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INTERNATIONAL CLINICS:

A QUARTERLY OF CLINICAL LECTURES

ON

MEDICINE, NEUROLOGY, PEDIATRICS, SURGERY,
GENITO-URINARY SURGERY,
GYNÆCOLOGY, OPHTHALMOLOGY, LARYNGOLOGY,
OTOLOGY, AND DERMATOLOGY,

BY

PROFESSORS AND LECTURERS IN THE LEADING MEDICAL COLLEGES OF THE UNITED STATES, GREAT BRITAIN, AND CANADA.

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VOLUME IV. SECOND SERIES. 1893.

PHILADELPHIA:
J. B. LIPPINCOTT COMPANY.
1893.

Printed by J. B. Lippincott Company, Philadelphia, U.S.A.

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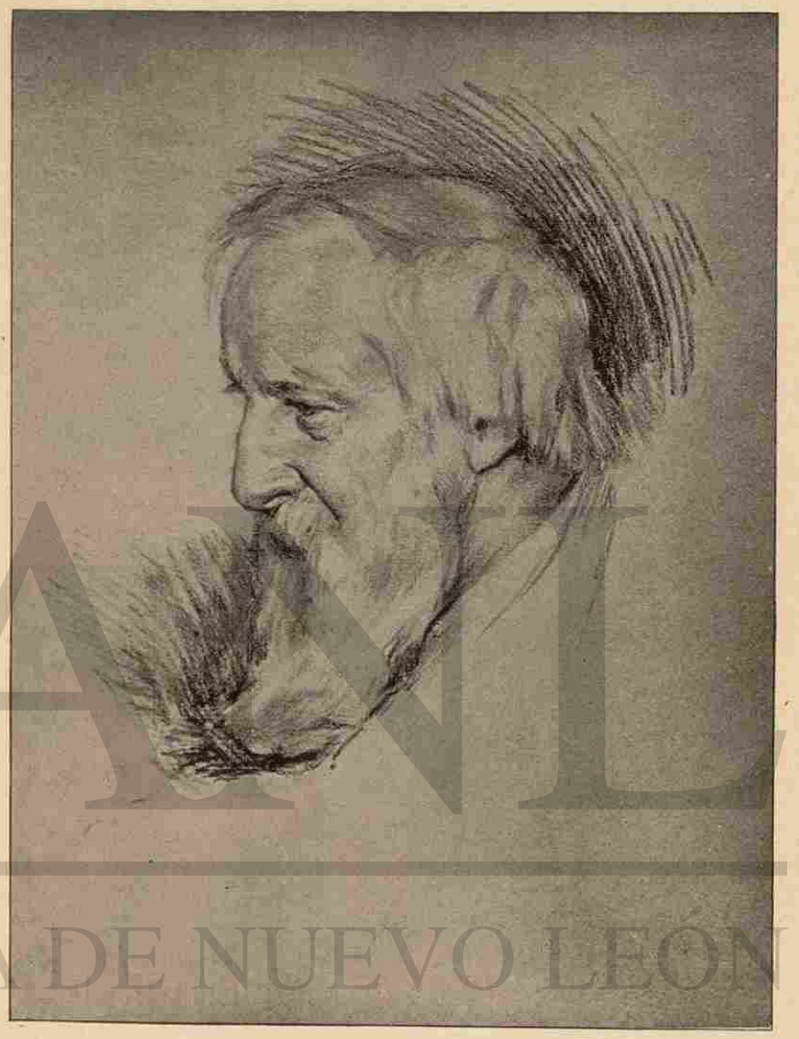

Henry D. Barrett

THE LATE PROFESSOR

Henry Ingersoll Bowditch.

BY FREDERICK I. KNIGHT, M.D.

Dr. Henry Ingersoll Bowditch was born in Salem, Massachusetts, August 9, 1808. His father was Nathaniel Bowditch, the eminent mathematician, and his mother Mary Ingersoll; parents who have transmitted in a remarkable degree to their descendants the honesty and strength of character peculiar to them. The father, as is well known, educated himself in hours which by others were taken for rest or recreation; and this hard experience led to restrictions in the education of the children, which, though some of them were afterwards regretted by the latter, may have been on the whole beneficent. As one instance, they were never allowed to devote any time to music, the study of which, considering the hard struggle in life before them, the father considered a waste of time, and likely to lead to greater waste in the enjoyment of it. That this and other restrictions were imposed with considerate tenderness is abundantly shown by the thorough respect and obedience of the children.

The only school in Salem attended by Henry, of which I have any positive knowledge, was the Salem Private Grammar School, situated on Green Street, which was kept at the time by John Walsh, son of Michael, of arithmetic fame. I know that Dr. Bowditch attended here in 1822, for I have seen a programme of an exhibition at the school in this year, on which he appears for a Latin dialogue with J. B. Bigelow. It is interesting to notice on the same programme the names of Henry W. Pickering, of this city, and Benjamin Peirce, late Professor in Harvard College. The fact that Dr. Bowditch was selected for a Latin dialogue argues, I think, that he was at this time a good deal more of a student than he used to represent himself. He was, however, not a "house-rat" (as we were accustomed to call boys who stayed in with their books all the time), but was a thorough boy, fond of out-door

exercise, full of life and innocent fun. On a photograph of the typical Salem house in which he was born and lived, he has marked an upper middle window as one from which he and his brothers used to pelt with beans the promenading boys and girls on Sunday, they themselves being allowed out on that day only to go to and from church. There is a tradition also of the subject of our sketch having had a hand in introducing some fire-crackers into a certain old lady's teapot.

The family moved to Boston in 1823, Dr. Nathaniel Bowditch having been invited to the Presidency of the Massachusetts Hospital Life Insurance Company, which afterwards under his management attained such wonderful growth and prosperity. In Boston Henry attended the Public Latin School, entered Harvard College as a sophomore, and graduated in the class of 1828.

Dr. Bowditch always represented himself as an indifferent student, as "students" were estimated in those days,—that is, he was not a dig for recitation-marks. None of his teachers appear to have excited any enthusiasm or admiration except Professor Charles Follen, to whom he was always grateful for having suggested and urged upon him the study of the German language. This was very likely the only personal interest ever shown in him by any member of the Faculty. However this may be, and student or no student by the gauge of the day, I am sure Dr. Bowditch was busy at something, for a more industrious man I have never seen. He was always occupied. I have wondered whether the non-use of tobacco might not have had something to do with this, knowing how often it serves its devotee as both companion and occupation. He apparently had one of those brains rested by change of work. He never sat still musing, or walked up and down thinking out the solution of any subject, but he thought with pen in hand.

After taking his academic degree, Dr. Bowditch entered the Harvard Medical School. What determined his choice of a profession I have not learned, except that his mother was desirous that her sons should take different professions, and he felt himself more inclined to medicine than to theology or law. There are now few living associates who can tell us of his immediate, enthusiastic devotion to his profession when once chosen, but of the fact there is no doubt. The medical Faculty at this time consisted of Drs. Walter Channing (Dean), John C. Warren, James Jackson, Jacob Bigelow, and John W. Webster. In September, 1830, he entered the Massachusetts General Hospital as medical house-pupil, and served one year, his colleague on the surgical side being Thomas R. Thomas, Jr. He received his medical degree in 1832, and

went to Paris, as was the custom in those days, to complete his medical education. It was natural that a man of his mind and home-training in regard to exact truth should have been soon attracted to Louis and his teachings, and eventually to have been thoroughly devoted to them. The numerical method, as it was called, the recording and analyzing of symptoms in a large number of cases without any preconceived theory of the disease, simply the recording of facts and drawing logical deductions from them, was then being expounded by Louis, whom Dr. Bowditch delighted to call master. So thoroughly did Dr. Bowditch always practise this method, so thoroughly did he identify himself with it, and so consistent was it with his own character, that one can hardly help feeling that, even if he had not had the advantage of Louis's teaching, he would have adopted such a method himself. His friendship with Louis was kept up till the death of the latter. If asked what he had learned abroad that was especially valuable, he, while admitting the many things in clinical and pathological work which were new to him, would undoubtedly have said, "What I value most is the proper method of observation and recording of cases."

Another great good fortune came to Dr. Bowditch in Paris, in that it was here he first met Miss Olivia Yardley, who was destined a few years later to become his bride; for Mrs. Bowditch had all the qualifications for his complement, whether it was in managing the exchequer, in making drawings of his microscopical preparations, or in the exercise of accomplishments which go to make up the amenities of life.

After a residence of two years in Paris he returned to Boston (in 1834), and established himself in practice. His office was at first on Bedford Street, soon afterwards on Otis Place.[1]

With enthusiasm he devoted himself to the propagation of the teachings of Louis, and founded in 1835 a society for medical observation, on the plan of the one in Paris, for practice in the correct observing and recording of cases. Its membership was small, chiefly medical students, the only physicians at its organization being Dr. John Ware and Dr. Bowditch. This was discontinued in 1838.

Soon after this, Dr. Bowditch was associated with Drs. Marshall S. Perry, Charles H. Stedman, and Henry G. Wiley, in a private medical school. They had about fifteen students. There were recitations and clinical instruction. The recitations were held at an infirmary for chest-

[1] Since writing the above, I have been informed by one of our profession, who settled in Boston about the same time, that he remembers visiting Dr. Bowditch, soon after his return from Paris, at an office on Washington Street, near the Marlboro' Hotel.

diseases, with which most if not all of the teachers were connected. Dr. Stedman was connected with the Marine Hospital at Chelsea, and the students went there regularly. Dr. Bowditch, in addition to his duties as admitting physician to the Massachusetts General Hospital, made the autopsies. These the students of his private school were permitted to witness. About 1843, Drs. Wiley, Perry, and Stedman were succeeded by Drs. George C. Shattuck, William E. Coale, and Samuel Parkman. Dr. Bowditch retained the position of admitting physician to the Massachusetts General Hospital from 1838 to 1845. Exactly how long the private school was continued I do not know.

Dr. Bowditch's first publication was a revision and alteration of Cowan's translation of Louis's "Pathological Researches on Phthisis," in 1836. His second was in 1838, a translation of Louis on "The Proper Method of Examining a Patient, and Arriving at Facts of a General Nature." His next was a long and spirited reply to some animadversions of Dr. Martyn Paine, of New York, on the writings of M. Louis. In 1842 he published probably the first case of trichina spiralis ever reported in this country. This was illustrated by excellent steel cuts, the drawings for which were made by his young wife from his own microscopic preparations.

From the moment of his settling in Boston, Dr. Bowditch interested himself in all that concerned the welfare of his fellow-men. With Charles F. Barnard, John L. Emmons, and others, he founded the Warren Street Chapel for the education and elevation of the young. He was superintendent of its Sunday-school, and endeared himself to every one in it. The children went to his office every Saturday afternoon for books, and the young men used to meet him on the Common at five o'clock in the morning to play cricket, they being clerks in stores and not able to go at any other time. One of them, however, says in a recent letter that he used to steal time from his dinner hour to call for a talk with Dr. Bowditch, who at that time was not oppressed with patients, and was always glad to see him. He well remembers that Dr. Bowditch was quite elated that his first year's income equalled that of Dr. John Ware's first year, namely, seventy-five dollars. Dr. Bowditch kept up his interest in the children of the Chapel long after they left it. One of them writes me that some years after leaving she received the present of a book from him, with a note saying he had intended to buy her a nice one, but on consulting his purse he found he could not do so and pay honest tradesmen, so he sent her a book called "Best Hours," which he for a long time had kept by his own bedside.

Dr. Bowditch had but just settled in Boston when the mobbing of

Garrison occurred, and henceforth till the proclamation of emancipation he was an active, zealous, uncompromising anti-slavery man. He was the intimate friend of Sumner, Andrew, Bird, May, and the other leaders of this at that time unpopular cause.

In 1846 the visiting medical staff of the Massachusetts General Hospital, which at that time consisted of three physicians, namely, Drs. Jacob Bigelow, Enoch Hale, and J. B. S. Jackson, was augmented by the addition of three more, namely, Drs. Henry I. Bowditch, John D. Fisher, and Oliver Wendell Holmes. He served here eighteen years. Any one who ever made a visit with him knows how thoroughly he did his duty both to the hospital and to the patient.

In this year (1846), Drs. Henry I. Bowditch, Charles E. Buckingham, George Derby, John D. Fisher, Samuel Kneeland, Jr., Fitch E. Oliver, Wm. H. Thayer, and John B. Walker revived the Society for Medical Observation. This society in its early days approximated more nearly to Dr. Bowditch's ideal of what a medical society should be than any we have had since. The members were accustomed to sit around a table with pencil and paper taking notes, and when the reader of a paper had finished, they were called upon to criticise. "The criticism," as one of the original members, Dr. Wm. H. Thayer, of Brooklyn, writes me, "was courteous, but unsparing, and from time to time some member resigned, being unable to stand it." Dr. Thayer also allows me to quote from a letter written to him, while temporarily absent from Boston, by Dr. Bowditch. The date of the letter is April 26, 1846. In it he says, "We have had one meeting of the Society for Medical Observation. Doctor, I have fears of that society falling into the common routine of talking societies. At this meeting we burst away constantly into a general, very desultory conversation; and when I took hold of the presented case, and said everything about it that was in my heart, whether severely critical or blandly complimentary, I saw the members were unprepared for such plain speaking. Now the doctor (that is, one of *us*) does not want another society for that purpose. He can get enough of *social conversation* at the other society; and many other things of great importance he learns there; but he does not get sterling, true criticism, a perfectly transparent truthful criticism on every paper presented, such as he *hoped* to get at the Observation Society. In our society we must get over *desultory conversation;* read strict papers, and stick closely to them, and let mere opinions and guesses go elsewhere. Each member must not only submit to the severest criticism, but he must be *thankful for getting it.* Otherwise I would not give one farthing for our society, and, for one,

I frankly confess that I shall leave it, and attend the '*conversazioni*' at Tremont Street."

Dr. Bowditch's contributions to medical literature now became more important. It is sometimes said that a man does his best work before forty years of age, but that could hardly be said of medical men of that time, certainly not of Dr. Bowditch. They did not rush into print prematurely, but waited till experience gave them the right to speak with authority. He published "The Young Stethoscopist" in 1848, when he was just forty, and his first communication on "Paracentesis Thoracis" in 1851. Probably his communications on this subject, appearing at intervals during the remainder of his active professional life, are more widely known, and have done more to extend his reputation, than anything else he has written. While Dr. Bowditch never thought of claiming the discovery of the method of removing fluid from the chest by suction, he appreciated at once the value of the method, and made such practical use of it as finally, after constant iteration and reiteration in societies and medical journals, to compel the profession not only of this country but also of the whole civilized world to the same appreciation of it.

In 1852 and afterwards, Dr. Bowditch gave courses of instruction in auscultation and percussion in the Boylston Medical School. This school, as some of my hearers will remember, was a private school, which, however, gave a complete course of medical education, had its own dissecting-room, infirmary, etc., but did not confer degrees. Dr. Wm. H. Thayer, who was one of the founders and a teacher in this school till he left the city, says that it was established for the purpose of getting more thorough hard work out of medical students than was the fashion of the time, and to encourage the graded system of study. If the Faculty had held together, the right to confer degrees would undoubtedly have been granted them by the Legislature. They did not hold together, and the school was discontinued in 1855. A school whose Faculty consisted of such men as Charles E. Buckingham, Edward H. Clarke, John Bacon, Jr., George H. Gay, Henry W. Williams, Henry G. Clark, and John C. Dalton, Jr., would surely have been a formidable rival of the Harvard Medical School.

Dr. Bowditch was appointed to the Jackson Professorship of Clinical Medicine in 1859, succeeding Dr. George C. Shattuck, who was transferred to the Hersey Professorship of the Theory and Practice of Medicine, vacant by the resignation of Dr. John Ware. He continued in this chair eight years. As a teacher he had as little capability for oratorical display as his master, Louis, but his careful examination of patients and analysis of symptoms rendered his exercises very attractive and highly valued by students. He continued and took great interest in the clinical conference, which had been introduced by Dr. Shattuck, and which became and has remained to this day a highly esteemed feature of clinical instruction at the school.

More than by his teaching, however, by his utterly unselfish zeal in his search after truth and the welfare of his patients, has Dr. Bowditch influenced those who came near him, and to-day hundreds are working on a higher level in consequence of their having known him.

In 1852, Dr. Bowditch wounded his hand in an obstetric operation. Septicæmia and a long illness followed. This caused him to give up midwifery, and as years went on, although he did not call himself a specialist, and although he continued to see all kinds of medical cases, especially in consultation, his practice became more and more limited to thoracic diseases, on which he had now become an authority.

In 1862 he published his exhaustive investigations on soil-moisture as a cause of consumption in Massachusetts, which, with the subsequent work of Buchanan, in England, in the same field, have proved beyond question that soil-moisture may be an important factor in the production of the disease.

During the Civil War he did everything in his power for the cause of the government and good of the soldier. Especially did he labor hard for the adoption of a proper ambulance system in our army, which was finally accomplished, largely through his efforts. He gave his first-born to the army, and bore his death in battle with heroic resignation.

My personal acquaintance with Dr. Bowditch began in 1863, when I joined the Harvard Medical School. I saw him especially at the clinical conferences which he held, assisted by Drs. Ellis, Minot, and others. Here was first exhibited to me one of the many fine qualities of Dr. Bowditch's character,—magnanimity to an opponent. A medical student, able but precocious and impudent, who was a candidate for the position of house-pupil, had taken upon himself to criticise Dr. Bowditch in a very contemptuous manner in the conference, and to continue his animadversions after leaving the conference, in a way which came directly to Dr. Bowditch's ears. It was thought by all that he would not get a vote from the staff for the position. He got one, and that was from Dr. Bowditch! who said that he considered him the most capable of the applicants.

The next year, feeling that I would like an opportunity to learn something more of auscultation than I was getting in the wards of the

hospital, and having heard that Dr. Bowditch had at times taken students as clinical clerks in his office, I went one afternoon to his house on Boylston Street, where he had been established since 1859, and found the waiting-room full of patients and the doctor rather "in a mess." As soon as I made known my errand he put a record-book in my hand, and set me at work getting histories of cases. There are others here to-night who have held this position in Dr. Bowditch's office, and who can testify how instructive it was. We took the histories of all the men and many of the women, and when it came to the physical examination in the inner office, from how few were we excluded! He would call us in and ask us to examine, as if for consultation, except in cases of some very few ladies. Dr. Bowditch thoroughly recognized a mutual obligation in this arrangement, and never begrudged the time to discuss and explain anything which was of interest. Not the least of the advantages gained by a student from being present in the office of a successful practitioner was the knowledge of how to deal with and talk to patients. In the latter part of March, 1867, I began private practice in Dr. Bowditch's office, to take charge of patients requiring laryngoscopic examination and treatment, and of other patients during his absence. During the twelve years I was thus associated with him there was never, to my recollection, an unpleasant word passed between us!

