney weighed only three ounces and a half.

The heart was very large—it weighed nineteen ounces when deprived of its contained coagula. All its cavities were dilated and hypertrophied. The left ventricle was especially thick and large. The aortic and mitral valves were slightly opaque by interstitial deposit, and the chordæ tendinæ seemed thick and shrunk. In the dilatation of the left ventricle and the shortened chordæ tendineæ there was ample explanation of the imperfect action of the mitral valves. There was no obstruction in the large arteries to explain the great

hypertrophy of the left ventricle; that lesion seemed most probably to be due to the thickened state of the capillary arteries of the kidneys, and the consequently impeded circulation through those organs.

The brain was carefully sliced, and to all appearance it was perfectly healthy; its substance generally was very firm; it was everywhere paler than usual; there was no morbid condition of the membranes. The specific gravity of the gray and white matter was carefully ascertained by Dr. Conway Evans, by the ordinary method for determining the density of a body heavier than distilled water, and insoluble in that fluid. The density of the gray matter of the *convolutions* was found to be 1032·71, and that of the white matter of the *centrum ovale* 1037·17, water being considered as 1000. On microscopic examination, the minute cerebral blood-vessels presented no indication of fatty or earthy degeneration.

The brain was subjected to chemical analysis, with the view of detecting the presence of urea. This was performed in the following manner:—About three fourths of the whole brain (including more or less of each of its segments) was cut up into small pieces, and treated with four successive portions of boiling distilled water, each portion, consisting of about ten ounces, being allowed to stand six or eight hours before the next was added. The brain, while thus macerating, was frequently stirred and mashed about with a glass rod. The washings, after being poured off, were mixed together and filtered. The filtered aqueous extract so obtained was now evaporated to dryness over a water-bath, and the dry residue, after being powdered, was again treated with four successive portions of boiling distilled water, observing the same precautions as before. The washings after being mixed together, as before, were filtered, and the clear solution evaporated to dryness over a water-bath, and after being thoroughly dried in a hot-water oven, the residue obtained in this manner was finely powdered and the powder boiled in five successive portions of ether. The ethereal extract so obtained was evaporated to dryness at a low temperature, and then treated with a little tepid water and allowed to get quite cold. It was then filtered through paper previously moistened with water, and the clear solution again evaporated to dryness at a low tempera-

ture, when a small quantity of the extract procured in this way (which would contain all the urea present in the brain operated upon) was placed on a glass slide, treated with a drop of strong nitric acid, covered with a bit of thin glass, and allowed to stand a little time; and then, examined under the microscope, a few crystals were seen, having all the characters of those of nitrate of urea.*

LECTURE XLIX.

ON PARALYSIS.

ON A CASE OF SYPHILITIC DISEASE OF THE DURA MATER, AND PERIOSTITIS.

I SHALL make to-day, Gentlemen, some remarks upon the case of a patient who, I find, has left the hospital. You have had, however, quite enough opportunity to make yourselves acquainted with all the particulars of it, as he has been some time under our observation, and I have frequently at the bedside pointed out the leading features of his malady. A clinical lecture delivered in the theatre, apart from the patient, has this great advantage, that we can discuss freely all the points of a case, many of which cannot be equally well commented on at the bedside, and some *ought* not to be referred to in the presence of the patient.

This is a case of peculiar importance and interest. It is an example of the way in which secondary syphilis affects the periosteum of different and even important parts, producing serious and possibly fatal, disturbance in their functions. Here you have first primary and then secondary syphilis then periostitis,

* In this, and two other cases of renal epileptic coma, I have sought, in vain, for evidence of the presence of carbonate of ammonia in the expired air and in the blood, as suggested by Frerichs. ('Die Brightsche Nierenkrankheit.')

first of external parts, and subsequently affecting the dura mater; then symptoms of brain disease, both local and distant; and, lastly, when the nature of the disease was detected, rapid and perfect relief.

Case CCXIX.—The patient was John Beglin, a compositor, aged thirty-four (vol. xxvi), admitted June 6th, 1851: the history of the case illustrates very well the ordinary course of many an ill-managed syphilitic case; it is this—that fourteen or fifteen years ago he contracted syphilis, and had a chancre and a bubo; for these he took, without medical advice, a large quantity of mercury, which salivated him freely; he took as much as 210 grains, or ʒiiiss, of blue pill, besides the external application of a great quantity of mercurial ointment. The consequence was, his gums became spongy, his teeth loose, his glands swollen.

After the chancre and bubo had healed, he continued well for seven months, and then an eruption came out over his legs, arms, back, and face, &c. For this he was treated with solution of antimony and nitric acid (he thinks), and in a short time cured. Soon after the disappearance of the eruption, symptoms of iritis came on. His description of the affection of his eyes is a tolerably exact and clear account of an attack of iritis. He says he first perceived a haziness before his eyes; they were very much inflamed, vision was much impaired, and he had belladonna applied. This makes it probable that some adhesion had taken place, or threatened to take place, between the margin of the iris and the anterior surface of the crystalline lens.

From all these evils he seems to have escaped pretty well, and he got married. He appears to have had no return of any unfavorable symptom till five years ago, when he applied as an out-patient to one of the hospitals in the metropolis for contraction and rigidity of the flexor muscles of the right forearm, and numbness in the same region. The nature of this symptom was not recognised; the case was looked upon as surgical, and the affection as local. The surgeon into whose hands he fell first applied leeches, then warm fomentations day and night; then, thinking there was some deep-seated abscess at the root of it, he made several free incisions, but all with no effect: so great was

the contraction that it was in contemplation to divide the tendons. Fortunately, however, this was not done, and the patient left the hospital, after having been in it twelve weeks, just as bad as when he entered it. The contraction seems to have been confined to the flexor muscles of the forearm: the elbow-joint was moved with perfect freedom, the wrist slightly, but the fingers were perfectly stiff. After leaving the hospital, he went from one to another without deriving any benefit. At last, a singular accident happened to him: he met a friend, to whom he related his symptoms, and who told him that he had suffered from an exactly similar attack, and that he had been cured by a certain medicine, of which he would give him the prescription, and which he had no doubt would cure him likewise. He accordingly took it in the prescribed dose, and it *did* cure him. In three weeks he was perfectly well, and not only had the contraction subsided, but the sensibility was restored, and from feeling very ill, and having no appetite, he became quite well and strong, and his appetite recovered. The medicine turned out to be iodide of potassium.

From that time he continued quite well, till six months ago, when he was thrown out of work, and got into that mentally and physically low state which is peculiarly favorable to the development of any disease, the seeds of which may have previously existed in the constitution. In consequence of this he went to Ireland, where he had some friends, and there, in Dublin, about four weeks ago, he had a fit, accompanied and preceded by severe pain in the head. He became alarmed, and returned to London, and shortly afterwards applied to this hospital as an out-patient, where the syphilitic nature of his disease was at once recognised by Mr. Bowman. He intended to continue as an out-patient, but having had a third fit the day after, determined to become an in-patient at once, and was admitted on Saturday, May 5th.

On his admission he appeared in a very low condition: his face was pallid, and he spoke tremulously: he complained principally of pain in his head, situated in the region of the left parietal bone, and also of great pain and tenderness over the head of the fibula of the left leg. The pain in the parietal region was not increased by pressure, but the region of the

head of the fibula was much swollen, and extremely tender to the touch. The pain both in the head and the fibula was severe, and became so much aggravated at night, as to deprive him of his rest,—a symptom bearing distinctly on the diagnosis. The situation of the pain in the leg was marked by a swelling of considerable size, which appeared to implicate not only the periosteum, but the ligaments of the joint and the fascia. At the situation of the pain in the head, there was nothing to be seen or felt; there was no evidence of inflammation affecting the pericranium; but the pain was very defined, both in character and in situation; it was fixed with variable severity in the parietal region, and never left it nor extended beyond it.

In addition to these symptoms, there was a very remarkable state of the *right* forearm—the side, you will observe, opposite to that on which the pain in the head existed. The muscles of the forearm were all in a rigid state, and the fingers forcibly bent into the palm of the hand, the flexor tendons being rendered quite tense by the firm contraction of the muscles. Moreover, there was a good deal of numbness of the forearm, and the rigid state of the muscles both impeded their action and weakened them. There was, in fact, imperfect paralysis both of sensation and motion.

So far, then, we have all the history of the case which can bear upon or be necessary for the diagnosis. I regret that we omitted to make inquiry into one point very interesting in reference to general pathology. He had been married within the last few years; and we ought to have inquired whether his wife had become pregnant, and whether she had gone her full time, and borne healthy or diseased children. That a man still tainted with syphilis will beget syphilitic children without exciting primary symptoms in the woman, is now well known; and the tendency which women impregnated by such men have to miscarriages is also a fact of great interest, especially with reference to the laws of the humoral pathology.

Now the existence of periostitis was quite evident, from the large swelling over the head of the fibula; and the history of the case, the aggravation of the symptoms at night, the influence of iodide of potassium, made this part of the diagnosis easy and certain; but what was the connexion between the

periostitis and the condition of the arm? Was it dependent on some local change affecting the periosteum of the bones of the forearm, and the tendons of the muscles, and perhaps also the nerves; or was it due to some morbid state of a distant part—the brain, for instance? I thought for a moment that the contraction and partial paralysis of the forearm might have been dependent on irritation from periostitis affecting the part itself; but very little reflection convinced me that an affection so extensive, and of such long duration, was not likely to result from a local cause, nor could I call to mind any case in which such symptoms resulted from a cause of that kind. On looking further into the case, I came to the conclusion, that the affection of the arm was caused by syphilitic inflammation of the dura mater—the internal periosteum of the cranial bones—in short, by an intracranial node, which caused pressure on the surface of the cerebral hemisphere, and gave rise to paralysis, with irritation and consequent muscular rigidity.

This diagnosis was founded on the following points, which our investigation of the case enabled us to make out. First, there was paralysis not only of motion, but also of sensation: this denoted that the paralytic state was due to an affection of the *nerves* rather than of the muscles. Secondly, the absence of all evidence of node, or other periosteal affection of the bones of the forearm, rendered a local cause very unlikely. Thirdly, the general history of the case distinctly denoted an affection of a syphilitic nature. And lastly, the fixed and constant position of the pain in the head, situated as it was on the side opposite to that on which the paralytic affection existed, indicated mischief in that situation. The occurrence of epileptic fits after the pain in the head and the affection of the arm had existed for some time, evidently associated that pain with cerebral disturbance.

The train of phenomena in this case was as follows: There was first syphilitic inflammation of the dura mater: this was slow and insidious in its progress, and did not at first cause any great pain: it then produced a certain amount of node; and this, by its pressure upon the surface of the hemisphere, caused irritation and paralysis, accompanied by spasm and rigidity, or an inflammatory state of the arachnoid and pia mater was in-

duced corresponding to the position of the inflamed dura mater, and exciting irritation of the surface of the brain; and, as the pain in the head increased, the paralysis both of sensation and of motion increased. The paralysis, imperfect as it was, and accompanied by rigidity, was just such as would arise from pressure or irritation on the *surface* of the brain.

I think, then, there can be no doubt that in this case we had syphilitic inflammation of the dura mater, exactly analogous to that affecting the periosteum of the fibula. Idiopathic inflammation of the dura mater is extremely rare: indeed, taking the word *idiopathic* in its strict sense, as implying the absence either of constitutional taint or of a poison introduced from without, I should say it never occurred. Besides local injuries, scrofula and syphilis are the usual causes of inflammation of the dura mater: of the scrofulous form we sometimes meet with an example in that produced by otitis spreading from the ear to the cranium, the primary disease being almost always scrofulous in its origin. Indeed, the further we advance in our knowledge of pathology, the more shall we find it necessary to give up ideas of idiopathic inflammations, and the more certainly shall we be able to trace inflammatory affections to some defect in the excretory functions, or to the introduction of some morbific material.

If anything further could be wanted to support this diagnosis, it is supplied by the perfect success of the treatment which was based upon it. From the history Beglin gave us, it appeared that his symptoms on a former occasion yielded to the influence of iodine. On his admission into the hospital he was put upon three grains of iodide of potassium three times a day. The effect was marvellous: almost as soon as it was given to him the symptoms began rapidly to give way, as the following daily report shows: On the 9th—three days after admission—the report is that the patient seems decidedly better, has an appetite, pain easier, looks better, sleeps well.

On the 12th.—Better in every way; is able to use his arm; pain in the head gone, in the leg less; swelling diminished.

17th.—Better in every way; pains in the head and leg quite gone; sleeps, eats, and drinks well.

19th.—Quite well in every respect.