From this time on Dr. Bowditch devoted himself to private practice and State medicine, excepting a short service as visiting physician at the City Hospital. He was largely instrumental in the establishment of the State Board of Health in Massachusetts (the first one in the country), and was its chairman for ten years. During this time many reforms were carried through against determined opposition, the greatest of these being the abolition of private slaughter-houses in our neighborhood, and the establishment of the abattoir at Brighton. He was also for a short time a member of the National Board of Health, established after the yellow-fever epidemic of 1878. He contributed valuable papers in this domain, notably the address on "Hygiene and Preventive Medicine," at Philadelphia, in 1876, and on "The Sanitary Organization of Nations," in 1880.

For many years he was a regular attendant at the meetings of the American Medical Association, and one of the most respected and beloved of its members. He was president of the society in 1877, the meeting being held in Chicago.

His spirit of reform led him in these later years to warmly espouse the cause of the admission of women to the Massachusetts Medical

Society, which was accomplished in 1884, and to advocate a more liberal attitude towards educated medical men who may profess doctrines to which we cannot subscribe. (The past, present, and future treatment of homœopathy, eclecticism, and kindred delusions which may hereafter arise in the medical profession, as viewed from the standpoint of the history of medicine and of personal experience. A paper read before the Rhode Island Medical Society in 1887.)

Dr. Bowditch, besides holding the principal positions which have already been mentioned, was consulting physician to the Massachusetts General, City, Carney, and New England Hospitals, a member of the principal medical societies in Boston, Fellow of the American Academy of Arts and Sciences, of the Paris Obstetrical Society, of the Paris Society of Public Hygiene; and honorary member of the Royal Italian Society of Hygiene, of the New York Academy of Medicine, of the Philadelphia College of Physicians, and of the New York, Rhode Island, and Connecticut State Medical Societies.

In 1879 Dr. Bowditch met with an accident to his knee, which ever after gave to him an appearance of general infirmity which did not belong to him. With this exception, he maintained his vigor to a remarkable degree until the last year of his life. Increasing deafness rendered his attendance at society meetings less frequent, but he maintained an intense interest in all that was going on in the profession. Within a very short time of his death, the last time but one when he was able to see me, he slapped me on the shoulder and said, "Come, doctor, tell me, aren't they going to make something out of this lymph business?" referring, of course, to the labors of Koch with tuberculin.

Dr. Bowditch revisited Europe three times, namely, in 1859, 1867, and 1870. These trips gave him an opportunity of renewing old acquaintances and making new ones among the profession abroad. He wrote home very full, interesting letters, which he afterwards claimed as his diary. Such vacations were enjoyed by Dr. Bowditch more than by most professional men, for he was a man of much general culture, who read and re-read his classics, was exceedingly fond and appreciative of art and of the best music, though, as I have before mentioned, this part of his education was purposely neglected by his father.

After the death of his wife, which occurred in December, 1890, his own life began to flicker; and, although he struggled heroically against the loss for the first six months, it was then evident that the beginning of the end had come to him also. After six months' tedious illness he died January 14, 1892, in his eighty-fourth year.

Dr. Bowditch's life was a very full one, distinguished, whether we consider him as a physician, teacher, citizen, or simply as a man, by courage, simplicity, zeal, industry, and an intense interest in progress. There never was a man who more completely disregarded consequences when he felt that duty dictated action; whether this was a criticism of current medical practice, or of the selfish motives of obstructors of sanitary legislation, the defence of a runaway slave, or the branding of a deserter from the army.

His simplicity of character was such that on acquaintance his bitterest enemies became his best friends. How true was this with regard to our Southern brethren! When the war was over, it was *ended* as far as he was concerned; and he was one of the first to welcome the grandsons of John C. Calhoun to his own hospitable fireside. Members of our profession in the South, who had regarded him as an arch-enemy, soon became his dearest friends.

As a natural result of his transparent simplicity, there was a playfulness in him which, as in his father, continued to the last.

It is unnecessary to dwell upon the earnestness of Dr. Bowditch's character before such an assembly as this. There was a reality in it which none of us will ever forget, and which never ceased till his object was accomplished, whether it was compelling the world to adopt paracentesis for the relief of pleural effusion, or the emancipation of the slaves, securing a lock of Highland Mary's hair, or disproving the authenticity of the so-called portrait of Ambrose Paré.

That his industry was remarkable is testified by the large number of pamphlets and almost innumerable scrap-books and manuscript notes upon all subjects left by him. This trait may have been inherited, and certainly was taught by his father, who once seeing some idlers lounging about in Salem said, "I wish I could have the time of those men."

Up to his very last years Dr. Bowditch's interest in progress of every kind was most enthusiastic. Nothing could dampen his zeal in the search of new truths. This led him to sympathy and association with the youngest of our profession. He would welcome promises of good to come from this and that new method of practice, as if he had not been already disappointed a thousand times. When they were a little past sixty years of age a classmate came in one morning and said mournfully, "We are all dying." "Hang it," retorted the doctor, "*we* are still *alive:* go to work;" and he himself continued always to act up to the well-known proverb, "*Work* as if you were going to live forever, *live* as if you were going to die to-morrow."

Of Dr. Bowditch's religious belief a word is certainly proper, but others may know more of this than I. He indignantly repelled for himself and the medical profession the charge of atheism so often made against it. When the news of his mother's death reached him in Milan, in 1834, his whole nature at first seemed to revolt against the loss, but on entering the beautiful cathedral this feeling was succeeded by a calm belief in God and his goodness, and his direction of all things for the best, which continued with him through life. On his return to Boston he diligently followed the preaching of Dr. Channing, taking copious notes, which are still extant with something like this endorsement made on them years after: "I do not destroy these notes, as they are evidence of what interested me at the time, but these things (meaning theological problems) have long ago ceased to interest me." He was always a reader and lover of the Psalms and other books of the Bible, but he "did not believe in creeds made by men."

Many here to-night will express their admiration of Dr. Bowditch's character more fittingly than I, but none feel it more deeply. "He was a man, take him for all in all, I shall not look upon his like again."

Medicine.

THE RELATION BETWEEN RENAL DISEASE AND DISEASE OF THE CIRCULATORY SYSTEM.

CLINICAL LECTURE DELIVERED AT THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA.

BY JAMES TYSON, M.D.,

Professor of Clinical Medicine, University of Pennsylvania, Philadelphia.

THE patient I now show you is introduced more especially to illustrate the relation between kidney-disease and heart-disease, a relation not always easily made out. I desire first, however, to contrast the form of disease presented with another variety of kidney-affection, of which we have had some examples before us. I allude to parenchymatous or tubal nephritis, while this is a case of chronic interstitial nephritis, of which the final result is the chronically contracted kidney, the essential anatomical alteration in which is an overgrowth of interstitial tissue. In the parenchymatous disease, you will remember, the lesion begins in the tubules. The cells swell, proliferate, distend and enlarge the tubules, whence results enlargement of the whole organ. The man before you presents no evident symptoms. He has no general dropsy. There is, however, a little swelling below the eyes, to which I am glad to be able to call your attention, as it is a symptom the importance of which is often exaggerated. Such swellings occur quite independently of Bright's disease, and some persons are particularly prone to them. Much more serious is a swelling of the upper eyelid, although this also occasionally occurs irrespective of disease.

Notwithstanding the fact that there are no evident objective signs of illness in this case, one may still be led to suspect it by an examination of the heart, even before examining the urine. No murmur is heard, but on placing the stethoscope on the aortic cartilage we note easily a sharp accentuation of the aortic second sound, whence at once hypertrophy of the left ventricle may be suspected. That such hypertrophy is present is proven if an enlarged percussion-area of the heart

downward and to the left can be demonstrated, or if there is displacement of the apex below and to the outside of its normal situation between the fifth and sixth ribs within the nipple-line. Such a state of affairs exists in this case. I will only add that it is not always discoverable, because a coexisting pulmonary emphysema may obscure it. Such a state of affairs always demands an examination of the urine, which one is almost sure to find albuminous, although the quantity of albumin will probably be small. Rarely also albumin will be found altogether absent. As in this case, too, you will most likely find a few small hyaline or slightly granular casts, or casts containing one or two small oil-drops; casts also may be wanting, but careful searching will seldom fail to find them. Finally, the twenty-four-hours' urine will be found increased to fifty, to sixty, and even to seventy ounces, and the specific gravity will be lowered, 1005 to 1012. Such a combination of symptoms—hypertrophy of the left ventricle, no valvular disease, small albuminuria, a few hyaline or pale granular casts, no dropsy—admits of but one interpretation,—chronic interstitial nephritis. Such a state of affairs may continue for a long time and the patient may be but slightly ill. Or there may be headache, throbbing in the head, morning sickness, and symptoms of indigestion.

I.

We have, then, in this case an association of kidney-disease with heart-disease. The former is interstitial nephritis, the latter hypertrophy of the left ventricle without valvular disease. Now, what is the order of these events? Without attempting to decide the question in the case before us, I may say that modern studies have made it pretty certain that this form of combined heart-disease and kidney-disease may occur in two ways: first, both conditions may result from one and the same cause; or, secondly, the heart-affection may be secondary to the kidney-disease and its direct consequence.

Whichever may be the more common,—and I do not think this is certainly determined,—the first is the easier of explanation. For this combined kidney-affection and heart-disease, a thickened state of the blood-vessel wall, known as arterio-sclerosis or arterio-capillary fibrosis, is held responsible. The thickening is more or less general, or diffused throughout the arterial system, but is more marked in the smaller vessels, and it is not confined to any one of the three coats. Usually it begins as an endarteritis, and the intima is therefore thickened, particularly as to its subendothelial connective tissue, but the muscular coat is also the seat of morbid changes not always the same.

Gull and Sutton, in a paper which has become historic, announced in 1872[1] that the changes in the muscular coat were chiefly of an atrophic character, and, although the methods of these observers have been much criticised, the most recent studies on this subject by Councilman and Arthur V. Meigs go to confirm their conclusions both as to the seat and the nature of the changes. Councilman[2] finds atrophic changes in the muscular coat, including greater or less destruction of the muscular fibre-cells, and the formation of a homogeneous hyaline tissue invading both coats, but especially the intima, where it produces decided thickening, and encroachment to a varied extent upon the lumen, sometimes amounting to occlusion. The capillary walls are likewise thickened, and sometimes, especially in the glomerule of the kidney, obliterated. Meigs's[3] studies also find these changes for the most part confined to the intima, which is decidedly thickened. To a less extent the intima of the veins is similarly involved. The picture of the changes thus briefly described may be obtained from a small artery taken almost indifferently from any tissue or organ of the body,—for example, from the muscular substance of the heart, the kidney, or the liver.

It is to be remembered, also, that there are at least two other forms of endarteritis, the first the nodular, where the changes are limited to small areas in the aorta and large arteries,—atheromatous patches, sometimes calcareous and sometimes fatty; the second the so-called senile endarteritis, in typical instances of which the aorta and all its larger branches are converted into rigid inelastic tubes. It is doubtful whether the latter should be called endarteritis, it being rather an infiltration of lime-salts as the result of impaired nutrition. It begins in the muscular coat, and the vessel-walls are thinned rather than thickened, their inner surfaces roughened, and their lumina irregularly dilated, while the vessels themselves are elongated, producing abnormal tortuosities. Far from being enlarged in this form, the heart is often smaller, the seat of brown atrophy. Similar atrophic processes affect the liver as well as the kidney, and the result in the latter organ is often a typical contracted kidney. The rationale of the renal changes in senile endarteritis is similar to that in the diffuse form. The cardiac hypertrophy is, however, absent, because the nutrition of the heart is so seriously interfered with that it cannot exert its usual reactive influ-

[1] Arterio-Capillary Fibrosis, Medico-Chirurgical Transactions, London, 1872.

[2] On the Relations between Arterial Disease and Tissue Change, Trans. Assoc. Amer. Phys., vol. vi., 1891.

[3] New York Medical Record, July 7, 1888.

ence. Hence it undergoes atrophy at the same time with the liver and kidney.

It cannot be said that these three varieties of endarteritis are always sharply separable one from the other, but it is the diffuse form which is the link between the hypertrophy of the left ventricle and the contracted kidney present in the case before us. Of its consequent changes the hypertrophy of the ventricle is most easily explained. The resistance in the blood-vessels stimulates the ventricle to increased effort, and there result increased arterial tension and hypertrophy. The degree of cardiac hypertrophy is sometimes enormous, the organ weighing as much as eight hundred and fifty grams, or twenty-eight ounces, and the average in twenty-seven cases studied by Councilman being over four hundred grams, or thirteen ounces.

The alterations in the kidney, which vary greatly in degree, being sometimes scarcely noticeable and sometimes extreme, are the direct result of an interference with its nutrition. The blood-supply to the renal elements being cut off, these gradually waste and ultimately disappear. The cells and tubules thus destroyed are gradually but irresistibly replaced by fibrous connective tissue, in obedience to the pathological law elaborated by Weigert, that parts destroyed are partially replaced by cicatricial connective tissue. This contracts and reduces the size of the kidney, and perhaps, also, in this contraction further destroys the proper kidney structure and thus augments the atrophy.

It has been said that in this form of combined kidney- and heart-disease there is no cardiac murmur. Nor is there, as a rule. It is not impossible, however, for the endarteritis of which we are speaking to creep along the walls of the aorta until it reaches the aortic valves and so structurally changes them as to make them rough or incompetent and give rise to murmurs.

The causes of this form of Bright's disease are, therefore, the causes of the endarteritis, and these are various. Among the most numerous are habitual excess in eating and drinking, the poison of gout, whether uric acid or something else, lead-poisoning, and syphilis. All of these conditions introduce an irritant substance into the blood which in the course of its circulation excites an inflammation of the inner membrane of the vessel. To a less degree probably the specific causes of all the infectious diseases must be included in this category,—possibly, even, malaria. The subjects are usually middle-aged men, between the ages of forty and fifty-five, but they may be younger. Councilman has found these changes more common in the negro than in the white race.

I am, however, one of those who do not believe that every instance of renal disease associated with cardiac hypertrophy is the result of an intermediate endarteritis. Interstitial nephritis is not the only form of renal disease which is associated with cardiac hypertrophy, although it is the variety most frequently thus associated. Any case of chronic nephritis is liable sooner or later to become associated with hypertrophy of the left ventricle, while pronounced contraction of the kidney may occur without general vascular change sufficient to explain the contraction.

How is the cardiac hypertrophy to be accounted for in this second set of cases? It must be admitted that the explanation is not so easy as in that where chronic endarteritis is present. I will first review the theories which have been from time to time given, for theories alone they must be acknowledged to be. The oldest, which may be termed the "chemical theory," was advanced in its cruder form by Bright himself, whose acute observation had not failed to notice the association of cardiac hypertrophy with the disease so deservedly coupled with his name since 1827. According to this hypothesis, the retention of excrementitious substances in the blood is responsible for the increased arterial tension and the hypertrophy of the left ventricle. At the present day George Johnson in England and Senator in Germany still hold this view. At one time Johnson held that the hypertrophied state of the muscular coat was the effect of its resistance to the onward movement of the noxious blood, while the coat thus thickened further reacted upon the effort of the heart to overcome it. More recently he ascribes this hypertrophy of the middle coat in the vessels of the kidney to an effort to cut down the supply of blood in accordance with the reduced demands of the small kidney. Recent studies by modern methods do not find the changes in the middle coat early described by Johnson, while experiments which have for their object charging the blood with urea and allied excrementitious substances have also failed to excite hypertrophy. It should be added, however, that it is not possible by experiment to produce precisely the conditions furnished by diseased kidneys, especially in the matter of duration. It is very generally conceded that uræmia is due to retained excrementitious substances which are in health eliminated by the kidney; while the experimental introduction of these same materials has as yet failed to produce uræmia. Here, too, it is to be remembered that sound kidneys are encountered, which by copious diuresis promptly eliminate the substances introduced.

The so-called "mechanical theory" of cardiac hypertrophy was

advanced by Traube, whose researches in 1856 gave a decided impulse to clinical study of the subject. According to Traube, the increased arterial resistance was caused by two supposed states: the first being an over-fulness of the vessel because of the diminished withdrawal from the blood of water for the formation of the renal secretion; and the second, that the movement of arterial blood into the kidney was hindered by the renal contraction itself. The first hypothesis was erroneous in the case of contracted kidney, where the urinary secretion is really increased, and the second is opposed by the fact that even ligature of the renal arteries fails to increase arterial pressure, because of the ample space elsewhere to take up the blood thus diverted. Nor did Cohnheim's further elaboration of the mechanical theory, which located the increased resistance more precisely behind the wasted glomerule, give any more permanent life to it.

I incline to the belief that the difficulties are best met by supposing the primary changes in the heart to be *compensatory* in their nature, set up with a view to making up for the gradual loss of renal substance. Such an action is paralleled everywhere in the physiological economy. Nowhere do we meet with loss of function which is not at once met by an attempt of nature to supplement it. The dependence of the urinary secretion upon cardiac pressure is well understood, and an increase of cardiac power is the most reliable means available for stimulating the action of the kidneys, when desired, in therapeutics. The diuresis which is so constant a symptom of the contracted kidney is certainly the direct result of a supplemental contraction of the left ventricle, which it is reasonable to suppose is induced for the purpose named, and results in hypertrophy.

This view receives confirmation in the subsequent course of the disease. So long as the free secretion which is the result of the compensatory action of the heart is kept up, so long the patient remains tolerably comfortable, and perhaps even for a time unconscious of the presence of disease. But an organ thus overgrown is apt sooner or later to suffer in its nutrition, and especially is this the case if its arteries be the seat of an endarteritis which interferes with the free movement of the blood and produces also fibro-myocarditis. And what are the further consequences? The strong propulsive power of the heart declines, the pulse falls away in tension and power and becomes more frequent and sometimes irregular. The urine secreted diminishes in quantity and assumes a darker hue. Fortunate is the patient if the specific gravity of the urine rises *pari passu* with its reduced quantity, as it indicates that the normal quantity of solids is kept up. Too frequently, however, this is not the case, and excrementitious substances accumulate in the blood, laying the foundation for uræmia. Headache, nausea, a foul and even urinous breath, may be superadded, and uræmia set in, preceded by drowsiness, or it may be ushered in suddenly with convulsions. Or another set of symptoms may supervene. The patient becomes short of breath, first on slight exertion, and later this very distressing symptom occurs without such exciting cause. This sort of asthma is often spoken of as uræmic asthma, as if due to the same causes as uræmia, and this may sometimes be the case. More frequently the failing heart is responsible. The organ is no longer able to move the blood onward, the lungs become engorged, aëration becomes imperfect, and hence the dyspnœa. For a time this symptom may be averted by whipping up the heart by cardiac stimulants, and the right ventricle even comes to the rescue for a time, and hypertrophies in its effort to overcome the now disturbed compensation. Subsequently this as well as the left ventricle may become dilated, and œdema of the lungs set in, with annoying cough and serous frothy expectoration, sometimes blood-tinged. Nor does general dropsy continue absent, but ensues sooner or later with the growing heart-failure. Our resources are now almost at an end, but are not exhausted, as even these symptoms sometimes subside.

Finally, it must be stated that not every case of interstitial nephritis is attended with hypertrophy of the left ventricle. In addition to the cases of contracted kidney from senile endarteritis already referred to, cardiac hypertrophy is apt to be absent in the interstitial nephritis of the weak and cachectic.

A pertinent question is one as to diagnosis,—as to whether it is possible to say of a given case that it is one of endarteritis with consequent renal cirrhosis and cardiac hypertrophy, or one in which the renal condition is primary. It is true that some symptoms may be referred to the vascular and others to the renal cause, and it is not unimportant to be able to recognize early the symptoms of arterial sclerosis before those of renal involvement make themselves apparent, since the latter indicate a more irremediable stage. Thus, the early head symptoms, such as headache and vertigo, are probably due to derangements in the circulation in the brain, while the apoplectic phenomena which often terminate the disease—when other symptoms would not lead us to expect the end—are directly due to defects in the vessel walls associated with the extreme intravascular pressure. A throbbing sensation in the head is included in the symptoms ascribed to intravascular pressure, and it is reasonable to suppose that when this occurs associated with

vertigo and without objective symptoms of Bright's disease, such as albuminuria and casts, it is due to primary vascular change. Late in the disease, when the objective symptoms are well defined, it may be ascribed to secondary hypertrophy. When the arcus senilis attends these two symptoms, vertigo and throbbing, there is probably endarteritis, although the arcus is more frequent with senile arteritis. There is no peculiarity in the vertigo by which it may be assigned to one or the other condition. It may be transitory, or may last long enough to force the patient to sit down, or even to cause him to fall, though even then the duration is apt to be for a few minutes only. A slow pulse is apt to be associated with these vertigoes, and sometimes an extremely hypochondriacal state, all prior to the albuminuric stage. A tendency to a mild form of epileptic seizure is described by the French authors.[1]

Epistaxis is another symptom which may be directly ascribed to the arterio-sclerosis, and it is often the first symptom to attract attention by its profuseness and frequent recurrence. On the other hand, palpable stiffening of a blood-vessel wall attainable by the finger is more apt to indicate senile endarteritis. High arterial tension as detected by the sphygmograph before it is noticeable by the finger, and before any signs of cardiac or renal disease present themselves, points to endarteritis.

There seems every reason to believe that the prognosis of arterio-sclerosis is more favorable before the renal and cardiac changes manifest themselves. It is desirable, therefore, that it should be discovered as early as possible; and attention to the facts named may lead to this.

The treatment of the combined kidney- and heart-disease is not different from that of interstitial nephritis. Even greater rigidity is necessary in eliminating nitrogenous food, and all food should be reduced to a minimum. A diet of milk diluted with water is the safest of all. Bread and butter may, however, be allowed, and even succulent vegetables easy of digestion, such as potatoes, peas, and string beans, while simple fruit-juices, as those of oranges and lemons, are allowable. Mental excitement and immoderate muscular exertion must be avoided, and the heart should not be overworked in any way.

The medicinal treatment especially directed to the arterio-sclerosis may be divided into that intended for the cure of the endarteritis and that directed to the relief of symptoms. The only drug from which results may be expected for the former purpose is the iodide of potas-

[1] Grasset, Du Vertige Cardio-vasculaire, Paris, 1890.

sium, which should be given a fair trial in doses as large as can be borne without deranging the stomach. For the relief of the symptoms more especially due to the sclerosis—viz., headache, throbbing, and vertigo—the nitrites are often useful, and this in my experience is their sole use in Bright's disease. Nitro-glycerin should be given in doses of one-hundredth of a grain every four hours, rapidly increased to one-fiftieth, as the smaller dose is often without effect. The aim should be to produce the physiological effect, which is a sense of fulness or a flushing. The sodium nitrite may be substituted in three-grain doses. It has the advantage of being more permanent in its effect, although it is slower in its action.

II.

The second form of combined heart- and kidney-disease to which I wish to call your attention is that wherein the heart-disease is primary. It is commonly disease of the mitral valve. The kidney-disease does not occur in connection with aortic valvular disease until mitral disease is superadded. It is well known that in mitral regurgitation, as soon as compensation ceases the blood accumulates first in the lungs, then in the right side of the heart, and finally in the venous system, engorging especially the liver, the stomach, and the kidneys. With the effects upon the first two I have nothing to do at present, although they are of a very positive character and generally manifest themselves sooner than the renal symptoms.

Let us first study the condition of the kidney itself. As stated, it is engorged with blood from the venous side. The renal vein and its branches are filled with blood as far back perhaps as the Malpighian body. This backward pressure resists the onward flow of the arterial blood, and the congestion is thereby further augmented. The effect is to swell the kidney somewhat and to darken its hue, which on section will be found more intense in the pyramids. A slight hardness also results, and the combination of these two conditions, color and density, has suggested the term *cyanotic induration* for such kidneys. Further changes are not marked or constant. There is sometimes a slight overgrowth of the interstitial connective tissue, and a slight tendency to degeneration in the cells.

Much more decided are the clinical phenomena resulting from such congestion. It is a well-recognized condition of copious secretion of urine that the blood should move freely through the kidney. A stasis is followed immediately by diminished filtration of water, the twenty-four-hours' quantity being reduced to from thirty to twenty ounces, and

even to less. As the solids at first at least remain the same, the urine is dark-hued, the specific gravity is high, the reaction is markedly acid, and a copious sediment of urates and uric acid makes its appearance as soon as the urine cools off. There is almost always a small amount of albumin found. Casts are sparsely, if at all, present, and are of the hyaline and faintly granular variety. Both red and white blood-corpuscles are also sometimes detected, as might be expected. This condition of the kidney and the symptoms are the direct results of the cardiac valvular disease. They may also be produced by any cause producing venous stasis, as pulmonary emphysema, chronic pleurisy, and thrombosis of large veins. Their effect is further to augment the symptoms of the cardiac disease. The circulation, already everywhere obstructed, is further impeded, there is dyspnœa, dropsy increases, the appetite fails, there are nausea and constipation. Sleep, already disturbed by dreams, becomes more so, and a more distressing picture than is presented by such a case is rarely met.

Yet these symptoms are often easily amenable to treatment so long as the heart-muscle remains capable of being influenced by digitalis. I have seen many a patient, apparently *in extremis*, gasping in orthopnœa and with legs heavy and almost bursting with dropsical effusion, completely relieved by a few large doses of this drug. But they must be large doses, not less than eight to ten minims or fifteen to twenty drops every three hours until an effect is produced. If digitalis fails, the tincture of strophanthus may be given in the same doses, or caffeine citrate in three-grain doses, each every four hours, or sparteine sulphate in doses of one-quarter to one-half grain. Purgation should not be omitted, but should rather be pushed to the production of watery catharsis. The ingestion of fluids should be restricted, and the Hay's treatment of dry diet with purgatives is sometimes useful. But I have found a restricted milk diet, limited to two ounces every two hours, associated with the drugs above named, more efficient.[1]

Diuretin is a remedy from which good results may be expected in these cases. My own experience with it has not been large, but I shall use it more in the future. The dose is from seventy-five to one hundred grains daily in solution, about fifteen grains being administered at a time in a tablespoonful of water. The rapid increase of the twenty-four-hours' urine from five hundred cubic centimetres to three thousand three hundred cubic centimetres is reported from its use.

[1] See some cases reported by the writer in a paper "On the Management of Obstinate Dropsies," Medical News, June 21, 1890.

Such a kidney is of course liable to become the seat of an acute or a chronic nephritis.

III.

The next form of kidney-involvement to which I call your attention as being secondary to disease of the vascular apparatus, commonly heart-disease, is more frequently seen on the post-mortem table than recognized in the living subject. It is *embolic infarction*, produced by the lodgement in some branch of the renal artery of an embolus derived from the heart or a blood-vessel. Its most frequent source is a fragment of vegetation or clot from a diseased heart-valve. An embolus may also arise from a thrombus in an artery, but more frequently it is caused by one in a vein. If from the latter, it must be carried first to the right heart and thence through the lungs into the left heart, and thence by the aorta to the kidney, and must of course be small.

The effect of the lodgement of an embolus in the kidney is a wedge-shaped hemorrhagic infarct, which in time whitens, contracts, and is ultimately absorbed, leaving a mere cicatricial mark.

Most frequently a renal infarct occurs without noticeable symptoms. Its occurrence, if looked for by reason of the presence of valvular heart-disease, might be ushered in by the sudden appearance of a small amount of blood in the urine. A sudden pain in the region of the kidney simultaneously occurring would go to confirm the diagnosis. No treatment is indicated, even if the event is recognized.

IV.

Finally, kidney-disease and disease of the vascular apparatus, and especially cardiac disease, may coincide accidentally, each the result of its own cause, and reacting the one upon the other in various degrees and variously aggravating the symptoms of each, so that it often becomes a very nice question to settle which is the preponderating disease. Fortunately, this difficulty does not always extend to therapeutics, the same remedies which are useful to one affection being commonly indicated for the other. A careful study of each case should, however, be made on its own merits, and due weight assigned to each factor of the disease.

CIRRHOSIS OF LIVER AND KIDNEYS; STENOSIS OF PULMONARY ARTERY; ACUTE PLEURISY; LOBAR PNEUMONIA WITH PLEURISY.

CLINICAL LECTURE DELIVERED AT THE BELLEVUE HOSPITAL.

BY ALFRED L. LOOMIS, M.D.,

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Reported by Dr. Ogden C. Ludlow.

GENTLEMEN,—The first patient which I present to you to-day is a married woman, forty years of age, who has had four children, and who has enjoyed good health previous to the present illness. She says that she drinks "very seldom," but, on further questioning, I find that she means by this "a little every day." She is accustomed to take whiskey-punch on going to bed, and occasionally also in the morning, and has been in the habit of doing this for the past five years. Her present trouble began with a slight swelling of the feet, which would be present on rising in the morning, and which was not painful. Almost at the same time her abdomen began to enlarge, and this has steadily increased, although the œdema of the feet has subsided. She has also had vomiting in the evening, and "dry retching" in the morning, which would be relieved by a little hot whiskey and water. Jaundice has developed gradually along with the other symptoms.

This history suggests a diagnosis of cirrhosis of the liver and kidneys.

On inspection, we find the abdomen uniformly enlarged; the sclerotic of the eye deeply tinged with yellow; the tongue dry and red at the tip, and covered with a light coating on either side. The skin is of a dark yellow color, such as indicates chronic jaundice due to some disease of the liver which gives rise to more than simple obstruction of the gall-duct. On closer inspection, you will notice that the superficial veins of the abdomen are enlarged, denoting ob-

struction of the portal veins. The abdominal enlargement which is present in this case might be due to gas in the intestines or to fluid in the peritoneal cavity; the question to decide is whether it is due to gas or fluid. On palpation, fluctuation is readily obtained, but this is not sufficient to establish the presence of ascitic fluid. You must change the position of the patient from one side to the other, and observe whether the portion which is uppermost is tympanitic on percussion, while the most depending portion is flat. On examination we find this to be the case, except that on the right side the percussion note instead of being flat becomes dull, due probably to the fact that a portion of intestine is adherent at this point. The abdominal distention makes palpation of the liver quite difficult, but it seems to be slightly irregular. Percussion shows hepatic dulness about twice the normal area in the line of the right nipple; over the left lobe it is relatively greater, for the dulness in the median line is fully four inches greater than normal. There is flatness over an abnormal area in the region of the spleen, denoting either fluid in the left pleural cavity or enlargement of the spleen. On palpation, the spleen can be distinctly felt below the free border of the ribs. The apex-beat of the heart is about one and a half inches above its normal level; the sounds are very distinct; the heart's action is rapid. There is no murmur. Pulse is 120, small and regular. There is no elevation of temperature. On percussion of the chest posteriorly, there is found an increased area of flatness on both sides, corresponding to the liver on the right and the spleen on the left, so that both liver and spleen are enlarged.

Fatty changes are sometimes associated with the cirrhotic or fibroid changes which occur in hepatic fibrosis, and under such circumstances the liver may be enlarged, a condition called fatty hypertrophic cirrhosis. The connective tissue, hepatic cells, and the radicals of the bile-ducts are all involved, and there is also obstruction of the portal circulation sufficient to cause ascites: with the enlargement of the liver there is enlargement of the spleen. Hepatic cancer is the only hepatic disease which might be confounded with this condition. This patient, however, gives a distinct alcoholic history, and the case has behaved like one of cirrhosis. It is quite probable that this disease existed for some time before the patient gave up work. The degeneration of the liver-cells and the change in the connective tissue, causing interference with the radicals of the bile-ducts, account for the jaundice.

Our second case is that of a woman, twenty-one years of age, whose family history is entirely negative as regards its bearing upon

her present condition. She has never had any acute disease except measles. She tells us that there has been some trouble with her heart as far back as she can remember, associated with pain along the central portion of the chest. She has always suffered from shortness of breath on going up- and down-stairs, and at such times she would become blue. She has been free from œdema of the extremities, with the exception of a slight swelling of the ankles. Her appetite and general condition are good. An examination of the urine shows it to be of a light amber color, acid in reaction, of a specific gravity of 1005, and to contain a mere trace of albumin. The quantity excreted is normal, and the microscopical examination is entirely negative.

On physical examination a murmur is heard, about which there have been differences of opinion. Its maximum intensity is directly over the pulmonic orifice, and it is heard to-day with the first sound of the heart, and is conveyed to the left. It is not heard in the carotids, nor at the apex, but at the apex you hear instead a distant diastolic murmur. I am told that the diastolic sound is at times more intense than at others. There is no hypertrophy of either the right or left heart. The systolic murmur has all the characteristics of one produced by pulmonic obstruction. It is perhaps one of the rarest forms of murmur, and indicates pulmonary stenosis. From the history of the patient it would seem that the change which produces this murmur in this patient is an hereditary one. When she exercises or has a paroxysm of coughing, she gets quite blue, and even now her fingertips and lips show this color. Stenosis of the pulmonary artery is usually due to arrested development during uterine life, and it is usually associated with a patent foramen ovale or an imperfect intraventricular septum. In some instances the lesion is very extensive, and the child dies immediately after birth. In cases in which the lesion is slight, as in this case, adult life is reached, and the symptoms of heart-disease are intermittent. These cases are rather curiosities of medicine than of practical importance.

Our third patient is a boy, who previous to the beginning of his present illness, two weeks ago, was in unusually good health. His father died of consumption, and his mother of the "grippe." His brothers and sisters are healthy. He has never been sufficiently ill before to be confined to his bed. The present trouble began with dizziness and "a short chill," and the physician attending him at this time told him he had the grippe. Within twenty-four hours from the beginning of this attack he experienced pain in the right side. There was no expectoration at first, but subsequently he had an abundant

yellowish expectoration, as at present. There has been at no time a high temperature, and no spitting of blood.

This patient's history is not exactly what we would expect in a case of acute pulmonary tuberculosis or acute pneumonia. The boy was perfectly well up to the time of this illness,—in fact, he said he was gaining flesh,—yet he suddenly developed active symptoms which could be referred to some acute disease of the lungs or pleura. Many of his symptoms unquestionably point to acute pleurisy, for he has had constant and severe pain in his side, increased by deep inspiration. But with this pain there has been a cough, with muco-purulent expectoration. There must be present then something more than a pleurisy. One of the students has wisely suggested that this patient has an irregular type of pneumonia. There are several irregular forms of pneumonia. Occasionally we meet with a lobar pneumonia that is slow in its development and in its resolution, and frequently leaves behind it more or less permanent lesions in the lungs. This is sometimes called chronic pneumonia. This form of pneumonia is most frequently met with in the aged, and usually complicates some chronic disease. The term "chronic pneumonia" is a very unsatisfactory one, for it may mean interstitial pneumonia, chronic lobar, or chronic lobular pneumonia; ordinarily, however, the term is applied to interstitial pneumonia. Another irregular form of pneumonia is a bronchopneumonia, which develops in an unusual way on account of some peculiar infection. This is the kind of pneumonia which is occurring so frequently with epidemic influenza. They are not frank lobar pneumonias, nor are they typical lobular pneumonias. They are accompanied by more or less pleurisy; they often involve a whole lung, the development being slow and progressive. The characteristic expectoration of pneumonia is never present; the temperature is never very high; the characteristic pneumonic countenance is absent, as well as all the other ordinary symptoms which are regarded as characteristic of acute lobar pneumonia. Physical signs indicate lobar consolidation, but the rational signs are against lobar pneumonia. These pneumonias are very slow of resolution, the physical signs of pulmonary consolidation often continuing for weeks, causing one to suspect acute pulmonary tuberculosis.

Now let us examine this patient. He is of the age and presents the appearance of one whom we should expect to have pulmonary tuberculosis. I have told you that the temperature in those irregular pneumonias to which I have referred never goes very high. This patient's highest temperature has been 102° F., and it has ranged

between 99° and 101° during the four days he has been in the hospital. This is about the range of temperature which I have seen in similar cases of so-called "grippe pneumonia": it is not the temperature you would expect to find in tuberculosis. He is anæmic, the eyes are clear, respiration is not accelerated, and there is a full expansion of the upper part of the chest, more marked on the left side than on the right. His pulse is now about 90, and is of fair quality. We notice particularly that he has that fair skin which suggests anæmia, yet his lips are red and contradict this first impression. The apex of the heart is in its normal position, and is beating normally. There seems to be very slight dulness in the left infra-clavicular region. On auscultation, the only difference noted in the respiratory murmur in this region on the two sides is, that on the left it has its normal "breezy" quality, while on the right side it approaches "rude respiration." You notice that even while talking he catches his breath and coughs; the voice is so feeble that vocal fremitus is not of much help in this case. The percussion note over the left side posteriorly is extra-resonant; on the right side there is dulness over the scapular region, which indicates pulmonary consolidation, and a flat note over the lower portion, such as we should expect to get over fluid or a thickened pleura. Just at the border of the scapula and under it on the right side, there is distinct bronchial expiration, which is not intense, and there is some vesicular element on inspiration. As you go a little lower you get typical bronchial breathing. When he coughs, a few crackling râles are heard, having a metallic quality. As you pass farther downward the bronchial breathing becomes less and less distinct, until it is scarcely audible at the base of the lung. You also get bronchophony until you reach the point where you began to lose the bronchial respiration, and then the voice-sounds seem distant. We have then the physical signs of consolidation in the upper and central portions of the lung; in fact, over all the posterior portion of the chest there is a bronchial character to the breathing, and in the central portion pure bronchial respiration. Very few râles are heard at any point over the chest, and those which are heard are over the central portion, corresponding to the area of most intense bronchial breathing. We are now prepared to make a diagnosis. From the history and temperature record I should exclude pulmonary tuberculosis, nor can it be regarded as chronic pneumonia according to the definition of this condition which I have already given. I regard it as a straightforward lobular pneumonia with pleurisy which has involved the entire lung posteriorly,—in other words, a broncho-pneumonia complicated by pleurisy. There is, of course, a

chance for a difference of opinion. Should an examination of the sputum show tubercle bacilli, I would certainly relinquish my diagnosis, but I do not think tubercle bacilli will be found, for I have seen a number of similar cases within the last few months, all commencing with the symptoms of epidemic influenza.