Now, if we had failed in our diagnosis, we might have kept this man for weeks in the hospital without doing him any good, and we might perhaps have sent him away just as he entered, instead of dismissing him cured of his malady, and in a state fit to resume his ordinary avocations. Nothing can give a clearer idea of the importance of a correct diagnosis than the history of a case like this. If the disease had not been arrested, it is probable that more lymph would have been poured out upon the dura mater—the pressure would have been augmented —the fits would have recurred; and the epilepsy, instead of being transitory, would have become confirmed: in fact, it is impossible to say where the effects of the disease would have stopped: whereas, by a careful investigation of the case, a due appreciation of the indications furnished by its history, and the administration of a small quantity of a medicine which may be always safely given within moderate limits, the man is restored to perfect health. If any one were inclined to attach but little importance to correct diagnosis, the instance of such a case is all that is necessary to prove the danger and absurdity of such views. It shows that the great step towards cure is the right understanding of the nature of the disease with which we have to grapple.

Now I ask—What has been the exact state of the dura mater in this case? I think the best way to answer this question will be to give you an account of the history and post-mortem examination of a case that occurred in my hospital-practice some years ago. These cases are generally capable of cure, if detected early; but if allowed to go on too long, new products are formed which permanently damage the brain, and are not susceptible of cure. The case to which I allude is as follows:

Case CXX.—A woman, aged thirty-one (vol. ii, p. 37), was admitted into the hospital some years ago, suffering from the following symptoms:—great loss of memory, fits, imperfect palsy of the left side, and pain in the right parietal region. She was a married woman and had lost her husband, and she attributes her illness to grief from this cause. About this time, which was between five and six years before her admission, the pain in her head began. She led an irregular life, was intem-

perate, and, being of easy virtue, contracted syphilis, and had chancre, sore throat, papular eruption, and nodes on the tibiæ. Two months before her admission she had her first fit; it came on shortly after she had eaten a hearty supper. She became insensible, was much convulsed, especially on the left side, bit her tongue, and after the convulsions had ceased she remained in a drowsy state for twelve hours. After this the pain in her head increased considerably, and was more fixed and defined; it was referred to the right parietal region. The fits likewise recurred frequently; they were always preceded by a sense of painful numbness in the left hand and foot. The *right* pupil was occasionally more dilated than the left, and the right upper eyelid hung lower than the left, as if the third pair of nerves on the right side were slightly affected.

Despite of active treatment, bleeding, local and general, the free exhibition of mercury, and the long issue in the scalp, this woman became more and more comatose, and had a severe fit, from which she never rallied. On examination we found the dura mater firmly adherent to the right parietal bone, and also to the visceral layer of the arachnoid, for an oval space two inches in its long, and an inch and a half in its short, diameter. At this part the dura mater was three or four times its natural thickness. Between the layers of the arachnoid there were two large masses of a yellow colour, like concrete pus, opposite to which were corresponding depressions or concavities on the surface of the cerebral hemispheres. At these points the cerebral substance was slightly softened, and redder than was natural. At the corresponding point on the opposite side of the skull a similar morbid alteration in the dura mater and the arachnoid was commencing. There was no disease in any other part of the brain.

Now this is a good typical case, illustrative of the way in which syphilis may effect the parts within the cranium, and the extent, when not early arrested, to which it may proceed. The dura mater, like any external periosteum, becomes affected by the syphilitic poison; then follows arachnitis; and lastly, cerebral compression, and perhaps red softening. It is not likely that our patient, Beglin, had such extensive disease as we found with this woman, otherwise he would not have recovered

from the paralytic affection so quickly. But I cannot doubt that changes of this kind would have taken place within his cranium if the progress of the disease had not been arrested. If the woman, whose case I have just related, had come into the hospital three or four months before the time she did, and had been put under a course of iodide of potassium, or other suitable remedies, it seems certain that she would have completely recovered.

Let me give you another example of a case of this kind, in which the disease had not gone so far as to be beyond the power of remedies.

Case CCXXI. — The patient was a young man, who followed the business of a chemist and druggist. There was a distinct history of syphilis. He had experienced a good deal of pain in the right parietal region, which had been treated as neuralgia. From this treatment no benefit resulted, and shortly afterwards he began to suffer from imperfect paralysis of the left arm and leg; and I noticed decided thickness of speech, his manner became dull, and his spirits much depressed. The paralysis then became general on the left side, affecting his face, and the scalp became tender over the painful region. I determined to put him under treatment calculated to relieve inflammation of the meninges, and accordingly gave him calomel and opium, and applied tartar emetic ointment to his head. In ten days his paralysis had vanished, and all his other symptoms had left him.

I may refer briefly to another case, which you have frequent opportunities of witnessing, as he is often an inmate of the hospital: his name is Coulson, and he affords a good example of very complete paralysis of the fifth nerve of the left side. I have no doubt that syphilis is at the root of the disease in this instance too. The man is subject to epileptic fits, the immediate exciting cause of which is intemperance. I think the exact diseased condition in this case is syphilitic inflammation of the dura mater in the vicinity of the fifth nerve and the Casserian ganglion; that lymph has been poured out, which presses upon the ganglion, and, including both the sensitive and motor portions of the nerve, suspends alike their respective functions.*

* This patient died many months after this lecture was given, and the

There are one or two more points touching cases of this class, with which I shall conclude what I have to say to you to-day.

And, first, as to the cause of the epileptic fits which occurred in three out of the four cases, and which I can scarcely doubt must have occurred in the fourth case had it been suffered to proceed much further. The occurrence of epileptic fits in such cases as these appears to me to denote that the brain itself is involved in the morbid action, either by extension of irritation from the diseased parts, or by the direct influence of the syphilitic poison; but in all the cases the simple existence of the diseased state of the dura mater did not seem sufficient to cause the fits. The morbid process had been of long continuance in the dura mater before the fits began. They seem to have been brought about by causes operating on the whole system—upon the blood—upon general nutrition—and upon that of the brain in particular, which must have already suffered a good deal by the neighbouring disease of the dura mater.

In some cases of meningeal disease, especially, I think, when the pia mater has been previously affected, we meet with fits of a very remarkable character—I have called them *epileptiform*. One arm, or both arm and leg on one side, become seized with convulsive movements, quite of the clonic or epileptic kind. These come in paroxysms; the paroxysm lasts a variable time, and then subsides, leaving more or less general exhaustion and disposition to sleep; but consciousness is not impaired. Yet there can be no doubt that such fits may pass into the true epileptic fit; for it is not rare to see a very complete epileptic fit commence with some local derangement of sensation or motion, or both.

Secondly, the extraordinary influence of iodide of potassium in controlling syphilitic periostitis is highly deserving of your attention. We hear a great deal about specifics, and many medicines have the name with very slender claims to it; but if there is anything, in addition to quinine, which deserves the name of a specific, it is the iodide of potassium, for syphilitic

arachnoid sac was found completely obliterated by old cellular adhesion all around the fifth nerve. The trunk of the nerve was wasted, and appeared under the microscope to consist solely of wavy fibres of white fibrous tissue.

periostitis. If you have a *pure* case, it acts like a charm, so that the treatment materially aids the diagnosis; for we may fairly set it down, that if the symptoms yield completely and at once to the influence of iodide of potassium, there is strong reason to suspect that the disease is probably syphilitic.*

But, although iodide of potassium unquestionably exerts a wonderful influence over these syphilitic periosteal affections, nothing is more certain than that its effect is far from being permanent. It is very common to meet with cases of periostitis which at first yield readily to the influence of iodide of potassium, and the patient continues well for a time, when all the old symptoms return. It was thus in Beglin's case: some time before his admission into the hospital he took iodide of potassium, and the symptoms yielded; but they afterwards returned, and were again removed under the influence of the same remedy. I think it not at all improbable that he may again have a return of the symptoms, and may once more come into our hands.†

The knowledge of these clinical facts teaches us that we must not speedily abandon the use of the iodide, or of iodine in some other form, in cases of this description. In the present state of our knowledge we can scarcely determine whether the iodine acts by eliminating the syphilitic poison, or as an antidote. Possibly it may act in both ways; it may at once promote the action of emunctories, and so increase the amount of matters excreted from the blood, and unite with the syphilitic poison, forming an innocuous compound, of which, however, the iodine element disappears more quickly than the syphilitic, leaving, after a time, the syphilitic in undisputed sway in the system.

In such cases we must trust to the repeated use of iodine as one element of cure, care being taken to watch the constitution

* In making this statement I believe I do not go too far; although it is not absolutely true that iodide of potassium is only useful in syphilitic periostitis. I have seen cases of periosteal rheumatism and gout affecting the fibrous tissue greatly benefited by it; and I have seen it do marked good in periostitis after fever. At the end of this lecture will be found a case very similar to that of Beglin, of which the apparent origin was an injury, and yet the disease yielded only to the iodide of potassium.

† *Vide* the sequel of the case, p. 851, et seq.

of the patient during its administration. And we may aid the influence of the iodine by the occasional use of mercury, either at the same time with the mercury, or, as I prefer it, alternately —that is, giving first a short course of mercury, then of iodine, then of mercury, and then omitting both, and using only tonic means, both medicinal and hygienic, resuming, if occasion should demand, the mercurial and iodine treatment. And you will also find great benefit from the prolonged use of well-made decoction of sarsaparilla, or of cod's liver oil, or of both.

But you must never lose sight of the fact, that *time* is an important element of cure in these cases; and, therefore, we must be careful not to weaken the powers of our patients by our treatment; lest, by so doing, we should not only retard their favorable progress, but give a stronger hold to the poison. We have seen, in the history of Beglin, how a state of enfeebled nutrition from bad living clearly favoured the development of the serious symptoms. It will be our duty, then, to encourage our patients to expect a favorable issue; to impress upon them the necessity of a steady perseverance in a general plan of treatment, and to point out the dangers of swerving from the hygienic or dietetic rules laid down for them.*

I am fortunately enabled to follow up the further history of this patient Beglin. It was on the 22d of May, 1849, when the preceding lecture was given upon his case. He had then just left the hospital. From that time till June, 1851, he continued pretty well; but he would call every now and then at the hospital, when suffering from pain in the head, which I suspect was often aggravated by intemperate habits and unhappy domestic relations.

Early on the 9th of June, 1851, he was brought into the hospital in violent convulsions, which were confined to the right side, the left being moved only by the action of the right. These lasted till 6 P.M., and in the night he had several short fits. On the 10th there was still some slight convulsive paroxysms, each lasting a minute. He had quite recovered his consciousness, but not his speech, and pointed to the left side of his head

* The lecture, as originally given, ended here.

as the seat of his pain, and protruded the tongue when told to do so. On the 11th he had a very violent fit, in which the body was bent over to the right side, with powerful, clonic spasms. The tongue was bitten on the right side. The urine was ascertained to be quite normal. Towards evening the fits subsided, and he was able to speak a little.

He had a blister applied to the shaven scalp, and two grains of calomel were given every four hours, mercurial ointment and savin ointment were applied to the blistered surface, and he was purged. The fits continued till the evening of the 16th, when he showed evident signs of salivation.

On that day they had been very severe, still affecting the right side. They ceased during the night, and at 7 A.M., on the morning of the 17th, he had one fit, and another at 11 o'clock, A.M. These were the last. Then followed a state of noisy delirium, which gradually subsided, under the use of opium and stimulants, in a week. He now complained chiefly of muscular pains, weakness and numbness of the right arm and leg, and of pain in the head on the left side. Five grains of the iodide of potassium were prescribed three times a day, and counter-irritation of the scalp was kept up opposite the seat of pain. From this time he improved steadily, and left the hospital in his ordinary state of health on the 18th of July.

From this time till the end of the year 1852, Beglin applied at the hospital occasionally, suffering from pains in the head, and in the right arm and leg. I do not know whether he had any fits during that time. There was no doubt that his habits were careless and rather intemperate, and such as would keep up the epileptic tendency. On the 7th of December, 1852, he was seized with severe pain in the left parietal region, having been out of sorts and unable to attend to business for some days previously. During this time he often had recourse to brandy-and-water to rouse him. The pain increased very much, and he suffered from frequent paroxysms of it during the whole of that day, Monday, and on the following day, Tuesday. On the Wednesday morning he was seized with a succession of fits, affecting chiefly the right side; after the eighth of which he sank rapidly. On this occasion he had not been brought to the hospital, nor had any iodide of potassium been administered.

Indeed, from the description given by his sister, it seemed that the fits were so violent, and succeeded each other with so much rapidity, that it was not likely that any ordinary remedy would have controlled them.

This poor man, knowing the interest we took in his case, left instructions that I should be informed of his decease, and permitted to examine his body. This, with the assistance of my friend, Dr. Hyde Salter, was accordingly done.

The body was in a state of good nutrition and was tolerably fat.

On sawing through the skull, the bone in the right temporal region was found much thickened. A small osseous spiculum projected from the inner surface of the left temporal bone.

The dura mater was easily separated from the calvaria. Opposite the thickened bone it was also slightly thickened. It adhered very closely to the arachnoid and pia mater on each side of the falx, and in its whole length. Here the Pacchionian bodies were very large, and adhered so intimately, that in removing the dura mater the pia mater came with it. The arachnoid was quite opaque in this situation. At the posterior and upper part of the left parietal region, and at about an inch from the longitudinal fissure, the dura mater was adherent to the subjacent membranes for about the space of a square inch, and so firmly, that in taking it off, it brought away not only the pia mater, but the cortical substance to a considerable extent.