[Repeated examinations of his sputum did not reveal the presence of tubercle bacilli. At the expiration of two weeks he was completely convalescent and left the hospital.]

THE CONSEQUENCES OF ATELECTASIS PULMONUM OR ALLIED CONDITIONS, AND THE DIAGNOSTIC DIFFICULTIES THEY CAUSE WHEN COMPLICATING OTHER DISEASES.

DELIVERED AT A MEETING OF THE EAST SURREY DISTRICT OF THE METROPOLITAN COUNTIES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

BY JOHN SYER BRISTOWE, M.D. LOND., LL.D. EDIN., F.R.C.P.L., F.R.S.

Incompleteness or want of expansion of the lungs at birth, or atelectasis, and lobular pneumonia in early life, such as results from measles, whooping-cough, and under some other conditions, lay, as is well known, the foundation of chronic degenerative changes in the lungs, of which the main anatomical factor is dilatation of the bronchial tubes, or bronchiectasis, and the main clinical indications are the symptoms of chronic bronchitis.

There are two varieties of dilated tubes met with in these cases,—the first, and more common, being that in which the tubes generally, or mainly the smaller branches, are expanded into cylindrical or moniliform channels, the arrangement and general appearance of which remind one of the fingers of a glove; the other being that in which the terminal portions only of the bronchial tubes are dilated, forming rounded cavities from the size of a filbert downward, with each of which an undilated bronchiole communicates. In the former condition the dilated tubes are separated from one another by crepitant lung-tissue. The latter condition is generally limited to one lung or to a single lobe, and the cavities are surrounded and separated from one another by a comparatively small amount of indurated fibroid tissue which represents what formerly was lung-tissue. In the one case the flask-like cavities appear to become developed in lungs or parts of lungs which either have never expanded or have undergone complete collapse or consolidation; in the other case the lesion would seem to arise in the midst of lung-tissue still subserving respiration. But in

both cases there is reason to believe that the early stage of dilatation is due to the accumulation of muco-purulent fluid in the tubes and their gradual distention thereby, and that the later stages are due partly to the continuance of this cause, partly to the stretching influence of inspiration over the already existing cavities.

It is not an uncommon thing to meet with persons, for the most part young, who have suffered, either from birth or from early childhood, from more or less continuous cough and muco-purulent expectoration, and present some degree of cyanosis together with the local indications of bronchitis, and in whom these phenomena are due to the condition of the lungs in question. In some of these cases, as might be supposed, the patients remain undersized and ill developed, present clubbed fingers and toes, and the cyanosis is as intense as it is in typical cases of congenital heart-disease. My object on the present occasion, however, is not so much to consider the pathology and symptoms of the morbid state to which I have just directed your attention as it is to narrate briefly a few cases which have come under my notice, wherein its association with some other pathological condition has interposed a difficulty in the way of accurate or ready diagnosis and rendered them specially interesting.

Case I.—The first case occurred more than thirty years ago, and I have been unable to lay my hands on the notes that were made of it at the time. But the essential facts, so far as concerns my present paper, remain vividly in my memory. One day, while in the wards, I was requested, in the absence of the physician under whose care he was, to see a patient in whom urgent symptoms had arisen. He was a man between thirty and forty years of age, who was suffering from the usual symptoms of acute meningitis, and who was lying in bed on his left side insensible and with retracted head. I examined him carefully so far as I was able, satisfied myself that he was dying of intracranial inflammation, and discovered that over the whole of the right side of the chest, which was uppermost, there were loud gurgling sounds such as might have resulted from extensive breaking down of lung-tissue. The man was too ill to allow of a thorough examination; and no history had been, or could be, obtained as to his state of health prior to the recent supervention of his fatal malady. Guided largely by the apparent extensive breaking down of the tissue of his lung, I came to the conclusion that he was suffering from tubercular meningitis coming on in the course of advanced or acute pulmonary phthisis. He died shortly afterwards, and the next time I saw him was in the post-mortem room. The examination of his head

revealed extensive recent meningitis, but there was no trace of tubercles. The right lung was studded from apex to base with globular cavities, varying, roughly speaking, from the size of a filbert to that of a pea. It presented, in fact, a well-marked example of the second form of dilated tubules to which I have already called attention. The other lung was essentially healthy. There were no tubercles in either lung. So far as I recollect, no further disease was discovered. Of course I was mistaken in my diagnosis; nevertheless, I do not feel particularly ashamed of my error. I regarded the case by the light of the post-mortem examination, and I regard it still, as one in which there was the accidental association of recent simple meningitis with long-standing disease of one lung, probably arising out of the non-expansion of the organ at the time of birth.

Case II.—My second case was that of a girl, eighteen years of age, who came into St. Thomas's under my care on March 14, 1889. The history she gave of herself was as follows. She had never been strong. When about twelve she had had a severe attack of measles, followed by bronchitis and inflammation of the lungs, which with relapses had laid her up for three months. She had ever since suffered from shortness of breath and a winter cough attended with thick expectoration, and once or twice had had attacks of what was called inflammation of the lungs. Her last attack of bronchitis had come on in the previous November, and had lasted ever since. She had been especially ill during the last fortnight. She had never spat blood or had night-sweats, and had not lost flesh. She was an undersized, pallid, round-faced, expressionless person, having the aspect and development of a girl of thirteen or fourteen, and a manner and look that suggested some feebleness of intellect. The terminal joints of the fingers and toes were bulbous. She looked ill, and complained of dyspnœa and of cough attended with abundant muco-purulent expectoration. The chest was rounded, and moved only slightly during respiration. It was generally highly resonant. Expiration was prolonged, and both expiration and inspiration were attended with rhonchi and subcrepitation. The heart's area of dulness was ill defined, but its position and sounds were normal. Her tongue was coated, her appetite poor, her bowels regular. Nothing was found amiss in the abdominal cavity; and the urine, which had a specific gravity of 1020, was free from albumin.

The history, the symptoms, and the results of examination were suggestive of chronic bronchitis; and the whole aspect and development of the girl pointed either to its congenital origin or to its super-

vention in childhood. After careful consideration, I came to the conclusion that she was suffering from chronic bronchitis associated with the presence of dilated tubes.

Four days after admission she had hæmoptysis, for the first time in her life, bringing up suddenly about four ounces of blood; and subsequently this frequently recurred in varying, but never in large, quantities. Her cough also continued very troublesome and attended with copious muco-purulent expectoration; and she suffered from constant dyspnœa. The results of the physical examination of her chest, though they varied from time to time, were never decisive as to the condition of things within. There were always generally distributed rhonchi and crepitations, which latter were mostly coarse, but sometimes and in some situations were fine. It was thought that the right side became a little duller than its fellow, and perhaps moved slightly less; and it was also thought that the vocal resonance and fremitus at the apices were more pronounced than they should be, and that occasionally the respiratory sounds were tubular in quality. But there was never clear evidence of the presence of pleuritic effusion, of definite consolidation, or of the formation of apex-cavities. The temperature, however, became high, and often varied between 102° and 105°; and she gradually became weaker and thinner and more and more seriously ill.

The coming on of hæmoptysis and of persistent febrile temperature very soon led me to reconsider my first-formed opinion, and to suspect that she must be suffering from pulmonary tuberculosis. The question then arose whether the case was one of simple bronchiectasis associated with bleeding from the congested mucous membrane of the dilated tubes, or whether it had been primarily one of bronchiectasis with tubercle supervening comparatively recently as an accidental complication, or whether her symptoms even from the beginning had been due to chronic tuberculosis alone. It was largely in the hope of being able to solve this question that the chest was repeatedly examined, but, as I have already said, without definite result. And with the same object the sputum was submitted to microscopic investigation, but no bacilli were detected. The view which I adopted was the second,—namely, that her case was one of old bronchiectasis associated with recent tubercle. I may add that during her residence in the hospital she once or twice had a little diarrhœa, and that on several occasions her urine presented a trace of albumin, but that she never had abdominal pain, or uneasiness or irritability of the bladder, or pain in micturition, or anything to suggest that there was serious disease in any of the abdominal or pelvic organs.

She died on the 20th of May. The result of the post-mortem examination was very interesting and instructive. Both lungs, but the right one more extensively than the other, were bound down by old adhesions. The left was large and generally crepitant; and, although it is not so stated in the post-mortem notes, I believe that some of the bronchial tubes connected with its lower lobe were dilated. The right lung was smaller and heavier than the other, and, though not solid, gave the impression of having been the seat of former consolidation and of having never perfectly recovered. Many of its bronchial tubes were dilated and intensely congested, and a few contained altered blood. Both lungs were studded with tubercles varying in size from that of a small pea downward, the larger being cheesy, the smaller gray. They were most abundant at the apices, but nowhere had induced continuous consolidation or undergone softening. The heart was healthy.

The left kidney was the seat of chronic tubercles in an advanced stage. The calyces were greatly enlarged, and presented ragged and breaking-down parietes; and the substance of the organ, but mainly the parts immediately bounding the calyces, was infiltrated with abundant cheesy tubercular deposit. The mucous membrane of the ureter and of the bladder presented a beautiful example of the tubercular process in an early stage. It was studded with circular areæ from the size of a sixpenny piece downward, which were not unlike patches of cutaneous ringworm or erythema circinatum. They were either slightly elevated rosy disks, or broad, slightly elevated rosy rings each encircling a somewhat depressed and eroded or excoriated, grayish or yellowish central area. The intervening net-work of mucous membrane was quite normal. The right kidney and the spleen presented a few miliary tubercles; and the lower part of the small intestine showed some small tubercular ulcers of recent origin. There was no tubercle in the uterus or the Fallopian tubes. All other parts of the body were free from disease.

The autopsy obviously confirmed the accuracy of the diagnosis which had been adopted,—namely, that the patient's disease, so far as the lungs were concerned, was in the first instance chronic bronchitis and dilated tubes secondary to some former inflammatory condition of the organs, and that the tubercular affection was of comparatively recent origin and a mere accidental complication. This latter had probably supervened during her last winter's bronchial exacerbation. The kidney-disease was of much older date, and was doubtless the source from which the lungs and other organs had received infection.

The condition of the bladder was something quite novel to me. I have, of course, seen tubercular ulceration of this part; but I had never seen it in so early a stage or presenting such characters as were observed here. Looking back on the case, I cannot help regretting that we did not examine the urine for bacilli. But, in excuse, I must repeat that, beyond the occasional presence of a trace of albumin, there were never any symptoms pointing to implication of the kidneys or of the bladder; and the albuminuria seemed to be sufficiently explained either by the indirect influence of her chronic pulmonary affection or by her nearly constant high fever.

Case III.—My third and last case came under my care on the 30th of last October, and died on the 21st of last December. The patient was a girl, fifteen years of age, who had suffered from shortness of breath and cough with expectoration ever since she was a baby. She had been always more or less livid, and of late dropsical. She had also latterly suffered much from sickness. She was an ill-developed child, presenting a universal dusky tint, with deep lividity of lips, nose, cheeks, ears, and fingers, general anasarca, chiefly marked in the lower extremities, and clubbed fingers and toes. Her chest was rounded and moved only slightly. The lungs were resonant and everywhere presented abundant and loud rhonchi. Her respirations were twenty-eight in the minute; she had a frequent cough, and expectorated much thick muco-purulent fluid. The heart's area of dulness was small; the apex-beat was just below and internal to the nipple; and there was forcible pulsation over the whole cardiac area and in the scrobiculus. There was a loud systolic murmur, best heard in the neighborhood of the apex, but audible also at the base, in the axilla, and in the back. It did not appear that any murmur originated at the base; but the second sound was louder over the pulmonic area than to the right of the sternum. The pulse was small, weak, and slightly irregular. The abdomen was large, measuring twenty-nine inches in girth, and obviously contained fluid. The liver was large and smooth, extending to within two inches of the umbilicus. Tongue furred; appetite poor; frequent sickness; bowels regular. The urine was turbid with urates, and presented a sp. gr. of 1028, and a trace of albumin.

During the first ten days she remained in much the same state as on admission; still short-breathed and livid, still coughing and spitting much thick phlegm, and still presenting the loud systolic apex-murmur; but her dropsy increased, and the girth of her belly advanced to thirty-one inches. She was then tapped; but only thirty-

two ounces of serum were removed. Nevertheless, the operation gave her some relief.

All this time I had been giving a good deal of thought to the diagnosis of her case. At first (looking to the deep cyanosis, the dropsy, the loud apex-murmur, and the life-long duration of her illness) I inclined to the opinion that she was suffering from congenital heart-disease, and that this was the direct or indirect source of all her sufferings. But as time went on I became more and more impressed with the facts that she had been suffering from specific bronchial symptoms from her earliest infancy, and that at the present time she was still suffering mainly from bronchitis, as was shown by her pulmonary symptoms, the physical condition of her chest walls and lungs, and the abundance and character of her expectoration. And then (acknowledging to myself that chronic bronchitis is not a necessary consequence of cardiac lesions, that cyanosis may be caused either by chronic pulmonary or by cardiac disease, and that it is not uncommon in cases of hypertrophy of the right side of the heart due to obstructive disease in front for regurgitation through the tricuspid orifice, and consequently a systolic murmur, to ensue) I came to think it probable that her illness had begun from atelectasis or broncho-pneumonia in early childhood, that dilated tubes with persistent bronchitic symptoms had followed, and that her morbid cardiac phenomena and cyanosis were later sequelæ of her original malady. Of course it still remained possible that malformation of the heart was present as an accidental accompaniment of the pulmonary disease.

During the rest of her sojourn in the hospital her symptoms varied somewhat, but on the whole she got worse. The ascites returned, and paracentesis was again performed. Her general dropsy increased largely, and to relieve it, Southey's tubes were inserted into the legs, and (these failing) subsequently several punctures were made from which much serum was discharged. But erysipelas attacked the left leg, much febrile disturbance ensued, and superficial sloughs formed. Nevertheless, after a few days of ingravescence, the local symptoms gradually subsided. During the last two or three weeks of her life she was extremely ill; far too ill, indeed, to allow of any careful physical examination. And, without any material change in the character of her symptoms, she died seven and a half weeks after her admission.

The following is the account of what we discovered post mortem. The body was generally œdematous; and on the dorsum of the left foot was a large area of ulceration, exposing the deep fascia. There were old adhesions, and about half a pint of serous fluid, in each pleura.

The right lung was heavy, of a uniform livid red color, smooth on section, and quite airless. The bronchial tubes were for the most part dilated to near the surface of the lung, and their walls thickened. The left lung was very œdematous, but crepitant, and its bronchial tubes were in the same condition as those of its fellow. The heart was somewhat enlarged, weighing nine ounces. The apex was formed equally by the apices of both ventricles. The left ventricle was healthy. The right did not appear to be dilated, but its walls were fully as thick and firm as those of the left, which, by the way, seemed a little thinner than natural. The right auricle was also thickened and dilated. The valves were quite healthy in structure and appearance; but I believe their competence was not tested. The abdomen contained three pints of fluid. The liver was large, weighing one pound twelve and a half ounces; its capsule was thick and opaque, and its substance tough and congested. The spleen and kidneys were large, firm, and congested. All the other organs were healthy. Looking at this case by the light thrown upon it by the post-mortem examination, there can be no doubt that there was no congenital cardiac defect, but that the patient had suffered from bronchiectasis from birth or early infancy, and that the anatomical and other peculiarities of her heart were simply the consequences of prolonged obstructive pulmonary disease.

In conclusion it may seem that the cases which I have narrated form a somewhat heterogeneous or motley group; and I admit that in a sense they do. At the same time the existence of the common factor "bronchiectasis" in all of them (forming as it were a common background, in itself interesting, to pictures otherwise dissimilar yet interesting also in their points of difference) has always linked them together in my mind. And it is because the cases have been thus interesting and instructive to myself that I have thought they might prove in some degree interesting and instructive to others.

ANGINA PECTORIS.

CLINICAL LECTURE DELIVERED AT THE POLYCLINIC HOSPITAL.

BY THOMAS J. MAYS, M.D.,

Professor of Diseases of the Chest in the Philadelphia Polyclinic, and Visiting Physician to the Rush Hospital for Consumption.

GENTLEMEN,—Angina pectoris, or stenocardia, is a paroxysmal disease which is characterized by intense pain in the region of the heart, a sense of suffocation, a fear of impending death, and very frequently a slow pulse. The pain usually radiates from the chest into and down the left arm, although sometimes it extends down both arms. It is a disease which, like asthma, epilepsy, and migraine, has a tendency to manifest itself in the early hours of the morning, and after death, in many cases, there is found more or less degeneration of the cardiac muscle, of its arteries, and of its nerves.

Now, what is the intimate nature of angina pectoris? Has it a local habitation in the heart, or does it extend throughout the whole arterial circulation? Those who believe that it is a strictly cardiac affection pin their faith on the knowledge that the heart is the principal organ which bears any marked evidence of degeneration after death. This is in great part true, but it must be remembered that there are a number of so-called functional diseases which kill but leave no discoverable morbid traces behind. Our means of investigation are not yet accurate and fine enough to detect all the evidence which is wrought by affections of this sort. This statement pertains especially to diseases of the nervous system,—a class to which angina pectoris undoubtedly belongs.

Angina pectoris, then, being a recognized nervous disease, what are its essential features, and what portion of the nervous system does it invade? In answering this, I think the facts warrant me in saying that it is a disorder of the nerve-influence which dominates and regulates the tension of the arterial circulation; that this influence, as in epilepsy and in asthma, is thrown out of equilibrium and deprived of

its inhibitory power, and, in consequence, a spasmodic contraction of the heart and blood-vessels follows; and that the degeneration in the heart and blood-vessels is secondary to the perverted innervation.

What, now, are the facts which go to support this opinion of its nature? In the first place, Dr. Brunton has unmistakably shown, from a series of sphygmographic tracings, that the blood-pressure rises greatly during an attack of angina pectoris, or, in other words, that the arterial tension is largely increased during a paroxysm, and, in the second place, he showed that amyl nitrite relieves the disease by diminishing the blood-tension. Now, diminution of blood-pressure may be brought about in one of two ways: first, by lessening the power of the heart; and, second, by dilating the blood-vessels; and by a series of ingenious experiments Dr. Brunton demonstrated that amyl nitrite lowers the blood-pressure in animals, not so much by weakening the action of the heart as by dilating the large and small blood-vessels, and that in all probability this dilatation is produced through the influence of the vaso-motor nerves. The sphygmographic tracings and the physiological action of amyl nitrite concur in showing that the essential nature of angina pectoris consists in a spasm of the circulatory apparatus.

AORTIC REGURGITATION AS A CAUSE OF ANGINA PECTORIS.

Angina pectoris, then, being essentially a convulsion of the heart and large blood-vessels, it is quite clear, from our general knowledge of the etiology of disease, that any cause which lowers the nutritional tone of the nerves which supply the circulation will also become an exciting cause of angina pectoris. One such cause is aortic regurgitation, which, as we shall see, is capable of producing a failure of nutrition in the cardiac muscle and in the wall of the arteries, for the disease in question is infinitely more liable to occur in disease of the aortic valves than in disease of any other valves of the heart. Indeed, it is a question whether it is ever associated with affections of the mitral, tricuspid, or pulmonary valves alone. I collected from the literature of heart-diseases fifty-seven cases in which angina pectoris accompanied valvular lesions, and found the valves affected in manner and frequency as follows: aortic regurgitation, forty-four; aortic stenosis, two; aortic regurgitation and stenosis, four; aortic and mitral regurgitation, five; mitral disease alone, none. The significance of these proportions becomes still more manifest when we reflect that mitral disease is perhaps the most common of all the valvular lesions. Thus, of one hundred and ninety-two cases of valvular disease, casually collected, the mitral

was affected ninety-four times, the aortic forty-seven times, the aortic and mitral thirty-four times, the aortic and tricuspid once, and the mitral and tricuspid sixteen times, showing that mitral disease is nearly twice as frequent as disease of the aortic valve. This indicates very strongly, then, that exclusive disorder of the mitral, tricuspid, and pulmonic orifices and angina pectoris are, at least, not concomitant conditions, and that aortic disease is favorable to the generation of the latter disorder. Is this peculiar relationship only incidental, or is it a signboard which teaches us an important lesson concerning the pathology of the disease under consideration? I am not one who subscribes to the doctrine of chance, but believe that all things are governed by immutable law, and I am convinced that here is an instance of such government, if rightly interpreted.

Now, what relationship does aortic disease bear to angina pectoris? Does it stand as one of its causes, or as one of its effects? When we carefully consider the baneful influence, both direct and indirect, of aortic regurgitation on the heart and large blood-vessels, I do not see how it can require any stretching of the imagination to conceive a causal relation of this affection to angina pectoris; for during each contraction of the hypertrophied ventricle the blood is forcibly thrown into the large arterial trunks, distending these greatly and momentarily raising the blood-pressure to a maximum degree, while during diastole the blood rushes back through the leaking valve, empties the vessels almost completely, and suddenly drops the blood-pressure to a minimum point. This shuttlecock motion of the body of the blood not only exposes the blood-vessels to extreme oscillations of positive and negative pressure, but also weakens their resisting power and impairs their elasticity, and, in consequence, arteritis and other degenerative changes are set up in their walls. Much more than this, in health, when the valves are intact, the blood moves in a constant forward direction, and the heart and arteries are nourished by a blood-current the tension of which is comparatively constant. In aortic regurgitation, however, the blood is in a persistent to-and-fro movement, and the heart and blood-vessels are deprived of their requisite nutritive supply in consequence. In other words, these structures are bathed with blood, but suffer from poverty of the same, because it is too restless to be appropriated. It is obvious, therefore, why and how aortic regurgitation leads to greater degeneration in the heart and blood-vessels than can possibly occur in lesions of any other cardiac valve, and also why it may be regarded as one of the causes of angina pectoris.

URIC ACID AS A CAUSE OF ANGINA PECTORIS.

Angina pectoris is also a common affection among gouty and rheumatic people. This does not happen so much because gout and rheumatism, by producing endocarditis, have the special power to cause disorder of the aortic valves, and in this way, as we have seen, engender the disease under consideration, but because of the increased formation of uric acid, which is incidental to the gouty and rheumatic diathesis.

In *Brain* (Part I., 1891, p. 63) appears an article by Dr. Alexander Haig on "Uric Acid in Diseases of the Nervous System," which has a strong bearing on the subject under discussion, and from which, on account of its exceedingly great interest, I shall take the liberty of making liberal quotations. Being himself a sufferer from migraine, and knowing the close affinity between gout and this disease, Dr. Haig states that he was led to determine the amount of uric acid in the urine before, during, and after a headache, and always found that it preponderated largely during the attack. He also found that mineral acids diminished and alkalies increased the amount of uric acid excretion, and that acids relieved his headache and alkalies aggravated it. He furthermore lays down the proposition that, if other things are the same, "arterial tension varies with the amount of uric acid that is circulating in the blood." He says (p. 69), "I have accumulated many hundreds of pulse-traces which I think prove absolutely that the knowledge thus obtained gives me practically complete power over the rate and tension of the pulse, which in the great majority of cases is under the influence of uric acid in one way or the other. . . . On the one hand [p. 80], by diminishing the alkalinity of the blood, I can free it from uric acid, relax the arterioles, cause pricking and shooting pains in the joints, or greatly increase any previous gouty or rheumatic pains, quicken the pulse, and produce mental happiness and well-being, relieving headache or mental depression, if previously present. On the other hand, by increasing the alkalinity of the blood I can cause it to be more or less flooded with uric acid, can slow the pulse, contract the arterioles and raise the arterial tension, greatly diminish the excretion of urine and relieve the joint-pains, producing in their place, however, a sluggish circulation in the brain, with general languor, depression, and disinclination for exertion, and, if there is much uric acid, perhaps even headache or a fit." In other words, he holds that the presence of uric acid in the blood accounts not only for the sense of well-being and exhilaration before an attack of migraine or of epilepsy, but also

for the slowness of the pulse and the hardness and fulness of the blood-vessels during an attack of these diseases. That which is true of migraine and epilepsy in this respect is also true of angina pectoris, for it is an equally well recognized clinical fact that this disease is at least sometimes preceded by a similar feeling of exaltation, and is always, as we have seen, accompanied by a retarded pulse.

ONE REASON WHY ANGINA PECTORIS APPEARS IN THE EARLY MORNING HOURS.

Such, then, being some of the morbid effects of an excessive quantity of uric acid in the blood, the interesting question arises as to when the blood is most alkaline, and when, in consequence, the uric acid is most liable to preponderate in this fluid. We know that the blood is less alkaline when the body is doing active work than when it is resting, hence we have every reason to agree with Dr. Haig when he says that the acidity reaches its highest point about midnight, or in the small hours, and then gradually decreases until nine in the morning, when it is less than at any other time. From many pulse-tracings in his possession he is also able to show that the pulse-tension is highest in the early hours of the morning. "Such fluctuations in the excretion of uric acid and its amount in the blood occur every day, and it is only when they are exaggerated by errors in diet, by exposure to weather, or by the action of disease that their effects may be serious, and pass over into the region of pathology" (p. 79).

This, then, gives us a key to the problem why epilepsy, asthma, migraine, gout, and other nervous disorders are most apt to come on during the early hours of the morning. A periodicity at about this time seems to be characteristic of all functional nervous diseases, and this is precisely the time when angina pectoris is most prone to make its appearance, and, it being a spasmodic disease of the vaso-motor nerves, there is every reason to believe that uric acid is one of its exciting causes. Dr. Haig's paper is one of the most important contributions to scientific medicine which have been made for a long time, and it demonstrates, as its sequel proves, that a search for the causes of disease inside of the body is of infinitely greater value to the progress of our art than the researches which have been made in the interest of an objective pathology.

WHEN DO AORTIC LESIONS GENERATE ANGINA PECTORIS?

The danger of the supervention of angina pectoris in a given case of aortic insufficiency depends very much on the seriousness of the

aortic lesion, and whether it owes its origin to a rheumatic, a gouty, or a nervous diathesis, as is shown by the histories of the two following cases, which are introduced here for the sake of illustration:

F., male, aged twenty-eight, had two attacks of rheumatism, the first one five years and the last one a year ago. Ever since the last attack he has been suffering from dyspnœa, especially on exertion, and also from a paroxysmal pain over his heart and chest, which comes on usually after midnight. The pain, which is also accompanied by a sense of thoracic constriction, is very severe, and radiates down both arms to the very ends of his fingers. During and for some time after the paroxysm his arms and hands feel numb and are partly paralyzed. Physical examination showed the existence of a double aortic lesion. His pulse beats eighty-four times in a minute. Under the use of rather large doses of strychnine, and of medium doses of phenacetin and guaiac, the attacks were checked entirely for two months, but recently have shown a tendency to recur, although in a more moderate degree than before he came under my care. The additional application of galvanism to the neck—the negative pole over the upper part of the sternum and the positive pole over the cervical portion of the spine—keeps the attack in abeyance. In this case there is obviously a rheumatic condition at the bottom of the trouble, which is as important from a therapeutic as it is from an etiological stand-point. The diathesis, which may be rheumatic, gouty, syphilitic, or nervous, often needs more consideration than the active disease itself. Remove this, and you frequently relieve the disease. This has been the principal aim in this case, and it has at least temporarily succeeded.

The influence of the absence of diathesis is well illustrated in the history of the case which is presented below, and which is almost a perfect fac-simile of the first case so far as the physical signs are concerned, yet there is no angina pectoris present. This is M., male, aged forty, who has been troubled with dyspnœa and an occasional fulness of the chest on exertion during the last six years. He is of powerful build, and has been accustomed to heavy work. He never had rheumatism, and follows the vocation of a market-dealer. Physical examination shows aortic regurgitation and stenosis. His pulse is seventy-two beats a minute. Now, both of these patients have double aortic lesions, —murmurs of about equal intensity in both, so far as the ear can tell, —yet their needs and general conditions are entirely dissimilar. The lesion in the former case originated through an attack of rheumatism, while in the latter it was brought about by overwork and lifting heavy weights.

The accompanying heart-tracings, which have been taken from these cases, will in a measure assist in explaining the difference in the condition of the two patients. The first cardiogram belongs to F. and the second to M., both of them taken directly over the apex impulse of the heart.

FIG. 1.

Cardiogram belonging to F., to be read from left to right.

FIG. 2.

Cardiogram belonging to M., to be read from left to right.

In explanation, I would state that each pulse-tracing presents four points for consideration: 1st, the up-stroke, which represents the systole of the heart; 2d, the form of the apex; 3d, the down-stroke, which represents the diastole; and, 4th, the connecting curve between two succeeding impulses, which represents the auricular systole. (This is wanting in some beat-traces.) If, as in the tracing of F., there is an up-stroke which is almost perpendicular, it is an indication that the blood meets with very little resistance in being expelled from the left ventricle into the aorta, and that, in spite of the murmur which is heard during systole, there is very little actual stenosis present. The apex being sharp denotes that the heart empties itself fully into the aorta during each contraction. The down-stroke marks a very slight incline forward during its descent, showing that the aortic valves offer very little resistance to the back-flow of blood from the aorta into the ventricle,—the regurgitation being almost complete. The undulation in the line between the end of the lowest fall of the lever and the rise of the next indicates the auricular contraction. In the cardiogram taken from M., it is seen that there is a straight ascending line

and a sharp apex, indicating, as in the first case, no resistance to the outflow of the blood from the ventricle, although the murmur of stenosis exists. The line of descent is not perpendicular here, but is marked with jagged undulations, illustrating that the blood in regurgitating meets with greater obstacles than in the case of F.; or, in other words, the aortic valves are more patent in the case of M. than in that of F., and the regurgitation is not so large.

We cannot assume, however, that, because the patient M. has been free from angina pectoris up to the present time, he will always enjoy immunity from it. From what has already been said, it is very clear that aortic regurgitation has an innate tendency to bring on angina pectoris, no matter whether the aortic lesion arises traumatically, as from sudden strain (see page 31), or is of rheumatic or gouty origin: yet so far my experience teaches me that not all sufferers from aortic lesions die finally from this disease. Very frequently the mitral valves weaken and become leaky, and then such persons are cut off by intercurrent pulmonary disease before sufficient time has elapsed to produce disintegration in the way above indicated.

VAGUS DISEASE AS A CAUSE OF ANGINA PECTORIS.

Another cause of angina pectoris is found in disintegration of the pneumogastric nerves. From our knowledge of their distribution and physiology it is quite evident that disease of these nerves must necessarily manifest itself in disorder of the structure and function of the heart. Forty-five years ago it was shown by the Weber brothers that stimulation of the pneumogastric nerves with galvanism had the effect of slowing the action of the heart, and if the current was made sufficiently powerful the heart was arrested in diastole for a short time, after which it began to beat although the galvanization was continued. The influence of these nerves on the heart is also illustrated in the case of Czermak, who by pressing his vagus against a bony tumor in his neck could stop the beating of his own heart at will. Concato (*Virchow u. Hirsch's Jahresbericht*, 1870, Bd. i. S. 144) describes two cases similar to that of Czermak, in whom slowing of the heart could be produced by compressing the vagus on the right side of the neck. In both cases the position of the vagus and of the carotid was abnormal. The sense of thoracic constriction, with an urgency to take deep inspirations, etc., which Czermak observed in his own experience, was not present in these cases.

Furthermore, post-mortem investigation shows that when these

nerves are diseased they play an important *rôle* in the production of angina pectoris, as is attested by the following histories:

Case I. (Leroux, *Le Progrès Médical*, 1878, No. 27.)—Male, fifty-three years old, saddler, suffered from angina pectoris for seven years; the attacks came on from six to eight times a day, and the pain radiated into the left shoulder, arm, and lower jaw. After death, which came on suddenly, it was found that the left heart was hypertrophied, the coronary arteries open and normal, and both phrenic nerves and left vagus were healthy throughout. The right vagus was patent until it reached the right bronchus, to which it adhered, and was embedded in a peritracheal gland at the same spot. This gland was surrounded by hardened connective tissue, and was discolored on the surface and calcified in the interior. The vagus was involved in it for two centimetres, and at the place of compression there existed a thickening of its sheath.

Case II. (Romberg, "Diseases of the Nervous System," cited from Fothergill, "The Heart and its Diseases," p. 271.)—A man, thirty-six years old, complained of nervous symptoms, and of his heart standing still. It intermitted for five or six beats. The aspect of the patient showed that something terrible was going on within him; he sat as if thunderstruck, speechless, motionless, his eyes wide open, and his consciousness unimpaired. When asked about his sensations, he stated positively that for a second or often longer he had a presentiment of coming arrest in the shape of internal restlessness and oppression; that when the stoppage took place a violent pain seized both sides of the thorax, extending to the neck and then passing up to the nape and the head; that the pain remained fixed in the latter for some time after the attack; and that when the attacks were frequent he could scarcely get rid of the sense of weight at the cervix. The attacks were brought on by emotion. During the intervals the heart presented no abnormal action and was quite healthy. The attacks increased in severity, and he died in a state of torpor. After death it was found that the large cardiac nerve was woven into a black knot the size of a hazel-nut; the left vagus was involved in an underlying, nodulated, dark-blue lymphatic gland. The phrenic nerve was also embraced in the diseased gland.

Then, again, experimentation on animals makes it extremely probable that the pneumogastric nerves have a strong trophic influence on the heart-muscle, for Wasslief (*Virchow u. Hirsch's Jahresbericht*, 1881, Bd. ii. S. 139) concludes, from his experiments on rabbits, that the fatty degeneration of the heart-muscle is due to the elimination of the

vagus influence through vagotomy. He also shows that in starving pigeons fatty degeneration of the heart-muscle occurred much earlier in those in which the vagi were cut than in those in which they were not cut. In a research on the trophic relations of the vagi to the heart-muscle, Eichhorst (*Centralblatt f. d. Nervenheilkunde*, 1879, S. 111) used birds, dogs, and rabbits in his experiments, and concludes that a constant fatty degeneration of the muscle of the heart followed division of the vagi, provided the animals did not die too early of pneumonia.

There is reason for believing that the sympathetic nerves which largely form the cardiac plexus are likewise concerned in the causation of angina pectoris, for Lancereaux (*Gaz. Méd.*, 1864, p. 432) reports the case of a male, forty-five years old, after whose death from this disease it was found that a tumor had compressed several branches of the cardiac plexus of nerves.

EMOTIONS AND SUDDEN MENTAL SHOCKS AS EXCITING CAUSES OF ANGINA PECTORIS.

Taking into consideration the close anatomical and physiological relation between the brain and the heart, through the vagi on the one hand and the sympathetic on the other, we are in a position to comprehend how emotional influences and mental shock of any kind are capable of inciting a paroxysm of angina pectoris. In health, even, the heart is frequently disturbed by thinking of it, as is attested by the medical examination of applicants for life insurance. The fear that some possible lesion may be discovered by the examiner frequently accelerates the heart's action far beyond its accustomed limit and capacity. Romberg ("Nervous Diseases," p. 6) relates the case of Peter Frank, who, while concentrating his attention on the subject of heart-diseases, during the preparation of his lectures, was attacked with such severe palpitations and an intermittent pulse that he fully believed himself to be suffering from aneurism. All these symptoms disappeared, however, after his work was completed and after he had taken a long rest and relaxation. Tuke says ("Influence of Mind on Body," p. 87), "Some years ago a medical student in Paris, on being initiated into the mysterious rites of a Masonic society, was subjected to the sham operation of venesection. His eyes were bandaged, a ligature bound round his arm, and the usual preparations made to bleed him. When a pretence of opening the vein was made, a stream of water was spurted into a bowl, the sound of which resembled that of the flow of blood, which the student was anticipating. The consequence was that in a few moments he became pale, and before long fainted away. There

is a case on record of a man who was sentenced to be bled to death. He was blindfolded, the sham operation was performed, and water allowed to run down his arm in order to convey the impression of blood. Thinking he was about to die, he did actually die. Imagination had the same effect as the reality. But it is impossible to say how much fear had to do with it; probably a good deal, as in the instance of the man reprieved after his head had been laid on the block and the fatal axe was about to fall. The reprieve came too late. The anticipation of death had arrested the action of the heart."