The gray matter of the convolutions was darker than is natural; this was especially remarkable in the convolutions corresponding to the adherent membranes on the left side, as above described. Here the brain substance seemed slightly hardened. The convolutions generally had a shrunk appearance, and the sulci were wide, and the subarachnoid fluid existed in considerable quantity. On the surface of each hemisphere, just above the upward termination of the fissure of Sylvius, there was a depression, as if one or more convolutions had completely wasted. The pia mater occupied the depression which corresponded in size and position precisely on both sides. Unfortunately it was found impossible to examine the brain microscopically.

All the other viscera were healthy.

The amount of recent disease here was out of proportion to the severity of the symptoms during the last day or two of life; and the evidence of old lesion, on the left side of the brain, was scarcely as much as one might have expected from the violent convulsions and severe pain, were it not that we knew that the patient had been subjected to very active treatment by mercury, iodide of potassium, and counter-irritation a year and a half before. And knowing how completely considerable periosteal thickenings on the shins and elsewhere disappear under similar remedies, it was satisfactory to find no greater marks of disease remaining within the cranium.

It can scarcely be doubted, I think, that had this man been in easy circumstances, and of temperate habits, he might have been completely cured, and that the epileptic attacks would have ceased, or assumed a much milder form.

I am indebted to my friend, Dr. Kennion, of Harrogate, for the particulars of a very interesting case, presenting many points of similarity to that of Beglin, and cured by the exhibition of iodide of potassium, but differing in the absence of any syphilitic taint.

Case CCXXII. — An officer in the Queen's service, while on duty in one of the colonies in September, 1851, received a violent blow from a pistol on the forehead, which stunned him for a short time, but the immediate effects soon passed off. Two days afterwards he was seized with a violent pain in the right temporal bone, which was exceedingly tender to the touch, but was not swollen or red. Being actively engaged in service, he used no remedies; he fell off rapidly in flesh and strength. Four weeks after this he fell from his horse in an epileptic fit, but without suffering any injury by the fall. He was now sent home, and arrived in England in January, 1852. From that time until June he suffered agonies from the pain; he had various returns of the epileptic seizures, and continued to lose flesh. In March, a swelling was perceived for the first time in the right temporal region.

Mercury, purging, leeches, blisters, had all been freely employed in this case without the slightest beneficial effect. At

length it was determined in consultation to suspend all other treatment, and to give large doses of iodide of potassium. At first he took five grains three times a day, and the dose was gradually increased to fifteen grains without producing any unpleasant physiological effect. Within a fortnight after the commencement of the iodine, the pain diminished in severity, and at the end of two months it entirely subsided. He had no return of the fits since August, at the date of Dr. Kennion's communication to me (November 22, 1852).

The influence of the iodide of potassium in these cases is of extreme interest. Periostitis, or, which is the same thing, inflammation of the dura mater, may be excited in strumous, rheumatic, or gouty states of constitution, and more probably in persons tainted with syphilis. The facts, both of clinical history and of treatment, bear upon the pathology and treatment of those cases of epilepsy which follow the syphilitic taint, and point to the iodide, and to mercury and sarsaparilla, as the remedies which ought never to be lost sight of in such cases.*

* I have lately seen with Mr. W. J. Jones, a middle-aged man, of irregular habits, who had primary syphilis some years ago. Shortly before my visit a considerable node formed on the right parietal bone. He suffered very much from pain in the head, and from giddiness, and he had several epileptic fits, which left him very much exhausted. After the first fit he had complete paralysis of the left arm, which lasted only half an hour. I prescribed iodide of potassium in large doses, and he speedily recovered.

In connexion with the subject of this lecture, interesting cases and valuable remarks will be found in Sir Everard Home's paper entitled, "Cases and Observations which show that inflammation is sometimes communicated from the dura mater to the pericranium" (Transactions of a Society, &c., vol. iii, p. 122); in the same volume, there is a striking case by Mr. Wilson, p. 115; reference may also be made to a paper by Sir Philip Crampton, on Periostitis, in the 'Dublin Hospital Reports,' vol. i; and to a valuable Lecture in Dr. Graves' 'Clinical Lectures,' vol. ii; and also an interesting paper by Dr. Reade, of Belfast, in the 'Dublin Medical Journal,' vol. xiii, new series; and a case by Dr. McDowel, in the 'Dublin Hospital Gazette' for April 15, 1854.

LECTURE L.

ON NERVOUS DISEASE.

ON A CASE OF ACUTE IDIOPATHIC TRISMUS.

WITHIN the last day or two, as you are aware, a patient has died within the walls of this hospital of idiopathic tetanus, or, more properly speaking, of that form of it which, from the muscles of the jaws being chiefly affected, is called trismus (τριζω, strido) ; and, as this is comparatively a rare disease, and, in the majority of cases, a fatal one, I am anxious not to let an instance of it pass without making it a source of instruction to you, by giving you some account of its clinical history and pathology.

CASE CCXXIII.—The patient, Henry Franklin, aged forty, was admitted into the Sutherland ward on Tuesday, November 6th, 1849. The following history of him was obtained :— On Sunday, October 28th, he awoke with a swollen face, and some feeling of stiffness about the jaws ; he attributed this to having caught cold, by sleeping with a window open. The feeling of stiffness increased, but he was able to swallow until Sunday, the 4th of November, seven days after the first accession of the symptoms ; but since that time he has been unable to perform the act of deglutition. On his admission (Tuesday, 6th), we found him with the face very much swollen in the region of the parotids, and with considerable fœtor of his breath ; and it was ascertained that he had been taking some pills which contained a small quantity of mercury : it is therefore possible that he may have been one of those persons who are affected by a small quantity of mercury, and that he was in a state of salivation.

We found also great rigidity of the masseter muscles, which prevented him from opening his mouth, and from either speaking or swallowing. His teeth could, at first, be separated just so far as to slip in a card, but no farther. The attempt to separate the jaws caused great pain, as is generally the case, if tension is applied to a muscle in a state of contraction, as in ordinary cramp, and in the treatment of club-foot. It appeared that the muscles of deglutition were also affected, as he stated that the attempt to swallow his saliva nearly choked him.

Upon further inquiry, we learned that his previous life had been temperate and sober; that he had had syphilis and secondary eruption, but no sore-throat or nodes on his tibia or elsewhere; that he had never been subject to fits of any sort, but that he had had brain fever. This was the first day we saw him, and my apprehensions were excited as to the nature of the case: I feared it would turn out what it afterwards proved. But this supposition was opposed by the absence of any manifest cause for tetanus. Excepting the exposure to cold, the slight salivation, and a small abrasion on the nose, occasioned by a fall that occurred the day before the accession of his first symptoms, there was no cause to which tetanus could possibly be assigned. The wound on the nose was very slight, and may be dismissed from the consideration at once. Cold is not an unfrequent cause of tetanus in tropical climates, where the alternations of temperature are great; but it is a rare cause in this climate. I have witnessed one such case, in which the tetanic symptoms followed the exposure of the patient to the open air during the whole of a very cold night. It is very possible that the exposure to the open window, all night, might have excited the disease in the present instance. Might the salivation have caused the tetanus? There are on record some cases of tetanus produced by salivation. But it does not appear from the history of this case, that the salivation was distinctly antecedent to the tetanic symptoms.

There was a certain amount of obscurity as to the origin of the disease, and I was not quite prepared to act at once upon the notion that it was tetanus; I therefore adopted a treatment less decided than I should otherwise have pursued. I ordered him fomentations to the jaws and throat, an enema of starch and

opium at night, and beef-tea enemata throughout the day: in fact, my treatment was very much of the expectant kind. I determined to see what the next day would produce, and contented myself with dealing with the existing symptoms.

The next day we found the symptoms of spasm still the same. The jaw was firmly clenched, and attempts to open the mouth produced great pain. He was still unable to swallow his saliva, which frothed out of his mouth at each expiration. The secretion was certainly unduly abundant. All this time he was perfectly sensible, clear, and composed. He was not at all excited in his manner, and, although he could not speak, he understood all that was said, and wrote down on paper coherent and intelligent answers to all questions that were put to him.

Here, then, we had no symptom but the local affection of trismus, which might have been the effect of the local application of cold; but my fears were that it was but the early stage of a genuine case of lock-jaw, and I stated my opinion to that effect. I therefore determined to try tobacco fomentation, which I have frequently seen used, and with great advantage, care being taken to uphold the strength of the patient. Tobacco has this great disadvantage,—it is a very depressing agent, and therefore tends to throw the patient into that very condition which would favour the fatal tendency of the disease. To avoid this, care should be taken to use the tobacco infusion weak, or the patient may be exhausted to a degree that may prove fatal.

At 7 o'clock of the evening of this day, a symptom supervened which left no doubt as to the nature of the malady: it consisted in a violent paroxysm, exciting to active contraction, not only the muscles of the jaw, but also those of the neck, chest, and back, the last producing partial opisthotonos; the sterno-hyoid, sterno-thyroid, and sterno-mastoid muscles were thrown into a state of rigidity, and this condition implicated the muscles of respiration, and so far interfered with that function, that the patient felt as if he should be choked, and jumped out of bed to endeavour to get air. This choking sensation was not caused by any contraction of the glottis, but was due to the impeded action of the muscles of respiration: in fact, the man was kept in a constant state of imperfect respiration, or rather, of forced expiration, the consequence of which was that the venous system be-

came highly congested, a good illustration of the way in which convulsion may give rise to congestion. The pupils were very much contracted *during the paroxysm*,—a circumstance of great interest, as showing a peculiar state of the nervous centres, connected with the excitation of the spasm; for, as soon as the spasm went off, the contraction of the pupils went off too; and, when the spasm was at its height, the pupils were contracted to pins' points. The pulse during the paroxysms rose to 120.

From this time the case put on a more severe form: not only the permanent contraction of the muscles and all the other conditions continued, but every now and then one of the paroxysms would come on, each succeeding one being more severe and longer than that which preceded it; leaving the patient greatly exhausted, and exciting in our minds the fear that he might be asphyxiated and die in one of them. If, however, there had been reason for such a fear, there was nothing to be done to obviate it; for, even had we performed tracheotomy, it would have availed little, for we should still have left the rigidity of the respiratory muscles unrelieved, which alone would doubtless have sufficed to produce asphyxia. But, in truth, death by exhaustion was the thing to be most dreaded: what we had most to apprehend was that which actually did take place—namely, that the increased frequency and severity of the paroxysms would at length produce a fatal exhaustion, and that the patient would sink rapidly after one of them.

The indication for treatment was therefore to *support;* and since it was impossible to get the patient to swallow (the attempt being immediately followed by an exacerbation of the spasm), the only alternative was, in default of one entrance, to try another. I gave him, therefore, a scruple of sulphate of quinine in a small quantity of water, as an injection, continued the beef-tea enemata, and being anxious, if possible, to diminish the pain and spasm, I ordered the frequent inhalation of chloroform,—the plan being to give it, not in large quantities, but in small and often-repeated doses, with a large admixture of air, so as to produce a gradual and soothing effect. By this means we were able to obtain resolution of the spasmodic condition of the muscles; but to arrest the paroxysms, or diminish their strength when on him, the chloroform seemed almost powerless.

At about a quarter to 12, A.M., on the 8th, the quinine enema was repeated: at this time he was lying in an unconscious state from the chloroform (under the influence of which he was constantly kept), with his muscles relaxed, and breathing tranquilly. About five minutes after 12 a violent spasm came on, the jaws knocked together with a distinctly audible click, the face became much distorted and livid, the eyes rolled upwards, and there was much frothing at the mouth, and great apparent difficulty of breathing, the sterno-mastoids violently contracted, and the head thrown forwards; in a minute or two this was succeeded by a well-marked opisthotonos, the body being much curved, and resting on the occiput and sacrum. In this state he died. His death may be said to have taken place by exhaustion, the immediate cause of which was the violent spasm.

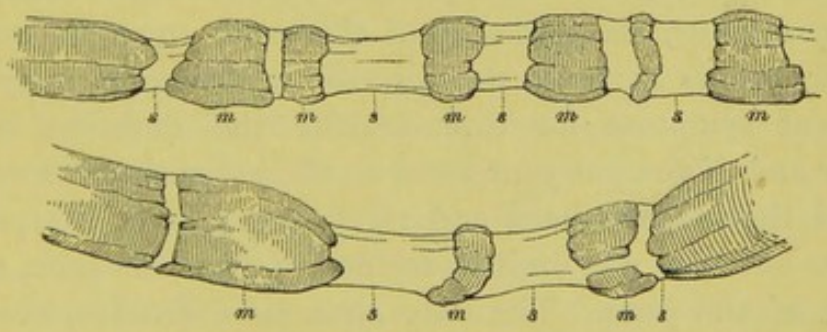
There was nothing in the previous history of this patient to throw any light upon the case; he had been a sober man, had had syphilis and secondary eruption, and an illness which was called brain fever; there was nothing either in the man, or the circumstances in which he had been placed, that could be considered predisposing to tetanus. So it is in all the examples of this disease we meet with; there is no connexion between the previous history of the patient and the disease; the immediate exciting cause, and the detail of the symptoms, constitute the entire history of the case.