John Hunter had an attack of gout when he was forty-one years old, and during the last fifteen years of his life he suffered and finally died from angina pectoris. He tells us that in his later life he became subject to violent disturbances of his heart when he was annoyed or anxious about any event. He says ("Works of John Hunter," edited by Mr. Palmer, 1838, vol. ii. p. 336), "At my country-box I have bees, which I am very fond of, and I was once anxious about their swarming, lest it should happen before I set off for town; this brought it on [an attack]. The cats tease me very much by destroying my tame pheasants, partridges, etc., and rooting up my plants. I saw a large cat sitting at the root of a tree, and was going into the house for a gun, when I became anxious lest she should get away before my return; this likewise brought on a spasm; other states, when my mind is much more affected, will not bring it on." Anger and anxiety had a very pernicious influence on his heart, and, knowing this, he said, "My life is at the mercy of any scoundrel who chooses to put me into a passion." And, indeed, his apprehensions were realized, for in a dispute concerning the admission of certain students to St. George's Hospital he was flatly contradicted by one of the governors. This so enraged him that he "immediately ceased speaking, retired from the table, and, struggling to suppress the tumult of his passion, hurried into the adjoining room, which he had scarcely reached when, with a deep groan, he fell lifeless into the arms of Dr. Robertson." An examination after death showed that his heart was small and strongly contracted. The coronary arteries had been changed into bony tubes, the aorta was dilated, and both the aortic and mitral valves were diseased.

SYPHILIS AS A CAUSE OF ANGINA PECTORIS.

The possibility that syphilis may produce angina pectoris is a subject of grave importance, and one to which sufficient attention has not as yet been directed. From our knowledge of the hydra-headed manifestations of this disease it would seem strange, however, if the heart escaped its contaminating influence. I have seen a number of cases of organic heart-disease and heart-pain the origin of which I attributed to a syphilitic source, but a true syphilitic angina pectoris I do not remember as having occurred in my experience. Other observers have been more fortunate in this respect, and during the last few years a number of such cases have been reported, and on account of their intrinsic value I shall abstract the histories of a few from an article by Professor G. A. Sacharjin, of Moscow, on "Die Lues des Herzens von der klinische Seite betrachtet" (*Deutsches Archiv für klinische Medicin*, 1890, Bd. xlvi. S. 388).

Male, aged forty, syphilitic, with frequent attacks of angina pectoris, which were followed by pulmonary œdema, and for which digitalis and other agents were employed to no purpose. Under an energetic course of sodium iodide treatment the attacks disappeared entirely, but returned later, although in so light a form that they were readily set aside by digitalis.

Male, aged fifty-five, infected with syphilis, although at an earlier period he passed through an attack of acute articular rheumatism, and was also addicted to the excessive use of alcohol. For more than a year he had suffered from severe attacks of angina pectoris, which were but indifferently relieved with nitro-glycerin. There was evidence of arterio sclerosis and aortic insufficiency, but aside from the stenocardiac paroxysms the patient was apparently well. Sodium iodide internally and inunctions of gray mercury ointment cured the attacks of angina pectoris, but had no effect on the aortic regurgitation or the chronic arteritis.

Male, aged —, is analogous to the last case. The attacks of angina pectoris were likewise entirely relieved by the same treatment.

Male, aged thirty-seven, addicted to alcoholic abuse for a number of years, and two years before he came under observation he acquired syphilis, which was followed by the customary secondary symptoms. Eight months previously he was suddenly attacked one night with angina pectoris. Two months ago he had his second paroxysm. Under the influence of sodium iodide and inunctions of mercury he was fully restored.

The author ascribes these attacks to the noxious action of syphilis on the heart and nervous system. What special portion of the nervous system he believes to have been affected is not intimated. May the vagi have been involved? That syphilis affects these nerves is attested by Vierordt, who relates a case (*Archiv f. Psychiatrie*, etc., Bd.

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xiv. S. 678) in which syphilitic infection produced multiple neuritis, great acceleration of the heart's action, exhaustion, and death from phthisis, after which it was found that the pneumogastric and sciatic nerves were disintegrated. If, as we have reason for believing, the vagi are affected in such cases, then the manner in which syphilis brings about angina pectoris becomes perfectly obvious, and also indicates a possible similar effect in the case of alcohol and other poisons, which have a like deteriorating influence on the nervous system. Indeed, Dr. Bean (*Gazette des Hôpitaux*, 1862, xxxv. 326, 329) relates eight cases of angina pectoris which were induced by tobacco-smoke, and which disappeared when tobacco was avoided.

CAUSE OF THE PAIN IN ANGINA PECTORIS.

Pain in the region of the heart is one of the constant symptoms of angina pectoris, and it is therefore of interest to inquire into the origin of this phenomenon. In considering this part of the subject we must bear in mind that the heart and large blood-vessels possess both sensory and motor nerves, which are supplied by the vagi and sympathetic. The sensory impulses conducted by the former reach the brain through the medulia oblongata, while those which are conducted by the latter pass through the spinal cord.

What, then, is the cause of this pain? In an exceedingly interesting paper "On Cardiac Pain in Angina Pectoris," in the *Practitioner* for October, 1891, Dr. Brunton expresses the belief that the cardiac pain in this disease "is generally due to weakness of the heart in proportion to the resistance which it has to overcome." In common with Dr. Grainger Stewart, he compares the heart in this disease to the bladder when over-distended with urine. The latter contracts against the increased resistance due to over-distention, and produces pain of the most excruciating character; while in the case of the heart it is not always a condition of over-distention, but one which practically leads to the same results. Its muscular fibres are thrown into a state of spasm, are made to contract in their weakness against a resistance which is difficult to overpower, and the pressure which ensues gives rise to the excessive pain.

Such a view of the production of pain in this disease is certainly very reasonable, and seems to be in perfect accord with the practical experience which we have on this subject. It is a well-known fact that persons who are affected with heart-disease—especially with ventricular dilatation—are very susceptible to attacks of pain in the region of the heart when they overtax this organ by exercise, or by

ascending mountain elevations. In other words, the resistance which the heart is to overcome by pumping a larger amount of blood into the blood-vessels, in these instances, is greater than its weakened capacity permits, and hence there is still greater distention, or at least a tendency thereto, and pain in consequence.

As instances of the injurious effects of excessive exercise on the heart, and in producing angina pectoris, I take the liberty of quoting the following histories, which are reported by Dr. V. D. Harris in *St. Bartholomew's Hospital Reports*, vol. xv., 1879, p. 86:

"Male, aged thirty, came under my care at Victoria Park Hospital at the beginning of March, 1879. He was a fair-nourished although anæmic man. He came complaining of attacks of dyspnœa and pain in the chest, indicating angina pectoris. He had been perfectly well, according to his account, up to the preceding boxing-day, when suddenly, whilst singing a solo in some musical entertainment at a large East-End theatre, he felt something give way in his chest, and had a spasm of dyspnœa, from which he occasionally suffered since. He did not know he had heart-disease. There was no history of gout, rheumatism, or syphilis in the patient or in his family. Examination of the heart showed a double murmur, probably in the aortic valves. The history and the physical signs appeared to indicate rupture of the aortic valves.

"Male, aged thirty-one, came to Victoria Park Hospital in June, 1879. Was quite well five years before; a great athlete, and was in the habit of lifting very heavy weights. One day he was lifting two fifty-six-pound weights, and felt something give way in his left side. Three weeks after he had the first attack of angina, after which time they always came on after exertion of some kind. He said the fit comes on suddenly, with intense pain in the chest, running down the inside of the left arm, and in the latter place it was as though the flesh were being torn off the bone with pincers. When the attack comes on he feels as though he could not breathe any more, but has to pull at the clothes about his neck. Sometimes the attacks are simply dyspnœa, but in these there is not so much pain, neither do they appear to him to be serious; the great seizures, occurring more rarely, have come on even from the exertion of getting up in the morning, and they appear to him to endanger life. Examination shows a lesion in the aortic valves; and from the history of the sudden onset of the heart-disease after tremendous exertion, the youth of the patient, and the absence of gout, rheumatism, syphilis, and atheroma, I am induced to believe it to be a case of ruptured aortic valve."

One of the ablest clinical articles on heart-disease in relation to pain, which has come under my notice recently, is that by Dr. James J. Levick, on "Heart-Strain and Weak Hearts" (*Transactions of the American Climatological Association*, 1888). After discussing the various phases of "heartache," he classifies the immediate exciting causes of sudden death in persons who suffered from heart-weakness, so far as his experience goes, as follows: "First, walking on slippery, icy pavements on a cold day. (The patient had walked three or four street blocks to church, and died soon after taking his seat there.) Second, hurrying to railway station immediately after eating a hearty meal. Third, driving for some miles a hard-mouthed horse. Fourth, riding a hard-mouthed horse. (The patient had been helped by gentle horseback riding.) Fifth, sawing off the limb of a tree in his own park. The limb required some effort to reach; the position was a constrained one. This gentleman had had frequent attacks of this disease (heart-pain), with a feeble heart. A violent paroxysm followed this exertion, and he died before medical aid could be obtained. Sixth, hurrying from one steamboat to another, carrying at the same time a heavy bag. Seventh, assisting to carry a trunk from the railway van to the station. Eighth, shovelling coal into the furnace in the cellar. Ninth, the act of sexual intercourse. Three cases of this kind have recently come under my notice. In the first, a married man, aged sixty-five, had a violent paroxysm of cardiac pain immediately following this act. The patient lived for more than six months, was liable to severe paroxysms of dyspnœa,—which he never had before,—and died suddenly as he arose from his tea-table. A post-mortem examination showed the absence of valvular disease, but the existence of a firm clot in the ventricle, which was evidently ante-mortem, and which doubtless was formed coincidently with the first severe paroxysm six months before. The second case was that of a gentleman, aged seventy-two, single, and remarkably hale and vigorous for his years, but who had at long intervals attacks of heart-pain. After a morning drive, his coachman driving, he visited (I use his own words) a lady and committed venery. He was almost immediately seized with an intense pain near the heart, but managed to walk home, a short distance, and I found him there with a cold skin, very feeble pulse, although he walked forward to receive me. He was immediately put under treatment, but death supervened rapidly. A somewhat similar case is reported of a judge of the Nottingham assizes, who was induced to go home with a young woman of the town, who testified before the coroner that immediately after having had intercourse with her he turned

on his side, gave a groan, and died. I have recently seen in consultation a fatal case of heart-failure in an elderly man, where the history pointed to this as the cause of death."

Dr. Alfred L. Loomis contributes a very interesting paper on "The Effects of High Altitude on Cardiac Diseases" to the *Transactions of the American Climatological Association* (1888), in which he details the histories of six cases of heart-disease which were affected very deleteriously by ascending mountain elevations of from one to four thousand feet. The heart-symptoms became aggravated in each case by the ascent, although in none were there any characteristics of angina pectoris developed. Five died, and one recovered after being brought nearer the sea-level.

In my own experience I met with at least one person with heart-disease in whom a severe attack of angina pectoris was provoked, and whose death was finally brought about, by ascending a mountain elevation about twelve hundred feet higher than his own home. It was that of a young man, aged twenty-two, who, when he was fourteen years old, suffered from chorea for nine months, directly after which he had an attack of articular rheumatism, which gave rise to some heart-trouble. At the age of twenty he had some difficulty with his heart, which was followed by profuse œdema of the lower extremities. This yielded to the judicious treatment of his physician, and from this time up to the time when he visited his aunt on the mountain elevation, and where he came under my care, he had been comparatively free from disease, and was able to do some work. Immediately after arriving at his aunt's house he was taken with a most excruciating pain in the cardiac region, and his sufferings here and along the course of the large arteries of the neck and arms were intense. Physical examination showed lesions of both the aortic and mitral valves. Œdema of the lower extremities supervened, but under the use of heart-tonics he rallied sufficiently to be able to return to his own home in the course of two weeks, where he died from copious hæmoptysis, probably due to rupture of a pulmonary blood-vessel, the day after he reached it. The autopsy showed an enormously hypertrophied and dilated heart, weighing a fraction over three pounds, with the aortic and mitral valves almost entirely obliterated. I think there is reason to believe that the attack was precipitated in this case by the relatively greater rarefaction of the mountain atmosphere, causing a disturbance in the already supersensitive equilibrium between the inside pressure of the blood and that on the outside of the body, and the resultant strain was too great for the previously weakened heart and arteries to endure.

All the evidence which has been adduced thus far shows that anything which disturbs the cardiac equilibrium, be this a rupture of the aortic valves, a sudden weakness induced by mental excitement, a simultaneous spasmodic contraction of its wall and of the arterial coats, or abrupt barometric transitions, will produce the same final results, —viz., an increased resistance within, and an inadequate power without, thus confirming Dr. Brunton's view of the generation of pain in this disease.

THE SIGNIFICANCE OF ATHEROMA AND OSSIFICATION OF THE CORONARY ARTERIES AND FATTY DEGENERATION OF THE HEART-MUSCLE.

There is a disposition on the part of many who have discussed this subject to attribute angina pectoris to atheroma and ossification of the coronary arteries; indeed, some have gone so far as to suggest that only those cases in which there is a morbid change of this kind, especially if this is associated with fatty degeneration of the heart, should be designated as genuine angina pectoris, and that this ear-mark should form the dividing-line between the true and the false forms of this disease. It is hard to account for the basis on which this argument rests, for atheroma and ossification, not only of the coronary arteries, but of the aorta, are quite common occurrences,—in fact, they may be regarded as different steps in the process of chronic inflammation of the intima, —yet angina pectoris is a comparatively rare disease, and is found independent of such degeneration. In the case of Leroux, quoted on page 34, the coronary arteries were open and normal; there was no fatty degeneration of the heart-muscle, but there existed an atheromatous condition of the aorta. Rothe contributes the following case (*Virchow u. Hirsch's Jahresbericht*, 1885, Bd. ii. S. 78), which shows no morbid change anywhere in the central portion of the circulatory apparatus beyond a dilatation of the arch of the aorta, and gives us reason for believing that extreme dilatation of the aorta may act as an indirect cause of death in this disease.

Male, aged fifty-three, while in his last attack of angina pectoris had an accelerated but regular pulse, unimpeded respiration, and was fully conscious. Chloroform, morphine, and amyl nitrite failed to relieve him. Death took place suddenly. On section, the thoracic and abdominal organs were found healthy, with the exception of dilatation of the aortic arch, the walls of which were hard and had lost their elasticity. The coronary arteries and heart wall were unaltered.

The same may be said of fatty degeneration of the heart as a cause

of angina pectoris. If we take for granted that this disease is fundamentally of a nervous character, it is readily seen that fatty degeneration may be a natural sequence of nerve-disorder. The experiments of Eichhorst which have been quoted on page 35 demonstrate that fatty degeneration of the heart-muscle follows section of the vagi in birds, dogs, and rabbits. We must remember, however, that fatty degeneration may also occur in muscle on account of inactivity, or on account of inanition sequential to nerve-section, and therefore these experiments are not wholly conclusive. But Wasslief's experiments, noted on page 34, establish the fact that among starving pigeons fatty degeneration occurred much earlier in those in which the vagi were cut than in those in which the vagi remained intact.

This gives us still more reason for believing that degenerations of the coronary arteries and of the heart-muscle are merely sequences and not causes of angina pectoris. Indeed, all the evidence, so far as I have been able to bring it together, goes to show that angina is essentially a disease which belongs to the spasmodic type, and that we might as well assert that bronchitis, bronchiectasis, emphysema, hypertrophy of the right ventricle, etc., are causes, and not secondary changes, of asthma, as to say that atheroma of the coronary arteries, fatty degeneration of the heart-muscle, dilatation and ossification of the aorta, etc., hold a causative relation to this disease.

INFLUENCE OF HEREDITY.

That heredity is a factor in the production of angina pectoris there can be no doubt in the mind of any one who has given thoughtful attention to this subject. It arises frequently among the members of neurotic families, who are also predisposed to asthma, migraine, epilepsy, hysteria, and pulmonary consumption. Dr. Ross states ("The Diseases of the Nervous System," vol. i. p. 577) that "hereditary predisposition can be traced in many cases of angina, and it is frequently found in members of families who manifest a tendency to other neurotic diseases, such as hysteria, insanity, and epilepsy. Attacks of angina may form a symptom of hysteria, precede or alternate with an attack of epilepsy, or constitute an intercurrent symptom of chronic mental disease." Dr. Anstie ("Neuralgia, and the Diseases that resemble it," p. 146) gives a *résumé* of three cases of angina pectoris in the families of which there existed epilepsy, asthma, softening of the brain, and other neurotic diseases.

THERAPEUTICS OF ANGINA PECTORIS.

The therapeutics of this disease resolves itself into those measures which give instantaneous relief and those which prevent a recurrence; and only such means will be referred to here as have been found serviceable in my own experience. We have seen that the essential nature of angina pectoris is a spasmodic convulsion of the heart and blood-vessels, and in order to meet the first indication we are compelled to resort to the administration of agents which possess the power of relieving this spasm. To the scientific acumen of Dr. Brunton the profession is indebted for its possession in amyl nitrite of an agent which is capable of dilating the heart and blood-vessels, of lowering the blood-pressure, and of relieving angina pectoris. This discovery he made in 1866; amyl nitrite has been employed by inhalation in the great extremity of this disease ever since. It is certainly one of the most valuable additions that have been made to the therapeutics of internal diseases for a long time, yet in some cases it fails to act favorably, chiefly, I think, on account of its application to doubtful pathological conditions. Such is the impression which I have been able to gather from my somewhat limited experience with this drug, and I can offer no better guide to its proper administration than that of correctly diagnosing the disease. The rule which is generally laid down, that amyl nitrite is applicable in those cases only in which there is no flushing of the face, does not seem to apply universally; and when we take into consideration that a spasmodic condition may exist in one part of the circulation and dilatation in another part at the same time, it is obvious that the absence or the presence of flushing of the face may be a very spurious index as to the state of the circulation in the heart and large arteries, at least in some instances, although as a rule there is present paleness of the face in a paroxysm of angina pectoris.

This opinion also seems to be confirmed by the personal experience of Dr. Madden, of Torquay, as he relates it in *The Practitioner* (vol. ix. p. 331), and from which, on account of its intense practical interest and of its strong bearing on this subject, I take leave to make a copious quotation. After announcing the remarkable benefit which he obtained from the use of the amyl nitrite, he states that on one point he certainly increased his wisdom. From his reading he had formed the opinion that it was suitable only in those cases in which the face was pallid during the paroxysm, and since his was always flushed he had given up the idea of even trying it, "and paid the penalty of hasty conclusions in the shape of a large amount of acute suffering."