It fortunately happened that we were able to have a post-mortem examination, under very favorable circumstances. It was made only four hours after death, when, if there had been any morbid appearances, they would have been free from the fallacy of post-mortem change. There was, however, as I had anticipated, no morbid appearance which seemed to offer any explanation of the disease: the nervous centres, both brain and spinal cord, were perfectly healthy—perhaps the brain contained a little more blood than usual—a fact not to be wondered at when we consider the violence of the paroxysms: the gray matter of the brain was perfectly natural, and its demarcation peculiarly distinct: indeed, this remarkable distinctness may have been morbid, and may have indicated an undue functional activity of these parts.

We examined the state of the masseter muscles, to ascertain

if any change had taken place in their minute structure, in consequence of the prolonged spasm which they had undergone. The result has been confirmatory of certain doctrines first laid down by Mr. Bowman, in illustration of the way in which the contraction of muscle takes place. When a muscle is thrown into a state of contraction, that contraction does not affect the whole mass of the muscle, or the whole of any of its fibres, at the same moment, but certain points or nodes are contracted, while the intervening portions are stretched by the action of the contracted ones. Thus you have a succession of contracted points throughout the entire length of each fibre of the muscle. Here is a drawing made by Dr. Hyde Salter, of a magnified view of two fibres, taken from the masseter of the patient, showing the peculiar condition, which was first described by Mr. Bowman, as existing in the muscles of persons who have died of tetanus, and which, as you will see, is easily explained by the view of muscular contraction to which I have just referred. You must not imagine that all the fibres were so affected: these were the two most marked specimens among many hundreds that were examined, some possessing the appearance in a slight degree, some not at all. Here you see the sarcolemma continuous throughout its entire length:

Two Fibres of the Masseter Muscle ruptured by the Tetanic Spasm.



m m m separated masses of sarcous particles; *s s s* sarcolemma.

the contained sarcous matter, however, is *not* continuous, but is broken up into isolated portions of various sizes, giving rise to a peculiar bulged condition of the sarcolemma, with collapsed constricted portions in the interval. Now, how is this appearance produced? The explanation appears to me to be as fol-

lows: each of the separated masses was the centre of a contraction at the time, when the muscle was in a state of violent spasm; the force of the contraction exercised at these points was so great, that the tenacity of the intervening stretched portions was not sufficient to resist the divellent force: they gave way, and the particles thus isolated when in a state of contraction, having no antagonistic force to restore them when the contracting force had subsided, remain, as you see them, in a state of permanent contraction, leaving intervals of considerable length between them.

These, gentlemen, are the main points to which I would call your attention in the history of this case: I shall now make a few observations of which this history is suggestive.

First, with regard to the name: what shall we call it? Two names are in frequent use for such affections—*tetanus* and *trismus*. I prefer *trismus*, as being more specific, localizing the affection, and showing its limitation to the muscles of the jaw. Tetanus is a generic name, and indicates the general existence of tonic spasm, without any indication as to its whereabouts. Tetanus can hardly be called a rare disease: trismus occurring in the adult *is a rare* disease. In the present case the affection was for eight days confined to the muscles closing the jaw; only within a day of his death did it extend to the muscles of the neck and back; and not till the very last stage did it so affect the muscles of the back as to produce opisthotonos: whereas, in ordinary tetanus, the extension of the spasm to all the muscles generally occurs early in the disease; opisthotonos may be a prominent symptom for three-fourths the duration of the attack; and, if you put your hand on the patient's arms or legs, you find the muscles hard and rigid; whereas, in the present case, the affection was limited, for nearly the whole time, to the muscles of the jaws. The only *essential* difference, however, between the two diseases is in the extent of the nervous centres, and, therefore, of the muscles implicated.

Sir Benjamin Brodie and others have adopted an excellent practical distinction between *acute* and *chronic* tetanus—the one running a short course, with strongly-marked symptoms, and terminating always fatally; the other of longer duration and of a milder form, and frequently recovered from. Traumatic tetanus

is very apt to be acute; but tetanus, however caused, frequently puts on the chronic form. Some of these cases last twenty days or more; and, when prolonged in this way, there is great hope of ultimate recovery. If such a case—or still more, if two or three such—should happen to fall into the hands of some very zealous supporter of any particular practice—if he is a great bleeder, or a free giver of opium—the treatment is immediately set down as specific, and the recoveries are appealed to in proof of its infallibility, the real secret of the successful issue being, that the cases were chronic and not acute. Now, in the case of our patient, it cannot be said that any part of the treatment was likely to weaken the powers of life: possibly the means adopted for their maintenance and prolongation might have been more energetically plied; the quinine might have been earlier administered, and perhaps more support might have been given; still nothing was done that could by any possibility have accelerated the fatal termination of his disease. Yet we see him admitted into the hospital on the Tuesday with slight symptoms of the disease, and dead on the Thursday morning. And this is an essential part of the natural history of all these acute cases—speedy and fatal termination, death taking place by exhaustion.

The peculiar points in this case, that especially call for remark, are the violence of the spasm, and its restriction for so long a time to the jaws. As I said just now, genuine trismus is rare in adults. In newly-born infants, however, trismus is not rare, particularly in hot climates: in the West Indies it used to be very common: it goes by the name of *trismus nascentium:* and though now, from greater care in food, cleanliness, and ventilation, it is much less frequent than formerly, it still carries off a great number of children.

I have said that this case illustrates the mode of death by exhaustion: this is an important point, and one I particularly wish to impress upon you. Death does not take place by asphyxia, nor by any particular effect on any especial organ, but from general exhaustion, induced by the protracted and unwonted nervous and muscular excitement. This, I think, is not sufficiently attended to in the treatment both of this and of many other diseases.

The case also illustrates the little hope we have of obtaining, by post-mortem examination, any other than negative information as regards the particular state of the nervous system; we can only draw our conclusions respecting the pathology of the disease from our knowledge of the physiology of the parts concerned. Now, reasoning on this principle, it may be laid down that the phenomena result from an exalted polarity of the centres supplying the parts affected. In the case of traumatic tetanus, the exaltation of the polar state commences in the afferent nerves of the seat of the wound: if the tetanus arise from cold, the exalted polarity commences in the nerves of common sensation distributed to the exposed part: from the periphery thus irritated the condition is propagated through the nerves to the centres, and the effects on the muscular system show to what portions of the nervous centres the exaltation of the polar force is communicated. This, however, does not afford an adequate explanation of the production of tetanus; for peripheral nerves, and even nervous centres, are often subjected to great irritation without giving rise to tetanus; and it is well known that it is impossible, even by severe mutilations, to produce tetanus in the lower animals: whereas a slight accidental injury (as when a horse picks up a nail) will often excite the disease in its worst form. It would seem that some peculiar state of the system—probably some peculiar condition of the blood—is a necessary precursor of this malady. Hence, no doubt, its greater frequency in warm and unhealthy climates, in over-crowded and badly-ventilated military hospitals, and among ill-housed, ill-clad, and ill-fed infants.

That tetanus may be produced through the blood is shown by the results of the administration of strychnine, which imitate the tetanic symptoms in a very striking manner: so that you may at will develope the general phenomena of tetanus in an animal by giving him strychnine, or injecting it into his blood, but you cannot cause it by external injuries.*

* For further remarks on this subject, see my Lumleian Lectures for 1849, on the Pathology of Convulsive Diseases. The present lecture was delivered in 1849, and printed in the 'London Medical Gazette' for November, 1849.

The diagnosis of the tetanoid condition produced by strychnine from the genuine tetanus, whether idiopathic or traumatic, has become a matter of

What was the exact portion of the centres affected in the present instance? The localization is pretty clear; it was evi-

medico-legal importance since the recent highly interesting trial of W. Palmer, of Rugeley, for the murder of J. P. Cook, through the agency of this powerful poison (June, 1856).

For the defence it was suggested that the tetanic convulsions under which Cook laboured for a short time on the Monday night, and more severely on the following Tuesday night, when he died, might have been due to idiopathic tetanus, or to traumatic tetanus, caused by sore throat. The tetanoid state from strychnine is only likely to be confounded with very acute tetanus, either traumatic or idiopathic. The existence of a wound would of course be some evidence in favour of tetanus; but, after all, the chief reliance must be placed on the symptoms. These are—

1. As regards the tetanoid state from strychnine. *a.* The *rapid* supervention of tetanoid convulsions affecting chiefly and with most intensity the muscles of the trunk and spine, causing an active and violent opisthotonos rarely met with in true tetanus. *b.* A rigid and tetanoid state of the muscles of the lower extremities, with somewhat less intensity than those of the trunk: the limbs are extended, and the feet drawn powerfully inwards by the action of the tibiales postici muscles. The upper extremities are affected also, but in a less degree, and the hands are generally semiflexed. *c.* The trismus or closure of the jaws exists only imperfectly, and the *facies tetanica* very slightly, or not at all: swallowing is perfect, but the mode of deglutition seems to be peculiar, the patient snapping at the liquid offered, and gulping it down with an effort, in a manner very similar to that in which hydrophobic patients swallow. *d.* The attacks of opisthotonos are very frequent, and seemingly exquisitely painful, and are ushered in by a cry more or less loud.

As regards tetanus—

a. The symptoms come on *gradually*, and the trismus is the earliest, the most prominent, and most important one. *b.* The facies tetanica is a very characteristic symptom. *c.* The attacks of opisthotonos are less frequent, less extensive, and less severe than in poisoning by strychnine. *d.* The extremities are the last parts affected, and suffer much less from the tonic spasms than other parts. *e.* Deglutition is slow and difficult, and sometimes impossible, owing to the spasmodic closure of the mouth.

In fine, trismus is the characteristic and primary feature of true tetanus; acute, violent, and frequent attacks of opisthotonos, when not preceded by trismus, and when ushered in by a cry, consciousness being retained, are strongly indicative of poisoning by strychnine.

In the case of Cook, the diagnosis of strychnine poisoning was rendered certain by the occurrence of two distinct attacks of convulsions with opisthotonos, each preceded by the administration of suspicious pills, with an interval of *complete suspension* for twenty-four hours. Such a complete remission of symptoms never occurs in tetanus; nor could the convulsive phenomena be referred to epilepsy, apoplexy, or any of the forms of hysteria.

dently the upper part of the spinal cord, the medulla oblongata, and its upward prolongation in the cranium, especially in the neighbourhood of the implantation of the fifth nerve. Indeed, the supposition that the disease was caused by the salivation would afford a very plausible explanation of the localization of the morbid action in this latter region; for in ptyalism the seat of the peripheral irritation must necessarily be the sentient filaments of the fifth nerve, which would, of course, propagate the irritation to the centre, and excite a similar state of the motor nerves, thereby giving rise to the spasmodic condition of the muscles of mastication, which are supplied by the same nerve.

And we may ask, further, what is the nature of the morbid process set up in these centres? To this it may be confidently replied, as the result of repeated examinations, that it is not inflammation, nor anything allied thereto. And it may, with quite as much certainty, be affirmed that it is a state identical with that which strychnine is capable of producing. Now, this does not reveal to the most careful observation any appreciable departure from the normal state. I have repeatedly examined with the microscope the spinal cords of animals killed by strychnine, and have never been able to detect, even with the highest powers, the slightest change which I could regard as morbid.

The condition of the nervous centres in tetanus is, as it seems to me, best expressed by the term *exalted polarity*,—a condition which gives rise to the undue development of the nervous force. A peculiarity of this state is, that whilst the polar force of the nervous centre is at all times exalted, it is liable to frequent fits of augmentation or exacerbation, during which more extensive and powerful effects are produced. In this way we explain the apparent subsidence of the affection of the spinal cord for longer or shorter intervals, and the subsequent excitation of it in severe paroxysms.

If another similar case were to present itself, what treatment should we adopt? I do not know that I should pursue any different plan from that which I have described to you in the case of our patient Franklin. I might, perhaps, give support earlier than I did in the present case. I should give quinine

freely by the mouth or rectum; if there were much difficulty of deglutition, I should throw up beef-tea enemata, and I should administer chloroform carefully. The administration of chloroform is objectionable on the ground that it is a depressing agent: now, in tetanus, you ought to husband and promote the strength of your patient as much as possible; the disease itself depresses more than enough; and an important aim in your treatment should be to enable your patient to sustain the reiterated shocks of the spasms: if, then, you give chloroform, take care at the same time to support him well, and be not afraid of giving stimulants—such as wine or brandy—if you think he can digest them, and especially if the difficulty of swallowing prevents the patient from taking more than a small quantity of nutriment at a time. The cases which I have known succeed the best have all been treated with close attention to upholding the powers of the patient. If you bear in mind that the great danger is of death from exhaustion, and act accordingly, you will not go wrong in this particular. There can be no use in bloodletting in cases of this kind, as frequently practised formerly, nor in active purgation; the bowels, however, should be evacuated daily, or on alternate days, but more than this is likely to be prejudicial. Neither is there any good to be derived from giving those excessive doses of opium, that have been used in so many cases; for, besides the negative evidence of the usefulness of this practice, there is this objection to it—that opium given in large doses tends to call forth that very exaltation of the polarity of the nervous system which we desire to combat. This is abundantly proved by experiments on cold-blooded animals. You may throw a frog into regular tetanus by opium; and, although actual tetanus cannot be produced by opium in warm-blooded animals, many circumstances tend to show that it may give rise to a state similar to, though much less in degree than, that which it can create in reptiles.

Are we to expect any good from hydrocyanic acid? I think not; and my objections to it are much the same as those against opium,—viz., that it tends to exalt, and not to lower, the polarity of the nervous centres. Those of you, who have seen the death of an animal from hydrocyanic acid, will at once

appreciate this objection. You will remember the violent epilepsy—the tetanic epilepsy, if I may call it so—the combination of tonic and clonic spasm, under which the animal suffers.

The use of galvanism has been proposed in cases of this description for the purpose of depolarizing the nervous centres, and has actually been tried by Matteucci. The long-continued passage of a current of galvanism tends to weaken and to paralyse a nerve or nervous centre, if the direction of the current be the same as that of the nervous force. In the case related by Matteucci, the spasmodic condition of the muscles completely subsided during the passage of the electric current.

Our patient, Franklin, was treated, in addition to the other means, by the application of cold over the region of the spine. Cold has a powerful effect in depolarizing the spinal cord. I have tried it with great advantage in this hospital in tetanus, in laryngismus, and in the convulsions of children. The best plan for its employment is to fill a bladder with some broken ice, and apply it directly to the spine; the ox gullets, from their length and cylindrical form, answer best; an intense degree of cold may be thus obtained in a very short time, and its effect on the circulation will soon be manifest, since cold has a very powerful influence in diminishing both the force and frequency of the heart's action; for this reason you must not apply it for too long a time, or over too great an extent of surface; you must watch your patient, and remove and re-apply it, as his condition shall indicate. In the cases in which I have tried it, I have found it manifestly beneficial in diminishing the intensity of the spasms.

With regard to the internal remedies, I know of nothing better than small doses of opium, belladonna, and conium. The two latter drugs certainly seem to me to have a depolarizing power, as is indicated by their influence, especially that of belladonna, in causing dilatation of the pupil. Still, a good depolarizing agent, which does not tend at the same time to weaken or depress the powers of the patient, is a great desideratum, and when discovered will no doubt prove an invaluable remedy in the treatment of tetanus. What, however, in the absence of such a remedy, the physician has principally to trust to is

support, in order that he may gain time to enable his patient to weather the storm, to sustain the attacks of the disease, until the source of irritation shall have ceased to exist, and its consequences have abated.

I must not conclude without calling your attention to the great importance of isolating tetanic patients, and protecting them, not only from the excitement caused by many persons surrounding the bed, but also from the influence of draughts or currents of air, or external mechanical stimuli, which are peculiarly prone to provoke the tetanic spasm. These patients ought always, when possible, to be put into a separate ward, and visitors should be excluded. The temperature of the room should be kept at a moderate degree, and care should be taken to exclude sound, and also irregular currents of air and light, as far as it can be conveniently done.

LECTURE LI.

ON NERVOUS DISEASES.

ON CASES OF CHOREA.

I MUST refer you to-day to several cases of chorea which have lately been in the hospital, to illustrate the remarks which I propose to make on the clinical history, pathology, and treatment of that disease.

CASE CCXXIV.—The first case is that of a boy named May, in Rose ward (vol. xxxiii, p. 187), which affords an interesting example of the connexion of this malady with rheumatic fever. The history is as follows :—The patient is nine years of age; he is a thin, but otherwise healthy-looking boy; his friends are all quite healthy, and none of the family have ever been similarly affected.

We find that he had enjoyed very good health until ten weeks ago, when he was attacked with rheumatic fever; his ankles were first affected with the rheumatic swellings, and soon afterwards the knees, elbows, hands, and wrists. All these joints became very painful and much swollen; he also suffered from profuse sweats, and was affected with a sense of weight and oppression about the chest. Under the treatment to which he was subjected (and this was by no means severe) he recovered; but for some time after the disappearance of the symptoms, the rheumatic state seemed to linger about him, until he began to exhibit signs of chorea, two weeks before his admission into the hospital.

The choreic symptoms seem to have come on in a rapid, almost sudden manner. The boy went to bed as well as usual, but in the morning, when his mother went to give him his breakfast, she was surprised to find that he could not hold his cup, and that he was quite helpless—indeed, much in the same state as he is at the present time, except that the symptoms were more limited to one side. He had lost the power of directing his movements properly; the motions of his limbs were exaggerated and ungovernable, and if he attempted to take hold of anything, his arm appeared to be violently jerked, in the right direction, perhaps, but usually beyond the object of his search, as if by some power over which he had no control.

Among the early symptoms which manifested themselves in this way was difficulty of deglutition, which came on and continued for some days prior to the more common and characteristic symptoms. The dysphagia was due partly to want of full controlling power over the tongue, and partly to a want of due harmony in the action of the pharyngeal muscles. This symptom is peculiarly interesting, from the marked connexion which subsists between this malady and emotional excitement; and it deserves your special attention in connexion with the pathology of this disease. Previous to his admission the boy could not walk or help himself in any way, and he was obliged to be washed and dressed by others.

On admission, he was unable to stand for any time without being supported; and if he attempted to walk or run, his limbs would be forcibly jerked about in various directions,

and he would fall if not upheld. When in bed, his arms and legs would be in constant restless motion, being thrust first to one side and then to the other, with an irregular jactitating movement.

His articulation was very imperfect, and if he attempted to speak he could only utter a few scarcely intelligible words in a whisper. The dysphagia, although much better, was still present.

When he was told to put out his tongue, it was protruded with the sudden thrust so characteristic of chorea, a feature of the disease, to which you will remember I have often called your attention, as one of its most constant and characteristic symptoms. The whole organ is forcibly and, generally, rapidly protruded from the mouth, appearing as if thrust between the teeth, and is retracted sometimes in an equally sudden manner, sometimes more slowly, and as if supported and guided by the teeth. There is also an awkwardness and an exaggerated character in all the movements, which extend even to the muscles of the face. These phenomena are due to a want of power of duly co-ordinating the various voluntary movements of the body, and doubtless depend upon some affection of that portion of the nervous centres in which the co-ordinating faculty is situated.

Another character of these irregular movements, which, however, does not exist in this case, is, that they are more evident on one side of the body than on the other; you will find this feature evident in some of the other cases which I shall presently relate to you. Chorea is very much a one-sided disease; that is, it affects one side more than the other; sometimes one side only is affected; hence it may be classed with cerebral affections, in which the morbid state of one side of the brain will extend to the opposite side of the body. It seldom happens, even although the irregular movements may exist on both sides, that we cannot detect more movement on one side than the other. The present case, however, seems to be an exception,—it is one of general chorea, and all the limbs seem equally disturbed in their movements.

What is especially interesting in the boy May, is the occurrence of the disease as a sequence to rheumatic fever; the case, too, affords a good example of the heart disease which is so

often associated with chorea, and which, in this instance, is clearly dependent upon the rheumatic attack. Upon listening to the chest, a bellows-sound was readily detected, which was most distinct at the apex, and became less so as you receded towards the base of the heart; this bellows-sound being synchronous with the systole, and occupying the situation which it did, must have depended upon such imperfection of the mitral valve as allowed of regurgation of the blood into the auricle at each contraction of the ventricle; it was also audible at the back, below the left scapula. The imperfection was probably the result of an endocarditis, which had formerly affected the mitral valve and the chordæ tendineæ, and created more or less of shrinking of the curtains of the valve and contraction of the cords, so as to prevent the perfect apposition of their margins, and the complete closure of the auriculo-ventricular opening.

This boy was taken ill with symptoms of chorea a fortnight before the date of his admission into the hospital, May the 8th. The treatment to which he has been subjected is sufficiently simple; it consisted merely of splashing with cold water every morning, and, at the same time, feeding him well. The splashing is effected by throwing one or more pailfuls of water over the patient, while he is standing in a large tub. This is a plan of treatment which we have followed with benefit in several cases in the hospital within the last few months; the effect of it in this case has been, that the boy, in about eighteen days, improved much in general nutrition, the irregular movements have diminished, and, at the present time, he is able to walk without assistance,—a result not more favorable than that which attends its adoption in most cases. The bellows-sound remains, and probably will remain during the boy's life; for it depends upon organic imperfection of the valves, and must necessarily be present as long as that imperfection exists.

Another point in the history of this boy, to which I have not yet referred, is the tendency in his family to rheumatic complaints; for we find that his elder brother, aged fourteen, also had an attack of rheumatic fever about a year ago.

A day or two before the symptoms appeared, this boy was much frightened by his sister, who had covered herself with a

white sheet, and appeared before him unexpectedly, while he was in bed. There is here, then, that which we so frequently—indeed, I might say, so constantly—observe, namely, the connexion of sudden fright with the origin of these cases. In this case the fright preceded the symptoms by a day or two; but, in some other of the cases to which I shall have to direct your attention, a much longer interval occurred between the fright and the manifestation of the symptoms. Although a certain diathesis seems to be always present in cases of chorea, the disease seldom occurs without some sudden emotional excitement, such as fright.

Case CCXXV.—The next case is that of Sophia Jackson, aged fourteen, in Lonsdale ward (vol. xxxiii, p. 89); she is one of two sisters, both of whom have suffered from chorea. She had always enjoyed good health, with the exception of the occurrence of an attack of rheumatic fever, when she was two and a half years old. About a week before her admission, it was noticed that her hands became fidgety, and this was especially the case with the right hand. In this case, the one-sided character of the disease is well illustrated, for the irregular movements are almost confined to the right half of the body.

Here, also, we have fright in the history; we find that, about three weeks before the appearance of the symptoms, she was met and accosted by a drunken man, and was very much alarmed at the time; but you will observe that, in this case, so long an interval as three weeks elapsed between the occurrence of the fright and the manifestation of the characteristic symptoms. Although this interval seems very long, I think we may fairly refer the excitement of the disease to this cause; in many instances, indeed, even longer periods have elapsed between the fright and the accession of the malady; and it is very rare indeed to meet with a case in which you may not find, on investigating the history, that there has been a fright from some cause or another. I have known it occur six weeks before the chorea manifested itself.

The mobility of the right upper extremity continued to increase in our patient, and soon the power of walking became

obviously impaired. The peculiar thrust of the tongue was also developed; and, upon examining the heart soon after her admission, we detected a bellows-sound over the base of the organ, and in the course of the large arteries. This was most probably due to the anæmic state of the patient, and, therefore, in this point differed materially from the bellows-sound present in the first case; this anæmic murmur disappeared as soon as she recovered. In this case we noticed a feature of not unfrequent occurrence, namely, that the urine was of high specific gravity, and generally contained a deposit of pale lithates, and an excess of urea was found in it several times. The high density of the urine was most marked when the choreic movements were most active, and it might, in part at least, be attributed to the increased waste of tissue consequent on the disturbed state of the muscles and nerves.

This patient was ordered an affusion of cold water every morning, and was kept upon a good, nutritious diet. The urine was carefully examined from day to day by Mr. Liddon; its specific gravity never fell below 1019, and frequently reached 1030, and once was found to be as high as 1035. As the patient improved in health, the urine fell in specific gravity, but was never found below 1019. Urate of ammonia was nearly always present, and oxalate of lime was frequently found mixed with it.

This patient was admitted on the 6th of March, and discharged quite well on the 12th of April.

Case CCXXVI.—The next case is of a much more serious character than that which I have just related. Mary Jackson (sister to the last patient), aged twelve (vol. xxxi, p. 160), was admitted on the 29th of November, 1850, and was a long time in the hospital before she recovered. Her parents and the other members of her family are healthy, with the exception of the sister, to whose case I have just now referred.