At the age of twenty-four he suffered from the effects of overwork, at which time there was also discovered some obscure disease of his heart and lungs. A short time before, his father died of angina pectoris,—the organic condition of the heart in his case being "atheromatous obstruction of the coronary arteries." Continuing to suffer from his heart- and lung-affection, he left Scotland the same year (1839) and went to Torquay, and in a few years he recovered sufficiently, resumed his practice in that place, and led an active life up to the time of writing. "On July 8, 1872, being fifty-seven years of age, feeling perfectly well, I was suddenly, and without the slightest warning, arrested by a severe attack of angina pectoris, the pain extending across the front of the chest, along the inside of the left arm, and across the chin." A medical friend was called in, and diagnosed a systolic mitral murmur, and counselled rest, which Dr. Madden did not take, but persevered in his ordinary work for ten days longer, when the severity and the frequency of the attacks compelled him to stop. "At first it seemed as if the quiet would prove curative, but in the afternoon of the fourth day, after taking to bed, I woke out of a doze with the severest and most prolonged spasm I had yet experienced. From this time the disease appeared to acquire increased violence. The attacks lasted, for the most part, for a quarter of an hour or twenty minutes, and recurred frequently at intervals of about three hours. Various remedies were tried, with little or no benefit. Morphia given hypodermically was the most useful, but it was impossible to employ it often enough without producing dangerous narcosis. At this time, when I was getting thoroughly worn out by the constantly-recurring pain, a friend, who happened to have in his possession a specimen of the amyl nitrite, suggested to one of my kind attendants the desirableness of giving it a trial, and furnished him with a small quantity. He consulted with his colleagues, and they unanimously advised me to make the experiment. I was willing enough to do so, and that night I was roused out of my first sleep by a sharp attack. I at once inhaled five drops, and the effect was truly wonderful. The spasm was, as it were, strangled at its birth. It certainly did not last two minutes, instead of the old weary twenty; and so it continued. The frequency of the paroxysms was not diminished for some time, but then they were mere bagatelles as compared with their predecessors, and consequently the drain upon the vital energies was greatly reduced. Under these improved circumstances, strength gradually returned; the attacks became less and less frequent, and finally ceased. At the time of writing these lines (October, 1872), I have not had an attack for five

weeks, and have resumed my ordinary duties, of course with care." In a private communication to Professor Gairdner ("Reynolds's System of Medicine," vol. iv. p. 590), in August, 1875, Dr. Madden states "that his confidence in the remedy continues unabated, but he has not required to use it for a considerable time."

After the introduction of amyl nitrite, professional attention was also directed to sodium nitrite and nitro-glycerin, which, on account of the resemblance of their action to that of amyl, were believed to be endowed with the same therapeutic properties, but this has not been borne out by practical experience. The hypodermic injection of morphine, from one-sixteenth to one-eighth of a grain, combined with one-twenty-fifth of a grain of strychnine, is useful in cutting short the attacks. So are also the inhalation of chloroform, and the application of hot poultices, mustard, and strong liniments over the anterior portion of the chest.

In the second place, what are we to do to prevent a recurrence of these attacks? First of all, our patient must avoid all exciting causes, such as fear, anger, or excitement of any kind, must do no heavy work, lift no heavy weights, and be guilty of nothing which will unduly raise the blood-pressure and thus throw an extra strain on the heart and large vessels. His food should be of easy digestion and at the same time highly nutritious, and for this reason he should live principally on milk, soups, meats of all kinds, and a fair proportion of vegetables, and drink plenty of water. In the next place, it is the physician's duty to endeavor to alleviate any organic disease of the heart that may exist, to neutralize any gouty or rheumatic diathesis that may be present, and to elevate the nutritional tone of the nervous system to the highest state of efficiency.

For the purpose of correcting any bad effects arising from organic deformity of the valves, it will be found that, as a rule, all our common cardiac stimulants—such as digitalis, caffeine, and strophanthus—are of very little value; in fact, sometimes they are positively detrimental. This may possibly be due to the fact that these agents influence the heart and the arteries too directly and too powerfully, and in this way create too great an immediate tension in the circulation; for strychnine, which I know from practical observation to be an indispensable drug in the treatment of this disease, also elevates the blood-pressure, but probably brings this about in a more gradual manner. In my experimental work I often noticed the pronounced difference with which digitalis and strychnine affected the frog's heart. Both enhanced its contractile power, but digitalis produced great irritability of the heart, and finally arrested it in systole, while strychnine did not increase its irritability, and instead of arresting the heart in systole it arrested it in diastole. This is a great difference between the action of the two agents which seemingly affect the same structure in a similar manner, and it may throw some light on the clinical experience which we have had with these agents.

Give strychnine, then, for this purpose, and in doses large enough to produce its effects. I rarely begin with less than one-thirty-second of a grain, and gradually increase to one-twentieth of a grain every four hours. The patient F., whose history is given on page 31, began with one-thirty-second of a grain of strychnine four times a day, but during the last two months it has been increased to one-twentieth of a grain four times, and he has been practically free from attacks since the increased dose has been taken. In a rebellious case I should recommend an additional daily dose of one-thirty-second of a grain of the same drug hypodermically. At the same time it may be given hypodermically once a day in one-thirty-second of a grain. Strychnine is one of our best permanent antispasmodic agents in the materia medica, not only in angina pectoris but in asthma, and it has also produced some very serviceable results in epilepsy. Phenacetin, antipyrin, antifebrin, quinine, arsenic, and atropine are also useful in elevating the nerve-tone of the arterial circulation. To counteract the rheumatic or gouty diathesis, guaiacum, salol, sodium salicylate, or colchicum must be given. The following combination is useful in many cases:

℞ Strychninæ sulph., gr. i;
Atropinæ sulph., gr. $\frac{1}{20}$;
Acid. arsenic., gr. $\frac{1}{2}$;
Quininæ sulph., gr. xxxii;
Phenacetini,
Guaiaci res., or salol, āā gr. lxiv.—M.
Ft. capsulæ no. xxxii.
Sig.—One capsule four times a day.

In addition to the above, the mineral acids must be given with each meal, and an occasional calomel purge must be employed.

The application of electricity is useful as a means of cure. Duchenne, Eulenberg, and Von Heubner speak very highly of it, and Bramwell, in his work on "Diseases of the Heart," says he has seen distinctly good results from the use of galvanism. He applies a current from about thirty elements, with the positive pole over the sternum and the negative pole over the lower cervical vertebræ. Personally, I have not had much experience with electricity here, but it is certainly as much indicated here as in any other neurotic disease.

FATAL CASES OF INTESTINAL OBSTRUCTION IN WHICH THERE WAS SIMPLE DILATATION AND HYPERTROPHY OF THE LARGE INTESTINE.

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Case I.—The patient, a young man, aged seventeen, was admitted to St. Thomas's Hospital on March 20, 1885. Inquiries, I believe, were made as to the habitual state of his bowels, but no history of constipation was obtained. He came ashore from a training-ship at Devonport ten days before admission, and since that date he had passed nothing by the bowel. After the constipation had lasted four days he began to have paroxysmal pains in the abdomen.

On admission he was a well-nourished, healthy-looking lad, complaining of pain in the abdomen and back, and of constipation. He sat up in bed leaning forward, in which position he felt easiest. The skin was cool, the temperature normal, the tongue clean. There was no sickness or hiccough. The abdomen was tympanitic and distended, especially above the umbilicus and on the left side, the parietes in the latter situation being rigid. The abdominal pain was paroxysmal, and pressure did not aggravate it. The chief seat of pain was the lumbar spine and the adjacent muscles, and there was tenderness in this position. The rectum was empty. The urine was 1030, loaded with urates, and contained no albumin; large quantities were passed without difficulty. The heart and lungs were healthy. The pulse was 92, full and regular.

A simple enema was administered, and after a few hours an enema of olive oil, but neither was retained and no fecal matter came away. On the evening of the same day a pill containing one grain of opium was ordered to be taken every six hours.

On March 21 (the day after admission) there was more fulness and distention of the abdomen, and it was tympanitic all over. He com-

plained of pain in the epigastrium, which seemed to be relieved by pressure. The pain in the back was still very severe. The patient continued to sit up in bed as the easiest position. The tongue was red and dry. There was some flatulence, but no hiccough and no vomiting. He had tenesmus several times, and passed a little blood. An enema composed of eight ounces of olive oil and two of castor oil was given, but not retained. Mustard, followed by liniments containing chloroform and belladonna, was applied to the abdomen and back. A grain of opium was given every four hours. His temperature, which had been normal or a little subnormal since admission, rose to 100° at 8 P.M., but soon dropped to the normal again.

On March 22 the pain was greater, although the patient had been under the influence of opium for thirty-six hours. The abdomen was still distended, but not more so than on the previous day. There was no vomiting nor hiccough, and there had been no action of the bowels.

At noon Mr. Croft performed abdominal section. The small intestine appeared healthy, but the large intestine was distended and deeply congested. About two feet of the large bowel was so affected, and it was thought that the sigmoid flexure was twisted on itself. The incision was extended upward for two inches, but it was found impossible to untwist the gut; it was punctured with a fine trocar and canula and some gas evacuated. The intestine was then brought to the edge of the wound and opened where the puncture had been made. Some dark-red fluid, consisting of mixed blood and fæces, escaped.

The patient bore the operation well, and for a few hours seemed much relieved. Four hours later the temperature was 97.4°, the pulse 120, full and incompressible. There had been no sickness nor hiccough. There had been a good deal of fecal discharge, and the iodoform dressings had been changed twice. Two or three hours later he became restless, and complained of pain about the wound. The temperature was 100.8°, and the pulse 148. At midnight (that is, about twelve hours after the operation) it was noted that he was suffering a good deal of pain, but that he had dozed occasionally. He had passed no urine since the operation. The temperature was 101.8°, and the pulse 142. He was taking a grain of opium every four hours.

All the next day the abdominal pain was very severe, at first being paroxysmal, but later on constant. The abdomen was much distended and very tender. He had no sickness nor hiccough. In the morning there was a good deal of fecal discharge from the wound, but none later in the day. The temperature ranged from 99° to 101°, and the pulse, which was feeble, from 125 to 148.

On March 24 (the day following) Mr. Croft performed right lumbar colotomy. Much flatus escaped when the ascending colon was opened, and subsequently a considerable quantity of fæces. For a few hours the patient was much easier. Later in the day the pain became very intense, in spite of repeated injections of morphine, and he died the same evening.

Post-Mortem Examination.—There was nearly general peritonitis, most intense about the abdominal incision. The artificial anus in the middle line of the abdomen was situated three and a half feet below the ileo-cæcal valve, and was formed by a portion of the sigmoid flexure. The gut for about an inch up was a little dilated, but below the artificial anus the whole sigmoid flexure was enormously dilated and hypertrophied, measuring in parts ten inches in circumference; it lay nearly in the median line of the abdomen, its mesentery being very broad and loose. No twist or abrupt turn in the intestine was found. There was a large amount of liquid and formed fæces in the affected intestine; the mucous membrane was much congested, and showed a few slate-colored adherent sloughs and numerous enlarged solitary glands. The rectum was moderately dilated, apparently not hypertrophied, and contained some semi-fluid green fæces; there was no stricture, ulcer, nor anything which might cause obstruction. Lastly, it may be mentioned that the cæcum was much distended with fæces, and that the artificial anus in the right loin was formed by a portion of ascending colon, six inches from the ileo-cæcal valve.

Case II.—The patient was a man, aged sixty-six, who was admitted into St. Thomas's Hospital, under my care, on August 5, 1892. His bowels had not been opened for five days. Three days before admission he began to have paroxysmal abdominal pain, and about the same time he had some vomiting, which recurred from time to time. I questioned him very carefully about the habitual state of his bowels, as I was inclined to consider the case as belonging to the class now under consideration. He declared that he had never suffered from habitual constipation, and when closely pressed only allowed that "he might miss a day."

The abdomen was generally distended, and every now and again there were acute attacks of pain referred to the hypogastric region. During the paroxysms it became harder and more prominent in this part and in the umbilical region. Nothing was felt per rectum. A simple enema was administered soon after admission, but it was returned almost at once and without result. Later in the day a warm olive-

oil enema was given by the long, soft tube, which passed quite easily nearly its whole length, but the oil was retained and there was no result. It was determined to try half an ounce of olive oil by the mouth every two hours; but after the second dose he began to vomit, so it was stopped. No flatus had been passed since admission.

The next day the pain became more severe and more frequent, so it was decided to perform abdominal section.

Sir William MacCormac made an incision three and a half inches long in the right iliac region, and the cæcum, being found distended, was sutured to the parietal peritoneum and the abdominal wall. The bowel was not opened, but later in the day it was punctured by a trocar and canula and much flatus evacuated, to the patient's great relief. He stood the operation well, the pulse remaining good. A hypodermic injection of morphine was given after the operation, and repeated later in the evening. He passed a good night; the pain was much less, and the distention of the abdomen considerably diminished. The tongue remained, as it was on admission, dry and furred; but he took nourishment well, the vomiting ceased, and the pulse was good.

On August 8 (the next day) the bowel was opened and a small quantity of fecal matter and some flatus escaped. During the subsequent two days the pain was much less, but there had been no evacuation by the rectum and very little by the artificial anus.

On August 11 the pain and vomiting recurred, so Mr. Ballance, in the absence of Sir William MacCormac, opened the abdomen in the middle line below the umbilicus, and found an enormously-distended portion of large intestine, which he punctured, allowing the escape of some fæces and flatus. The pulse had been getting weaker during the preceding two or three days, and it was occasionally irregular. The patient died the day after the second operation.

Post-Mortem Examination.—The artificial anus in the right iliac region had been made from the cæcum. In the middle line of the abdomen, extending from the umbilicus to the brim of the pelvis, there was an enormously distended and hypertrophied portion of large intestine, consisting of the lower part of the sigmoid flexure and the upper part of the rectum. The centre had been punctured and fæces could be forced through the opening. The rectum, as low down as the anus, was also much dilated, but not nearly so much hypertrophied as the bowel above. The descending colon was of normal size throughout. The first part of the sigmoid flexure, which was little, if at all, dilated, passed directly upward parallel to the course of the descending colon, and was concealed by the dilated portion of the sigmoid flexure. The

transition from the undilated to the dilated portion was rather abrupt, but there was no constriction, ulceration, or stricture. The mesentery of the sigmoid flexure and rectum was thickened, and unusually loose and broad. The length of the dilated and hypertrophied sigmoid flexure and rectum was twenty inches; the circumference of the former was ten inches, and of the latter seven to eight inches. The muscular bands of the affected portion of bowel were enormously hypertrophied. The gut contained much semi-solid fæces, but no hard lumps; the mucous membrane was not ulcerated. There was no ulcer nor stricture at the lower end of the rectum. The cæcum and the ascending colon were distended with fæces, but not hypertrophied. The small bowel was of normal size, and contained ordinary fecal matter in most parts; but there were some coils which were collapsed and nearly empty.

There was a good deal of bile-stained mucus in the large intestine and in the lower portion of the small intestine, but there was no change in the mucous membrane.

The stomach showed an excess of mucus and some recent hemorrhages.

The lungs were emphysematous, and there was hypostatic pneumonia along the posterior borders.

In both these cases there was no mechanical impediment below the dilated and hypertrophied large intestine, and the question therefore arises as to the causation of this condition. That the intestines may undergo enormous dilatation and hypertrophy from prolonged constipation is undoubted. An interesting communication by Dr. Bristowe,[1] to which I shall refer, deals fully with this point.

After narrating cases in which dilatation and hypertrophy of the large bowel dependent on constipation were suspected during life, Dr. Bristowe proceeds to give other instances in which the condition of affairs was found, after death, to be such as I have described. In these cases there was good reason to believe that the actual cause was prolonged constipation, and in all three there were great dilatation and hypertrophy of the large intestine, with extensive ulceration of the mucous membrane from fretting by the fecal lumps.

Reverting to my two cases, I may say that we were not able to satisfy ourselves that the patients were the subjects of habitual constipation. I am not sure that this was as strictly inquired into in the

[1] Clinical lecture on the "Consequences of Long-Continued Constipation," British Medical Journal, May 30, 1885.

first case as it might have been, but in the second case I carefully cross-questioned the man myself more than once, and I failed to obtain a history of the cause which I strongly suspected. The clinical history in both appeared to indicate that the condition was rather acute, but there can be no doubt that the dilated and hypertrophied bowel had existed for a time long antecedent to the onset of the urgent symptoms. I suspect (and Case II. supports the view) that the abrupt onset of symptoms was due to some misplacement or twist of the affected bowel. If constipation as a cause be excluded, I am inclined to the belief that the undue laxity of the mesocolon and mesorectum, such as was found post mortem, might account for the dilatation and hypertrophy. It was suggested to me by Mr. Bland Sutton that possibly a fissure of the anus might give rise to the condition of things which I have described. I must admit that this did not receive special inquiry, but I believe that its presence would probably have been detected had it existed.

Before speaking of the treatment of these cases it may be of interest if I give the notes of the case of an infant who died of obstruction which appeared to be due to prolonged and neglected constipation. I have never seen or read of another example in so young a subject.

Case III.—The patient was a male infant, aged eleven weeks, who came under my care on August 27, 1891. It was stated that the bowels had been natural until three weeks after birth, when he had pain, vomiting, constipation, and abdominal distention. These symptoms continued until admission. The child had been brought up on the breast alone.

On admission the abdomen was found greatly distended and tympanitic all over. The coils of intestine were visible through the abdominal wall, and peristaltic movements could be seen at times. A catheter was passed four inches up the rectum, and a little flatus and fecal matter escaped. The child remained much in the same condition, the bowels being unopened, and there being occasional vomiting, until September 18, when there was a free evacuation of fæces, and later in the day small quantities of fecal matter were passed after an enema of olive oil. The next day the bowels were again freely relieved, but the child, who had been getting more and more feeble since admission, died the same day.

During life we suspected the true condition of affairs, but the child's state contra-indicated operation even during the first few days after admission.

Post-Mortem Examination.—There was found to be great distention of the lower two feet and a half of the small intestine and of the large intestine as far as the junction of the descending colon and sigmoid flexure, but there was no stricture at this point. The sigmoid flexure and the rectum were empty. There were many ulcers laying bare the muscular coat of the large intestine and of the lower few inches of the small intestine. There were no tubercles.

In discussing the treatment of the first two cases one has to bear in mind the difficulty in diagnosing the condition during life. If there is good reason to suspect it, the chief means to be adopted are kneading or massage of the lower part of the abdomen, the exhibition of laxatives, such as large and repeated doses of olive oil or castor oil, and copious enemata administered by the long tube. I agree with Dr. Bristowe that the use of drastic purgatives should be avoided.

One must admit the probability that such measures as I suggest may be without avail, and that the urgency and obstinacy of the symptoms may call for abdominal exploration. Provided that the diagnosis be established (and this, of course, is the difficulty), the best means to adopt would be to expose the distended sigmoid flexure by an incision in the middle line below the umbilicus, to rectify any misplacement, and to endeavor to force through the anus part, at least, of the fecal contents, or, failing in this, to make an artificial anus.

GASTRIC ULCER.

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY CHARLES G. STOCKTON, M.D.,

Professor of the Principles and Practice of Medicine in the Medical Department of the University of Buffalo; Physician to the Buffalo General Hospital.