The history of this case, as taken by Mr. W. Brown, is as follows:—Nine weeks before her admission she was frightened, as her sister had been, by a drunken man; a few days after this she felt pain in the right arm and leg, and experienced a tingling in the fingers, which became restless and fidgety. Soon after,

the left extremities became similarly affected; then came twitchings in the face; and, in the course of a fortnight from the time of the fright, the irregular movements became general, she lost the power of standing or walking, and her articulation was almost completely destroyed. She was in this state when admitted into the hospital. The jactitating movements were extremely well marked; and you will remember that I often called your attention at the bedside to the striking contrast which the irregular jerking clonic convulsions of the muscles exhibited, when compared with the tonic spasms of tetanus. As is always the case in the more aggravated instances of this disease, any attempt to restrain the movements, as by forcibly holding down the legs or arms, invariably tended to their aggravation, and excited them very much in some other part of the body which had not been subjected to restraint. From the jerking movement of the muscles, the pulse could not be distinctly felt. The heart's action was extremely rapid—136, but varied as to rate of movement; and there was a decided mitral bellows-sound.

It is of some interest to notice, that this girl was the second in the same family who was affected with chorea. The two children are of very similar make and habit; their nervous systems are alike; and, probably, their power of generating the nervous force is similar. It is thus that chorea often attacks members of the same family who are of like build and constitution, and have been equally exposed to emotional excitement. But it cannot be said that chorea is hereditary, *i. e.*, handed down from parent to child.

The treatment pursued in this case was just that which had been adopted in the others, namely, at first the splashing with cold water night and morning, and the subsequent administration of quinine and iron, with occasional aperients. The effects of this treatment were soon manifest. It was commenced on the 30th of November; on the 3d of December the movements had become much less, and the heart's action was greatly reduced in frequency; on the 6th she was so much quieter that the pulse could be counted at the wrist, and it numbered 74; on the 8th, she was ordered a grain of quinine thrice a day; and, on the 14th, citrate of iron was substituted for this in the

dose of three grains thrice a day; on the 17th, she began to walk with the assistance of the nurse; and, on the 20th, she was able to walk without assistance. She now began to use her hands, and could grasp feebly, but not so well with the right as with the left. She gradually but slowly improved under the use of steel, with occasional purgatives, good diet, and cold splashing; but it was not until after the expiration of the long period of upwards of three months from her admission, and of five months from the invasion of the disease, that she was discharged quite cured.

During the first three weeks of the treatment of this case, the specific gravity of the urine was very high—1040, 1037, 1030; it afterwards fell to 1020. The mitral bellows-sound had not disappeared when she left the hospital.

Case CCXXVII.—The next case is that of Emma Skinner, aged ten, who was admitted April 12th (vol. xxxii, p. 122); she is about ten years of age, the period of life at which chorea is most apt to occur. Chorea is really a disease of childhood; and although we sometimes see symptoms somewhat resembling those of chorea at the adult period, and at more advanced ages, such cases are clearly exceptional; and it may be questioned whether they are due to exactly the same morbid condition as that which gives rise to the ordinary choreic convulsions of early life. Chorea generally occurs between the ages of nine and fifteen; but, as I have just stated, we occasionally meet with it in patients a few years younger or older, and sometimes symptoms of a similar kind occur in persons of advanced age.

This case illustrates a point which you should bear in mind in reference to chorea, and many other diseases of the same class—I mean, the great disposition of the malady to recur. A child who has had one attack of chorea is always liable to the occurrence of a second at some future period. The same tendency is met with in all paroxysmal diseases. It appears that the patient in this case has been on two former occasions in this hospital for chorea. The present attack came on about three weeks ago, when she was recovering from scarlet fever, at which time she was no doubt in a state of very enfeebled nutrition, a condition peculiarly favorable to the manifestation of

chorea. There is no evidence that any distinct fright occurred in this particular instance; in the every-day life of most children, and especially of those of the lower classes, there are numerous causes of mental emotion, which, although trifling and transient, are amply sufficient to excite nervous disturbance in a weak child. Much the same symptoms were present in this as in the other cases; the choreic movements were strictly limited to the left side, and the case afforded a striking illustration of the one-sided character which the disease is so apt to assume, the right side being wholly free.

The urine also was of very high specific gravity, and frequently deposited pale lithates in considerable quantity. This deposit of lithates is, as I have mentioned, very common, and likewise of grains of lithic acid, which are often as numerous and as large as we see them in adults of gouty or rheumatic habit. Crystals of lithic acid also frequently present themselves on a microscopical examination of the urine, among which are often found crystals of oxalate of lime in great numbers. Generally speaking, the density of the urine is highest in those cases in which the movements are most general and most active; and it falls steadily with their diminution and with the restoration of a greater controlling power on the part of the patient.

You cannot fail to notice, that the general character of the urine in chorea bears a marked resemblance to that in rheumatism. This and other circumstances have led me to associate the state of constitution, in which chorea is apt to occur, with the rheumatic diathesis. The proneness of patients of rheumatic constitution to attacks of chorea, the tendency to a repetition of the attacks in both diseases, the frequency in chorea of endocardial affections, so common in rheumatic states, all point to this affinity between the two disorders.

The four cases to which I have now directed your attention give you a good view of the clinical history of chorea. What is the nature of this malady?

It is easier to say what chorea is not, than to describe what its essential nature is. We may regard it as a disease dependent on a debilitated state of the system, which does not in any way arise from an inflammatory or hyperæmic state of any part of the great nervous centres, or of other organs. Indeed, it is

impossible to fix upon any particular organ of the body, in which anything like structural lesion exists as a constant feature, in cases of chorea. The disease is one of functional disturbance, rather than of organic change; and this is borne out by the results of post-mortem examinations; for almost without exception we fail to detect, in those cases of chorea which terminate fatally, any morbid alteration which, physiologically, could give rise to the phenomena; and in the vast majority of cases, we find all the viscera in a perfectly healthy condition, at least so far as we are enabled to make out with the means at present at our command.

The structures which are obviously affected in chorea are the nerves and muscles. Doubtless a morbid state of both exists; but it seems most probable that the disturbed state of the muscles is excited and maintained by a deranged state of the nerves and nervous centres. If I were to refer to any particular part of the brain, as more particularly the seat of that disturbance which gives rise to the development of the peculiar phenomena of chorea, it would be to that which may be regarded as the centre of emotion. The remarkable frequency with which the attack of chorea is traceable to fright as its cause points clearly to this part of the brain (which has the most extensive connexions with, and influence over, other parts of the nervous system) as the *primum movens* in the production of choreic convulsions. The chain of phenomena would then be as follows: first, a peculiar diathesis; then, a more or less enfeebled nutrition; thirdly, a strong mental impression, which disturbs the centre of emotion, and, through it, deranges the action of more or less of the nervous system, and of a corresponding portion of the muscular system.

And now as to the treatment. You will readily admit that it is not a little important to have it impressed on the mind of the practitioner, *in limine*, that he has not to combat any inflammatory, or hypersthenic, or disorganizing process. He has simply to improve the condition of the blood, to amend general nutrition, to calm the nerves, and to infuse tone into the muscles.

Of course, if any source of irritation exist it must be removed, such as a deranged state of bowels or worms in the intestinal canal. These causes of irritation, however, occur much less

frequently than is generally supposed. Nevertheless, they deserve early and close attention; but a system of purgation carried on too long may prove highly pernicious. If worms be present, let them be expelled if possible, whether they be regarded as cause or simply as concomitant. The most common of these are the ascarides, which have their habitat in the rectum, and which may be expelled by enemata of a strong solution of salt in water or in an infusion of quassia.

Attention to the fulfilment of these indications is all that is essential to the cure of the vast majority of the cases of chorea which come before us in practice. But you will find great advantage, in most instances, from the free use of cold water by affusion to the surface of the body. I have treated many cases most successfully, simply by attention to diet, and by cold affusion, practised once, twice, or thrice a day, and without the use of drugs of any kind. You will remember, that this practice was followed very speedily by marked benefit in the third of the cases which I have detailed to you to-day. The girl had been under tonic treatment for two months prior to her admission, with but little benefit; but on the fourth day of the use of the cold affusion there was a very marked improvement in both the frequency and the violence of the movements; and it was evident that the affusion exercised a decidedly calmative influence upon the nervous system.

Sometimes, however, you will find yourselves balked in the use of this valuable curative agent, in the case of an irritable, or excitable, or ill-tempered child, or of a nervous, fidgety mother, or a prejudiced nurse. When you have to contend with these difficulties, you must be guided by circumstances, either in giving up the resource, or in gradually leading the patient to the full use of it.

I wish to impress upon you that it is to such hygienic treatment as I have described that we must look *mainly* for the cure of chorea. But there can be no doubt that much good may be obtained from the simultaneous and careful employment of drugs of the tonic class. Of these the metallic tonics rank first, especially iron and zinc, or quinine, or some form of bark, of which you will find the liquor cinchonæ of Mr. Battley one of the most useful preparations, or mineral acids; sometimes also cod's liver oil and sarsaparilla.

There are certain after-effects of chorea sometimes met with, which you should remember, and be prepared to treat. A child may be brought to you with one side completely paralysed, or with palsy of a limb, and upon questioning the friends, you will find that the child has been left in this condition after an attack of chorea.* Occasionally this paralytic state occurs as a precursor to the choreic attack, but this is rare. What are you to do in such a state? Does this condition indicate the occurrence of any important lesion? In my experience certainly not; most of the cases get well in a short time. You should persist in the tonic plan of treatment, and endeavour to improve the health as much as possible. If, however, the paralytic state should last for any time, it is important to use means to exercise the paralysed muscles, otherwise they would suffer in nutrition, and a permanently weakened state of the muscles would result. For this purpose, nothing is better than the careful use of galvanism. A gentle and slowly interrupted current from the battery may be daily transmitted through the paralysed limb or limbs, for a quarter of an hour or twenty minutes at a time; and the limbs may be frequently rubbed with a coarse towel or a flesh-brush.

The prognosis may almost always be favorable in chorea, save in those terribly acute and general cases which, by the violence of their movements, rapidly exhaust the powers of life. But, even in such cases as these, we shall be more successful, when we can succeed in divesting ourselves of the notion that such symptoms can only arise from a hypersthenic or inflammatory state. I may here mention, by the way, that the cold affusion, well managed, and repeatedly applied at short intervals, has great influence in controlling the convulsions in these cases of general chorea.

It is not easy to answer a question which is often asked, especially in private practice,—namely, as to the probable duration of a choreic attack. Generally speaking, the restoration to health, in such cases, is a slow and tedious process; and, I think, you will find it more so in private than in hospital practice, because we cannot subject our patients to the same dis-

* *Vide* ante, Lecture XLVII, p. 799.

ipline, when they are exposed to the excitements and indul-gences of home life, as when under the surveillance and direc-ion of strangers, in a well-regulated public establishment. It s a very mild case of chorea which will recover in three or our weeks; more frequently they require double that time,— often two or three months, or even five or six months, as in the ase of Mary Jackson.

LECTURE LII.

ON NERVOUS DISEASES.

ON LOCAL HYSTERIA, AND ON CATALEPSY.

Let me call your attention to-day to two interesting cases of nervous affection. The subject of the first is still in the house, but that of the other case left it a few days ago. I regret that, in consequence of the transitory nature of the symptoms from which this second patient suffered, and from the attack having occurred at midnight, few of you had an opportunity of witness-ing it. Her symptoms were of a very interesting nature, and not often met with. For these reasons, and notwithstanding that so many of you missed seeing the case, I think it may, in some degree, supply the failure of this opportunity, if I detail the case to you, and make some remarks on its pathology and treatment.

Case CCXXVIII.—First then, we take the case of Harriet B—, aged thirty, in Lonsdale ward, for the notes of which we are indebted to my clinical clerk, Mr. (now Dr.) T. Bridg-water (vol. xxxiii, p. 177). This patient was admitted on the 7th of May, 1851. The main point of interest about the case is, that she suffered acute pain in the right hypogastric region of the abdomen; the pain was so acute that she could not bear

the slightest pressure on the affected part; even the weight of the bed-clothes was painful to her. This pain was her chief complaint, and for the relief of this she was admitted to the hospital. It would be impossible to find an instance of a more strictly localised pain than that afforded by this case, nor could I bring before you a more striking example in which relief from pain was peremptorily demanded from the physician, as the one thing complained of by the patient. Cases of this kind you will often meet with hereafter in your practice, and you will do well to note carefully such as may come before you now, and the treatment which may appear to be serviceable; for nothing contributes to professional success more than the power of readily appreciating the nature and causes of pain, and tact in applying suitable and efficacious remedies.

Severe abdominal pain, whether general or limited to a spot, is at once suggestive, both to practitioner and patient, of inflammation within the abdomen; it may be of peritonitis. This latter malady is, as you know, a severe one, and, in the majority of cases, fatal. How important, then, is it to be able to determine whether such pain is due to a cause of this kind or to some other!

The pain under which this woman suffers is referred to the right hypogastric region, over the position of the right ovary, and the painful part occupies a circular space of about two and a half or three inches in diameter. There is extreme tenderness to the touch; even when the integuments are pinched up with the utmost gentleness, and with every care to avoid compression of the subjacent parts, she shrinks and appears to suffer great pain. *Deep* pressure on the corresponding region of the left side causes pain likewise, as if by implicating the right side.

There is another very important feature in the case which deserves especial notice; it is this, that when her attention is much engaged, she certainly suffers less, although she cannot be said to be free from pain. Upon making a careful examination of the part, we could detect no tumour; and, upon pressing deeply, no abnormal condition of the ovary could be discovered; and, although there was pain, there was no increase of it to a proportionate extent.

Her history we found to be as follows:—She is unmarried; but, many years ago, she fell a victim to the seduction of some improper person, and, having become pregnant, she suffered a very severe labour, in which the perinæum was lacerated; in consequence of this injury, she was under surgical treatment in the hospital for some time.

Three months before her admission into the hospital, she had a severe attack of diarrhœa, accompanied by vomiting. Her bowels have since continued in an irritable state. The catamenia have always been irregular, and there has been a constant drain, more or less, from leucorrhœa, which increased in quantity as each menstrual period recurred.

On her admission into the hospital, she was still suffering from diarrhœa, but there was nothing very unhealthy in her expression of countenance, nor was her abdomen unduly swelled. The abdomen walls were lax, and the bowels were but slightly tympanitic. It appeared that the pain came on at the same time as the diarrhœa, and at first was aggravated by taking food.

This curious and not easily explicable connexion between the hypogastric pain and the state of the alimentary canal led me to direct the early treatment to allay irritation by checking diarrhœa, which, if allowed to continue, would have impaired her general health, and aggravated the other symptoms under which she laboured. With this view, then, she was ordered to take astringents with opium, and under the influence of these remedies the diarrhœa was checked, but the abdominal pain and tenderness remained as before; hence it became the more important to determine the precise nature of this pain; and, with this view, let me conduct you through the various steps of the diagnosis.

We will suppose ourselves at the patient's bedside; and, having attentively listened to her history, and heard her complaints, we find that she suffers great pain upon the slightest pressure in the right hypogastric region, in the position and over a space such as I have already described. Upon further examination, we find the pain nearly as great under slight as under deep and heavy pressure.

What, then, are the conditions that might give rise to such a pain as this?

The first and most obvious cause, which the history at once suggested, was that the pain had its seat in the irritated bowels. It was in the region of the termination of the ileum and commencement of the colon; and there is no cause so fruitful of diarrhœa as irritation of those parts of the intestinal canal. But the objections to this view of the case were as follow:— There was no tympanitic state, such as bowel irritation sufficient to create so much pain would infallibly produce. Again, the bowel irritation was easily controlled very soon after her admission into the hospital, yet the pain remained in all its intensity.

Secondly, it might have been due to a local peritonitis; but the objections which I have just urged to the pain having its seat in an irritated bowel apply with equal force to peritonitis. And, as an additional objection, also applicable to both views, I may here state the fact that the pain was as great when the integuments were gently pinched up, as when deep pressure was made; whereas, in peritonitis as well as in inflamed bowel, firm and deep pressure would cause by far the greatest amount of pain.

A third cause might have been inflammation of the ovary. To this view, however, was opposed the absence of any swelling or tumour in the hypogastric region, and of increased pain on deep pressure. And, on examination *per vaginam*, Dr. A. Farre could not detect anything wrong with either ovaries or uterus.

Any inflammatory affection of the abdominal muscles, or of the iliacus internus or psoas muscles, was insufficient to explain the pain, because the movements of the trunk or of the limbs could be effected without pain; and, indeed, with all three of these conditions it may be regarded as quite incompatible, that our patient had never had, since she came into the hospital, that amount of fever which would accompany a severe internal inflammation.

Lastly, the symptoms might be referable to the peculiar state of the nerves, superficial as well as deep, which gives rise to that kind of pain which conventionally (and, in the present state of our knowledge, conveniently enough) we call "hysterical pain." This pain, also called a nervous pain, is no doubt just as acute and severe to the patient's feelings as any pain from injury or

organic lesion, although, in the parts in which it is seated, we can discover no alteration of structure whatever.

You see that we have excluded from the diagnosis peritonitis, enteritis, and inflammation of the ovary. Let us now inquire how far the hypothesis of the pain being hysterical is borne out by the accompanying phenomena.

The great tenderness of the skin to the slightest touch is very favorable to this view, which is likewise supported by the fact of the little difference in the intensity of the pain, under deep and under superficial pressure.

The slightest touch on any other part of the body makes the patient suddenly shrink back with a greater or less expression of pain—a character which belongs to all these hysterical affections. Often when you bring the finger close to the skin there is an expression of pain nearly as great as if you had actually touched it.

Certain features in the general constitution of the patient confirm this view; she is evidently of an hysterical constitution, and exhibits that peculiar appearance of countenance which I have often pointed out to you by the name of "facies hysterica," characterised by a remarkable depth and prominent fulness, with more or less thickness, of the upper lip. There is also a fulness and obviously drooping condition of the upper eyelids. This drooping conformation of eyelids is at once a mark of beauty, and of that from which many beautiful women suffer very much, namely, the hysterical state of constitution.

She also exhibits that irritable state of spine under which hysterical patients are apt to suffer. The least pressure on a spinous process causes her to shrink, and to complain of a pain shooting forwards from the point pressed on; and, as is so often the case with patients of this temperament, she complains of pain from pressure, in whatever part of the body it may be applied; wherever she is touched, whether the finger comes in contact with her arms, or back, or any other part of the body, she exhibits an undue degree of sensibility, and shrinks.

Then there is yet another point about the case, which I have not mentioned. We find, on referring to the history, that the patient never menstruated regularly. In the vast majority of cases of hysterical affections you will find something wrong

about the uterine function; and, although I am not prepared to lay it down positively, that the disease (as its name implies) depends upon this disturbance, you will find in the many forms of hysterical affections that may be brought under your notice, that it will rarely happen that there is not some deviation from the healthy action of the uterus exhibited in the disturbance of the menstrual function. A large proportion of hysterical patients suffer from leucorrhœa; in others the catamenia are insufficient in quantity, or occur at irregular intervals. Sometimes the menstrual secretion recurs at short intervals, and is very profuse, and we have that condition to which the term "menorrhagia" has been applied. Perhaps there is an irritable condition of the uterus, causing almost constant pain and tenderness in the uterine region. No one of these conditions is constantly observed, but in almost every case of hysteria there is some form or forms of uterine disturbance.

Thus, then, we arrive at the conclusion, that the symptoms in Harriet B—'s case are to be referred to an hysterical state of constitution. The case is a good example of one form of local hysteria, to some of the other forms of which I shall briefly call your attention.

Of all these conditions, that which has been called "irritable uterus" is by far the most formidable. It is characterised by exquisite pain and tenderness in the region of the uterus,—even the slightest pressure over the organ, or the least touch applied to it in the examination *per vaginam*, causes great suffering, and the patient shrinks in consequence. At the same time there is no enlargement or other organic change in the organ.

Another form of local hysteria is a pain of very frequent occurrence under either breast, and, what is rather curious, more commonly found under the left than under the right breast. Leucorrhœa is so often concomitant with this pain, that whenever a patient complains of it, I invariably ask if she suffers from leucorrhœa; or conversely, if leucorrhœa be present in any quantity and for any time, I am led to inquire about the local pain. That there is a connexion between the leucorrhœa and the pain in the side scarcely admits of a doubt; for the pain is more severe if the leucorrhœa be profuse, and it very

commonly happens, that as soon as the healthy condition of the uterine functions has been restored, the pain will disappear. My theory of the production of this pain is this :—I believe that in cases of this kind there is not only an irritated state of uterus, but also of one or both ovaries, and the pain is more immediately associated with the irritable ovary, the nerves of which, implanted, as many of them are, in the spinal cord, reflect the irritated state on to the nerves of the submammary region. It is an interesting example of a reflected sensation.

A third form of local hysteria is one with which you should be familiar, and able readily to diagnose. It manifests itself in that pain in the stomach, which is commonly called "gastrodynia." We must be very careful to distinguish this from the pain resulting from ulcer of the stomach, as an essentially different treatment would be applicable to each. One form of ulcer of the stomach very often occurs in young chlorotic women, amongst whom also we most frequently meet with the gastrodynia ; hence the importance of paying close attention to the symptoms, which will enable us to distinguish with certainty the one from the other. An important difference is, that in the hysterical affection the pain may occur at any or at all times, whether the stomach be empty or full ; nor is there any constant relation between its development and the ingestion of any particular kind of food. On the other hand, the pain from ulcer of the stomach is distinctly influenced by the taking of food, but especially of that kind which it is the province of the stomach to digest. Patients with ulcer will tell you, that as long as they abstain from food they have little or no pain ; but as soon as they eat anything they begin to suffer, and they are not easy until digestion has been completed. Hysterical patients, on the other hand, will tell you, that the pain comes on as soon as they rise in the morning, and continues perhaps all day. You will also find that these patients exhibit more or less of the hysterical constitution. With reference to the diagnosis between these two kinds of stomach pain, you should always inquire if there has been vomiting of blood, as this forms a most important symptom in the history of ulcer of the stomach. Or, if there have been no vomiting of blood, you should inquire whether it may not have passed through the pylorus into the

intestinal canal, in which case you will find, upon inquiry, that the patient has passed very dark matter from the bowels; the stools, in fact, exhibit a pitchy character in those cases in which blood from the stomach passes by the bowel. The effects of the treatment to which the patient is subjected will also often enable you to distinguish the true ulcer of the stomach. Beer and stimulants, and all acid drinks, greatly aggravate the pain from ulcer, while they often tend to relieve the hysterical affection. Bismuth acts very favorably in the latter, but not so well in the former.

Another form in which we find local hysteria manifesting itself is exhibited in that condition called "hysterical spine." You have a good example of it in the patient whose case has led to these remarks. If you pass your finger down the spine, you will find some places very irritable; perhaps the painful situation may be confined to one particular spot, or it may extend over three or four spinous processes, or the whole spine may be affected. Wherever it be, you will find that the patient suddenly shrinks as soon as the irritable part is touched, and appears to suffer, and no doubt does suffer, exquisite pain. Now, this condition has been over and over again mistaken by careless practitioners for disease of one or more of the vertebræ; and in consequence the unhappy patients have suffered from all the artillery of physic; leeches, blisters, and setons have been applied to the spine, and other antiphlogistic measures have been resorted to, but without any effect beyond, perhaps, aggravation of the pain. The most important point, by means of which you may distinguish vertebral caries from the hysterical affection is this,—in the vertebral disease the pain is not so excessive, and is always fixed in one part, and it will be found to increase gradually as the disease advances.

Hysteria likewise affects joints. A patient has a pain in her knee or her hip, or some other joint. I have no doubt that in these cases there is not only an affection of the sentient but of the muscular nerves likewise; for the muscles are either spasmodically affected, or relaxed, or paralysed, occasioning difficulty and awkwardness in the movements of the joint.

Many instances of what are called neuralgic pains are referable to the hysterical state, and the case of Harriet B— is one of

them. But you must take care to distinguish the true neuralgic from the hysterical affection. They are different diseases, although probably nearly allied. In the true neuralgia there is an altered nutrition of the affected nerve as decided as that of the tissues of a joint affected with a transient attack of gout. Such, however, is probably not the case in hysteria.

Our patient, Harriet B—, has continued in the hospital for a considerable time. Repeated examinations tended only to confirm the diagnosis I have given, and she has had two or three hysterical fits. On her admission she was treated for the diarrhœa under which she was then suffering. The diarrhœa ceased, but the pain remained unabated.

Since that time the treatment has been chiefly such as would check leucorrhœa, improve tone, and promote general health. The tepid shower-bath, the cold hip-bath, mild aperients with galbanum, and the citrate of iron, were administered for some time. The result was a very marked improvement of the general health.

Notwithstanding this improvement, the pain in the hypogastrium continued troublesome, although diminished in severity. Local treatment was employed; leeches, blisters, and opiate applications were applied, without good effect. At length, fearing that these applications tended rather to aggravate the pain, by fixing her attention upon the affected part, as undoubtedly they are apt to do, I applied a blister to the corresponding spot on the left side, and kept it discharging for some time. This treatment was attended with the happiest result; the pain left the right side, but, as if in illustration of its true nature, she now complains of a pain in the left or blistered side, less severe, however, than the original pain. But from the great improvement in her general health, and the abatement which has already taken place in her symptoms, there can be no doubt that time is now the most important element in her cure.*

Case CCXXIX.—I shall now proceed to the second case; it is, I think, well worthy of your attentive consideration. The

* This patient remained in the hospital till August 31, and was then discharged very much improved in every respect.

patient's name is Amelia D—, and she is thirty-eight years of age. (Vol. xxxiv, p. 64.) I am sorry that she is not still in the hospital. The fact is, that the urgent symptoms for which she was admitted yielded so quickly, that she remained in the house only a very short time. Still, the symptoms were so remarkable, and the case so rare, that I feel I should not be doing right were I not to bring it specially before you.

In giving you a history of this case, I cannot do better than quote from the graphic description in the notes made by Dr. Hyde Salter, who, as house-physician, saw her from the moment of her admission.

"On June 5th, about ten o'clock in the evening (Dr. Salter states), I was called to a patient just brought into the hospital by a policeman, who had found her speechless in the streets. I saw a woman sitting in a chair, looking quite intelligent and coherent, although evidently in great excitement and distress; her expression was anxious, and she looked from one to the other in an inquiring and imploring manner; her teeth were fast clenched, and her lips parted. On being asked who she was, she shook her head; when asked what was the matter with her, she pointed to her mouth and masseter muscles; when asked if her jaws were locked, she nodded her head. I then tried with all my force to separate them, but could not; then I felt her cheeks over the masseters, and found these muscles contracted into hard knots, which sufficiently explained the closure of the jaws. When questioned as to the cause of the tetanus, she clenched her fist and struck her left cheek, implying that she had had a blow there. I said, 'Have you had a blow there?' She nodded her head. We asked if she could write. She shook her head, and clasped her hands in a despairing way. We then asked her if her husband had given her the blow. She nodded assent eagerly.

"By degrees, putting all sorts of questions, and getting nods or shakes of the head, as the case might be, I learned that she lived in the neighbourhood of London, that she had been struck by her husband that day at two o'clock, and that the blow was the cause of her illness; also, that she was thirty-eight years of age. All these things afterwards proved to be true. While answering our questions as well as she could, she

suddenly, and without any warning, slid from her chair in a state of apparently complete insensibility, and would have fallen on the floor had she not been supported. Then commenced a series of spasms more strange and horrible than can be conceived or described; tetanic and clonic, partial and complete, symmetrical and irregular, varied in every conceivable and inconceivable way. First she had opisthotonos, then she was thrown forwards, then she twisted round and writhed like an eel, then she would throw herself forward and raise herself into a sitting posture, then she would roll over and over, then a slow undulation or wave of spasm would pass over her from head to foot, producing different movements of her limbs as it passed down, like a dog dying of hydrocyanic acid poisoning."

You might almost be led to think that this description is overdrawn, but I can myself bear testimony to the accurate statement of the facts, as I was present part of the time. Then, continues Dr. Salter, "the tonic spasm would suddenly become clonic, and she would throw out her arms and draw them back with great force. The legs would be affected in a similar way, or drawn up to the body, so that the heels were close to the buttocks, or thrust stiffly out. When holding her hand, she would suddenly clutch mine with such force that I could not disengage it. Meanwhile, her face was undergoing all sorts of contortions, at one time expressing rage, at another intense fear, then a sneer, then a fixed and rigid stare; the eyes might be rolled upwards or downwards; the pupils dilated and insensible to light; then the elevators of the upper lip and ala of the nose would jerk, perhaps on one side or both, exposing the teeth. The depressor anguli oris would draw one corner of the mouth down on the chin, or the platysma throw the skin into a state of rigidity;" but, as Dr. Salter remarks, it is quite impossible by description to convey an accurate idea of the extraordinary contortions of the patient.

One of the most remarkable features in the case was the rigid tonic condition of the whole muscular system. Every muscle employed in any movement became at the same time as hard as a board, and the movement was not so much executed with rapidity, as with great force, and even slowness. But in watch-

ing her attentively, it was plain that the movements, varied and irregular as they were, did not partake strictly of the character of involuntary movements. They were evidently influenced by a will, but by a diseased and an ill-directed will. While these spastic movements of the muscles continued, there appeared to be complete insensibility to surrounding objects; but when they ceased, her intellect seemed quickly to recover itself, and she could talk freely and collectedly. At this time she gave us her history, which we afterwards ascertained to be correct. In the midst of her narrative her eyes suddenly became fixed, and she stared at us for a short time and fell off into another paroxysm, and went through the same series of varied movements and contortions as before. She continued in this state, alternately in fits and quiet, for nearly two hours, and then slept.

Now, on being called to a case of this kind, you would have, on the spot, to decide upon its real nature, without the help of any history on which full reliance could be placed. Such was our case when we first saw this patient; and, therefore, before I give you any further details, I shall state to you the conclusion arrived at by Dr. Salter and myself, and the grounds of our decision. We had to distinguish these extraordinary muscular movements from those of trismus, of tetanus, and of epilepsy.

The distinction from trismus was sufficiently easy. The fact of her coming to herself so soon, and the subsidence of the spasmodic action of the muscles, were quite sufficient to enable us to determine that the condition was not trismus, and the absence, in the history, of all those causes which ordinarily give rise to this affection also tended to the same conclusion. In the same way that we were enabled to say that we were not dealing with a case of trismus, we also came to the conclusion that it was not one of tetanus. Trismus and tetanus are but degrees of the same affection. In both diseases the muscular movements are distinctly involuntary, and often excited by the application of some stimulus to the surface. In this case, however, the movements bore a decided resemblance to the voluntary class, and could not be excited at the will of the observer.

At first sight, the affection bore a greater resemblance to epilepsy than to any of the other forms of convulsive disease;

but there were certain points which enabled us very positively to decide that it was not epilepsy. These were the nature of the movements, and the affection of the consciousness. The movements partook more of the tonic than of the clonic character; and although at times very much varied, they were combined and regular, and directed to an end and by a purpose, rather than irregular, rapid, and, as it were, explosive, as is the case with the convulsions of epilepsy.

The most important feature was the state of the consciousness, and by this chiefly we were enabled to decide against the epileptic nature of the case.

In epilepsy, complete coma, with total loss of consciousness, forms an essential and specific character. Convulsions form no necessary part of the epileptic paroxysm. There may be in epilepsy a complete absence of convulsions, but the insensibility is invariably present. Thus we often meet with instances in which there is sudden and even momentary loss of consciousness, without any convulsion whatever. Such cases are not less truly examples of the epileptic paroxysm, than if the most violent convulsions were also present. Attacks of this non-convulsive kind constitute what the French call *petit mal;* but, in many instances, it is rather the *grand mal,* for the brain and the intellectual powers suffer much more after repeated attacks of this nature than after fits, in which the convulsive movements form the prominent feature of the disease.

In the case before us, however, we had no complete loss of consciousness; there was, undoubtedly, an affection of consciousness, that in which a person may be insensible to all ordinary external stimuli, and quite without the perception of what was going on around her, but at the same time aware of the altered state of the mental powers. In epilepsy the patient knows nothing of his mental state during the paroxysm; he is only conscious of his state before and after it—and the period of the fit is to him as if it had never existed.

The nature of the movements showed that consciousness was not wholly in abeyance; they exhibited a co-ordination and a regularity, which we never observe where there is complete loss of consciousness. She bent her fingers in a steady regular manner, as if the muscles were obeying the mandates of her

will, and her body was contorted by a combined action, forming a striking contrast with the jerking movements which we see in a state of insensibility. I say, then, that in this and in other cases of a similar nature, we have no complete loss of consciousness; the apparent insensibility depends upon an intense concentration of the attention on one particular object. Let me illustrate what I mean in this way. We all know that when the mind is very much interested and occupied with any particular subject or train of thought, one is very apt to take no notice of occurrences which are taking place close to, or in immediate connexion with, one's person; and to be unconscious even of noises; nay, one may suffer a moderately severe pinch, without being sufficiently roused from the state of reverie to direct attention to any but the one object; in fact, the mind is fixed on that one object to the complete exclusion of everything else for the time.

The state of consciousness into which such patients as Amelia D— fall is of this nature, although very different in degree. What is commonly called "absence of mind" is an analogous affection of consciousness. The attacks are generally brought on by some powerful mental emotion—grief, anger, jealousy,—which overwhelms and suspends all other intellectual operations, while its influence lasts.

Then, if the disease were not trismus, nor tetanus, nor epilepsy, what was it? All the symptoms and the history of the case combine to show that it was an example of a highly developed hysterical paroxysm or fit, or more exactly, of that peculiar and aggravated form of hysteria which is called *catalepsy*.

The history, which has been carefully collected and recorded by my clinical clerk, Mr. Maurice Davis, shows that our patient was a highly excitable, hysterical person, who has been subjected to moral and perhaps physical influences also, well calculated to keep up that state. She tells us that she enjoyed good health until she reached the age of ten, when she experienced a sudden fright, and fell, in a fit, two stories down a well-staircase; from that time until her marriage (at the age of fifteen), she had the fits repeatedly, generally twice a week, and sometimes oftener. Since her marriage, she has had the fits less frequently, but has never been entirely free from them.

It is difficult to determine what was the precise nature of these fits, whether they were epileptic or hysterical. Her age was more favorable to the former; but the subsequent history, and the fact elicited from her, that she was advised to marry in order to get rid of the fits, rather indicate that they were of the hysterical kind. At the same time, it is unusual for a girl of ten years old to exhibit the symptoms of hysteria. We rarely find any manifestation of the hysterical condition until after the appearance of the catamenia. She was persuaded to marry at the age of fifteen, and, as she had no great affection for her husband, domestic squabbles occurred very soon after their union, and in some of these disagreements her husband used violence, and, in consequence, she had a return of the fits. At last they agreed to separate, but circumstances obliged them to have occasional interviews, and of necessity scenes of violence were repeated on each occasion. It was at one of these rencontres that the present attack commenced. The influence of moral causes in inducing the paroxysm is very manifest in this history. Ever since fifteen years of age, she stated, the slightest excitement would bring one on. She is evidently a woman of strong passions and violent temper, and possibly her partner was as much "sinned against as sinning." The same hysterical cast of features existed as in the case of Harriet B—, with which I commenced the lecture, and there were many of the same peculiarities of the hysterical constitution.

There is a curious family history in this case. Her paternal grandfather and paternal uncle had fits, and she was the mother of nine children, eight of whom died, seven of them in convulsions!

And now let me refer briefly to treatment. I would just remark here, that the same general principles of treatment are applicable, whether the affection be chiefly manifested in a particular part of the body, as in the case of Harriet B—, or whether it affects the system generally, as in the case of Amelia D—.

Before, however, we discuss the treatment, we must endeavour to form some notion of the nature of hysteria. What is this hysterical condition? To answer this question properly would, I fear, occupy me too long, but I may tell you what it is not,

by which you may be guided in your practice, and be enabled to meet the disease by the proper treatment. Now, in the first place, hysteria does not result from inflammation. It is not a disease of an inflammatory type, and no part of the system whatever is in a state of inflammation. Hysteria, no doubt, occurs chiefly in persons who have a peculiar character of nervous system, very often inherited from nervous or gouty parents. It depends partly upon this original conformation of nervous system, and partly upon a depraved state of general nutrition affecting the whole body, and accompanied by a morbid state of the blood. It is always very much influenced by a disordered condition of some of the great emunctories. Hence we must take care that the powers of the patient are not in any way diminished by the treatment to which she is subjected, or by the occurrence of fluxes from any part of the body which may tend to lower her strength. We must also be careful not to overlook any derangement of the digestive organs; we must uphold the strength, and endeavour to improve the patient's health in every way.

Although the hysterical condition is mainly physical, and, as such, transmissible from parent to offspring, you will not lose sight of the fact, that it is readily affected by moral influences. A sound education, as regulating the habits, is of primary importance to the hysterical patient, and a judiciously directed moral treatment is essentially necessary to give full effect to such physical remedies as may be applicable to the case. On the other hand, an ill-regulated mind and an imperfectly subjugated will are among the most frequent moral conditions which tend to excite and prolong the disease.

When you meet with a case such as the present, in which a violent paroxysm has occurred, what are you to do? Are you to look on and do nothing? The friends will anxiously inquire if nothing can be done to relieve the patient. I confess that I cannot tell you of anything which will, with certainty, quickly cut the paroxysm short, but at the same time certain means may be had recourse to, for the purpose of diminishing its violence and duration. Whatever tends to rouse the patient, without alarming or creating exhaustion, is likely to prove useful, such as shaking her without violence, or throwing cold

water in her face, or splashing her well with water. It is desirable to get the bowels to act freely as soon as possible. Means of this kind were used in the case of Amelia D—, and an enema, containing half an ounce of spirits of turpentine with an ounce of tincture of assafœtida, was administered. She took, likewise, for a short time, twenty minims of the fetid spirit of ammonia, and ten minims of tincture of hyoscyamus in camphor mixture at short intervals.

If the patient has been indulging too freely in beer or spirits (as is often the case with persons brought into the hospital), or if you have reason to suspect that any indigestible food has been taken, an emetic or the stomach-pump will be of great service. It rarely happens that taking blood, or other antiphlogistic treatment, is requisite.

The patient, Amelia D—, recovered in three or four hours from her fits, and slept the rest of the night. Next morning she was perfectly tranquil, and could not be prevailed on to remain in the hospital.

Catalepsy occurs in men, and, judging from what I have seen, in a form much more severe and fatal than in women. In men, as in the other sex, it is associated with exhausting causes and demoralising influences, and is altogether a more serious malady, destructive to mind as well as body. At some future time I may have an opportunity of discussing the disease as it occurs in males.

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