GENTLEMEN,—The patient is a physician, forty-four years old, and since the age of twenty-one he has practised his profession. His father died at seventy of what is believed to have been gastric ulcer of that form which has been described to you under the name of round or perforating ulcer or the ulcer of Cruveilhier. The patient's mother is living, at the age of seventy. He has one brother who suffers from indigestion, and four sisters, one of whom has some heart-difficulty and two have slight dyspepsia. When a student at this college, the patient suffered intensely from indigestion. He subsequently practised medicine, and, although he has been active and able to work, he has been rather delicate and has suffered with painful dyspepsia during all this time. About four years ago he had a very severe hæmatemesis, while suffering from pain and distress in his stomach, with vomiting. Subsequent to the hæmatemesis he vomited mucus and had great pain in the epigastrium and right shoulder. After this severe attack he became better than he had been for some years, but from time to time his trouble has recurred, with slight vomiting of blood and mucus and with pain, inability to eat, loss of sleep, and it became necessary for him to take anodynes. He came to me about three weeks ago suffering intensely from pain in the epigastrium and right shoulder; he was vomiting persistently, the vomited matter containing large amounts of mucus. Three or four months ago he had vomited a small amount of blood. He was digesting little, and was very much excited from his long-continued pain, and his nervous condition was not improved by the use of morphine, of which he was taking one-quarter grain hypodermically every six hours. Ice-cream he had found to be about the only nourishment that he could retain, and often his stomach would not tolerate even that.

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The diagnosis had been made of gastric ulcer and dilatation, the dilatation being supposed to depend upon contraction of the pylorus which often accompanies the cicatricial change of ulceration. On examination, there was a moderate succussion-sound to be heard, and by the usual methods of percussion and bimanual palpation I found that the lower border of the stomach came about to the umbilicus. Although this could not be considered to indicate an actual dilatation, it did represent a relative dilatation, since the doctor had not taken food in any quantity for a long time. The stomach, though relaxed, had sufficient power to expel the mucus which was in it, and which, to my mind, proved that there was present gastric catarrh, the result mainly of excessive vomiting.

Now, on making light percussion, with the patient on his back and without using the pleximeter finger, I am compelled to say that the stomach is dilated, since the lower border reaches at least one and a half inches below the umbilicus. Not only is the percussion-note clear to this point, but the succussion-sound also. At a point half-way between the ensiform cartilage and the umbilicus and two inches to the right of the median line there were formerly induration and great tenderness, but these signs have disappeared. The patient is taking more food now than before, and the signs of dilatation have probably developed as a result. For breakfast this morning he took beefsteak, farina pudding, bread and butter, and a glass of milk, and he has since had a second half-pint of milk. He suffers no pain nor tenderness, he is rapidly gaining flesh and color, and he is feeling well. He has taken no morphine since his entrance here, three weeks ago.

We must admit the diagnosis of gastric ulcer with dilatation. The patient was *in extremis* when he entered, suffering intense pain, almost starving,—for he had been obliged to nourish himself by the rectum when it was not possible for him to retain a little ice-cream. Let us see if we can account for his condition and for his improvement.

The round or perforating ulcer, which was beautifully described by Cruveilhier many years ago, has been accounted for by some investigators as a result of thrombosis or embolism in some small vessel near the pylorus, for the lesion usually is found near the pylorus, along the lesser curvature, and on the posterior wall of the stomach, though it may elect other points. To my thinking, this theory is not satisfactory. I believe the ulcer is to be explained on the basis of some neuropathy, like that of hæmatoma auris in the insane or that of symmetrical gangrene in young people,—Reynaud's disease. Again, if herpes selects certain points for its appearance on account of some

neuropathy, I think we may similarly explain gastric ulcer, which selects some point of the stomach where there is a local weakness, as due to a devitalization, so to speak, of the tissues, which are then attacked by the gastric juice and digested, leaving the characteristic ulcer with abrupt walls, and having, therefore, the peculiar punched-out appearance. The ulcer does not extend to surrounding tissues, it may be present without catarrh of the stomach, and it may occur singly or in groups of two or three. Moreover, the neuropathic theory is probable, because, as a rule, gastric ulcer occurs in neurotics, and especially in young females; although occasionally it is seen later in life, as in this man's case and in that of his father. For these reasons I think it is fair to suppose that the disease is due to a nervous defect rather than to a thrombus or an embolus. Besides, we do not usually find evidences of thrombosis occurring in other parts of the body coincidently with the ulcer; and it seems to me extraordinary that thrombosis or embolism should occur so uniformly in certain individuals of a certain age, at a certain spot, and produce such constant results.

The doctor has suffered from an excess of hydrochloric acid for many years, and this history belongs to gastric ulcer for two reasons: and first, because any distinct local irritation in the stomach which does not cause catarrh of the stomach usually excites an excessive secretion of hydrochloric acid. The passage of the stomach-tube, the use of the gastric electrode, or almost anything which will excite the mucous membrane, will do this. But I am satisfied that the hydrochloric acid is there for other reasons, for when the ulcer is healed and there is no longer any irritation from it you will still find the excess of hydrochloric acid in such patients. I believe that gastric ulcer is almost uniformly associated with that form of dyspepsia which now goes under the name of hyperhydrochloric acid dyspepsia. This is a form of indigestion which gave a great deal of trouble before we understood its nature. To-day it is comparatively easy to control.

When the doctor has not been suffering from the hemorrhage which the ulcer occasioned and the catarrh to which the vomiting gave rise, he has suffered from hyperhydrochloric acid dyspepsia. He is now in perfect comfort, in spite of the abrupt stoppage of the morphine.

Believing that there was catarrh besides the ulcer, the catarrh being due to vomiting, I thought it best to give the stomach complete rest for a time, and the patient was nourished by the rectum for two or three days,—not on account of the ulcer, but because of the catarrh. If he had been vomiting blood without signs of catarrh, I should still have given his stomach rest for the sake of the ulcer,—in other words,

to prevent the hemorrhage. He was given at the same time local sedatives and antacids, which I thought would soothe the stomach and neutralize the excess of hydrochloric acid, which was not simply surmised, but was demonstrated by an examination of the gastric contents. The mixture was

℞ Cerii oxalatis, 10;
Bismuthi subcarbonatis, 20;
Magnesii carbonatis levis, 40.
M. et S.—Half to one teaspoonful every two to four hours.

Under the effect of the magnesia his bowels, which had been constipated, soon became loosened. The mixture was then modified to

℞ Cerii oxalatis, 10;
Bismuthi subcarbonatis, 20;
Cretæ preparatæ, 20;
Carbonis ligni pulv., 10.
M. et S.—Half-teaspoonful every two to four hours.

This had a quieting effect on the bowels and corrected the previous prescription. One might have given, in place of this, five or ten grains of black oxide of manganese (C. P.), either alone or in combination with bismuth and cerium. These agents are gastric sedatives, while chalk and magnesia are antacids. Using these mixtures,—one when the bowels were constipated and the other when they became loose,—the evacuations were regulated, the stomach became quiet, and his pain passed off so that he got along without morphine, although he was still very nervous.

Then he was fed, and the feeding once begun was continued regularly, steadily, frequently, and bountifully. He was given peptonized milk every two hours, first four ounces, then six, then eight. When you begin to feed a patient with gastric ulcer, you must feed him freely, because the presence of even a little food will stimulate the secretion of hydrochloric acid in excess, and thus cause greater irritation of the ulcer. When the feeding is begun the stomach must not be allowed to become empty. After having fed the patient on peptonized milk for a while, I began giving him meats and egg albumen. These were given rather early to this patient, and were continued at short intervals because the albuminoids are very soon changed by the gastric juice into an acid peptone, with a proportionate decrease of hydrochloric acid and thereby diminution of the irritation to the stomach. Theoretically this practice is right, and practically it is right sometimes, as it was in this case; but you must remember that these patients have a tendency to the

over-secretion of hydrochloric acid, and the stimulation of the secretion by the albuminoid food is not always properly counterbalanced by the formation of peptone.

After peptonized milk, I began with eggs in this case, and later added meats, and when I was satisfied that the hydrochloric acid secretion was being controlled and that the ulcer was healed, I began giving starches. Now, some of the French advise starchy food to the exclusion of albuminoids in gastric ulcer, and for the reason that with meats they found the acid secretion increased. I find that while starches do stimulate less the secretion of hydrochloric acid, they are not well digested, and consequently they give rise to fermentation, which, in turn, causes distress and the stomach has no rest. For their digestion starches require a faintly acid or alkaline medium. I believe, therefore, in giving starches late, not until the excess of hydrochloric acid has been controlled. The patient has now come to the point where quite a general diet is taken, and without producing distress. He still has occasionally pain in the right shoulder, but it does not arise from the stomach; it is simply a habit of pain. An analogy may be drawn from trifacial neuralgia produced by a decayed tooth which may be awakened by exposure to cold weeks or months after the removal of the tooth.

For the future we must endeavor to control the hydrochloric acid excess and to prevent the return of the peculiar condition which gives rise to the ulcer. To meet the former indication, he must have recourse to a diet which is most likely to agree with him, which will give him strength and yet not over-stimulate the secretion of hydrochloric acid. That is to say, his diet must be much like that on which he is at present, amplified somewhat, but he must deny himself fruits, which, by fermenting, are apt to cause disturbance, and other substances whose digestion is difficult or which excite the stomach, such as condiments and sauces. This diet, which maintains the quality of the blood and the tonicity of the nervous system and which keeps the body as a whole well nourished, meets in part the second indication also. In addition the patient should be careful not to overdo, should take the proper amount of rest, and if, in spite of all precautions, his blood falls below par,—that is, if he suffers from anæmia,—this also should receive appropriate treatment.

Now, there is another aspect of this case, for the doctor has also a dilatation of the stomach. What brings that about? It is due sometimes to stenosis of the pylorus. We once thought this the usual cause, but we know now that it is comparatively an infrequent cause of dilatation

in proportion to the whole number of cases. I do not think that stenosis, if present at all, is marked in this case, for the patient empties the stomach fairly well. You may suggest that his dilatation is probably owing to relaxation of the stomach, the result of his neurotic condition. This is possible, but there is another way in which I prefer to account for it here. Anything that gives rise to local irritation in the neighborhood of the pylorus tends to produce a spasmodic closure of the pylorus. So, any food coming against the sensitive area of ulceration would tend to shut up the pylorus and in this way produce virtually a stenosis, that we may call a functional stenosis. I think it is in this way that his dilatation has been caused, for it has long given him more or less pain for his stomach to empty itself. Now if we keep the contents of the stomach bland and unirritating and not over-acid, it will be possible for them to pass downward without causing much irritation, and if we keep the stomach free from ulceration it will be able to empty itself comfortably. The use of internal faradization, lavage, or massage will probably not be called for here.

The case is of great interest in showing the heredity of the disease, by occurring in a man instead of in a woman, and in one of advanced years instead of in a young person. It illustrates the tendency of the disease to continue for a long time, the tendency of dilatation to occur with ulcer, and I think it shows also the tendency of dilatation to occur as a result of irritation at the pylorus.

FATTY DEGENERATION OF THE HEART ASSOCIATED WITH A SLIGHT DILATATION.

CLINICAL LECTURE DELIVERED AT THE PHILADELPHIA HOSPITAL.

BY J. M. ANDERS, M.D.,

Professor of Medicine in the Medico-Chirurgical College, and Visiting Physician to the Philadelphia Hospital, etc.

J. H., white, Irish, aged fifty-five years; occupation when young, a farmer, later, a blacksmith. Family history: father died of typhoid fever; mother died at the age of ninety-seven of natural causes. Has one brother, in Scotland, living and well; one sister, well. No family history of rheumatism. Was always healthy as a child. At blacksmithing he worked thirty years, and during that time he was in perfect health excepting for colds, which were generally very severe and caused much coughing. When younger he drank pretty heavily, and then became quiet and steady. He states that his sprees used to cure his colds. Has had five children, three living and two dead. The three living children have no heart-trouble. Has never had rheumatism; had malaria in 1888, lasting several months, after which he had swollen feet for two weeks; since that time, if he catches cold, they always become slightly swollen. Denies emphatically all venereal history. During the spring and summer of 1887 was in a Pittsburg hospital for five months. Symptoms then were palpitation, dyspnœa, slight œdema of legs, anginoid attacks, and feeling of faintness. Was admitted for the first time to medical ward of the Philadelphia Hospital, June 1, 1889; condition then was as follows:

Dyspnœa on slight exertion, especially when going up-stairs or carrying weights, with marked cardiac palpitation, and the return of anginoid attacks, the pain radiating towards the right shoulder; great depression, pulse 50 and irregular; on exertion heart's action showed gallop rhythm. Remained in house until September; then went out, and returned in November. Left again April 15, 1890, and returned August 7, 1890. After admission, was in bed for five weeks; symp-

toms: great dyspnœa, palpitation, and considerable œdema of feet and legs. Physical signs on admission were those of slightly dilated right heart and probably fatty heart. Impulse very feeble, and area of impulse not increased. Pulse was slow, exceedingly weak, irregular, and intermittent. Dulness extended towards the right, a little beyond the normal boundary-line. Œdema disappeared and condition of heart slightly improved; no anginoid attacks since date of last admission. Present condition, December 13: impulse scarcely perceptible, though it can be distinctly felt. Area of percussion dulness slightly lessened, owing to moderate emphysema. On right side percussion dulness extends slightly beyond normal limit; the upper boundary-line is in the fourth interspace. On auscultation very weak, short, distant, first sound; no murmur present, heart-action very irregular and sometimes intermittent.

The history of this case, together with the present signs and symptoms, points to the probable existence of fatty degeneration with occasional slight dilatation. The diagnosis of fatty degeneration, however, can never be made to a certainty. You should examine carefully into the different elements of causation in each case. Let us here glance hastily at the different varieties of this affection and at their well-recognized causes.

Fatty degeneration is frequently associated with valvular disease, especially in those cases in which the walls have become hypertrophied. Now, the condition under which fatty degeneration is developed and the reasons for these changes have been given you when discussing the subject of dilatation. But fatty degeneration also attacks the normal heart, and it is to this variety that the present case apparently belongs, since you will recollect that neither valvular disease nor hypertrophy has been discovered.

The causes, for practical purposes, are divided into two classes,—namely, general and local. Among general causes I may mention chronic alcoholism, profound anæmia, certain poisons, such as phosphorus, arsenic, etc., wasting diseases, failing nutrition of old age, prolonged fevers producing parenchymatous degeneration, etc. Of the local causes the most important are pericarditis and sclerosis of the coronary arteries. In this connection I would have you remember that disease of the coronary arteries is very generally associated with general atheromatous changes, hence its existence or non-existence is in the majority of instances to be judged of by the condition of the radials. From the history of this case it will be seen that three general causes must have been at work,—namely, general atheroma, chronic alcoholism,

and anæmia. Most probably one local cause has been operative,—namely, atheroma of the coronary arteries,—for the reasons which I have before mentioned. Now, malarial anæmia is very apt to lead to fatty degeneration of the heart, according to my own observation.

Apart from the history of the case, we have among the most reliable points on which to base a diagnosis a permanently feeble impulse, a weak, irregular, and at times intermittent pulse,—either very rapid or very slow,—a fatty arcus senilis, the presence of angina pectoris, or, as in this case, of anginoid attacks; at times œdema of the feet, Cheyne-Stokes breathing, and pseudo-apoplectic attacks; additionally we have the physical signs which we previously mentioned in detail.

When chlorosis or chronic anæmia has preceded the coming on of the manifestations of this disease, there ought to be no difficulty about the diagnosis. The latter disease being associated in the present case corroborates strongly our diagnosis. You should always distinguish fatty degeneration from fatty overgrowth of the heart, when possible. Fatty accumulations around the heart occur in very stout persons. Dyspnœa is apt to be constant, though much increased on exertion; cough and asthma are common. The pulse is rapid, weak, occasionally slow; it becomes irregular and intermittent at times, and in my opinion this is evidence of the fact that infiltration has led to fatty degeneration of the muscular fibres. The physical signs do not differ materially from those found in fatty degeneration. The area of percussion dulness, however, is apt to be somewhat increased. Syncope frequently occurs.

The prognosis of fatty degeneration of the heart is, as a whole, very grave, especially in those instances in which the disease is associated with advanced atheroma; it is less grave, on the other hand, when due to anæmia or chlorosis, causes that are frequently in part or wholly removable. Hence your prognosis should be guardedly favorable under these circumstances.

Treatment.—Endeavor to remove the cause. Another leading object of treatment is to sustain the integrity of the heart-muscle. The remedies best calculated for this purpose are iron, arsenic, and strychnine. Such patients should lead very quiet lives, making no straining efforts whatsoever; especially is straining at stool to be avoided. In most other respects the treatment is the same as that indicated for cardiac dilatation. Where digitalis, however, is indicated on account of associated dilatation, it must be given very cautiously.

NERVOUS DEBILITY; PLEURITIS.

CLINICAL LECTURE DELIVERED AT THE RUSH MEDICAL COLLEGE, CHICAGO.

BY NORMAN BRIDGE, A.M., M.D.,

Professor of Clinical Medicine in the Rush Medical College, Chicago.

THIS young man has been here before. Previous to coming here he had been a heavy drinker; had been troubled with vomiting, gastric catarrh, and nervous debility. He complained also of disorder of his bowels,—of having a passage immediately after eating each time. He had lost flesh very extensively and rapidly,—nearly a pound per day. He comes back now to report. He says he feels like a new man.

He was given for the chief treatment the stale bread and milk diet. He has stopped his drinking. He says he feels well; his diarrhœa and vomiting have ceased. He complains of a discharge of mucus from his nose. I do not think that needs any particular attention. He has partaken of this food about five times a day, and often a swallow between-times. The first two days his bowels did not move at all, and yesterday he went out twice. He has gained three pounds in a week. This is a very interesting account. We found him without fever, and I only threw out a hint that he had some zymotic disease that had lowered his flesh. Now he says he has had no trouble with his stomach, no diarrhœa. His digestive system has been at work throughout the whole day, and not simply three brief periods of the day. At the same time each task put upon it has been so light that it could be accomplished easily: hence there has been no longer a tendency to diarrhœa, and he has gained three pounds in weight. While that does not disprove that he had a zymotic disease, it does not increase the likelihood that he had such an affliction, and I am disposed to take it all—that is, the results—as an example of what can be done by proper regimen. He has had a tonic of quinine and strychnine in small doses, and a little pepsin. The case is very satisfactory. It would have been more satisfactory as a scientific study if we could have had him upon this regimen a week without his taking any medicine at all. It would

have been more fair to science, but not so fair to him. Patients want and have a right to get well as rapidly as they can. Let us continue the treatment. Always when you find yourself on the right track, stick to it.

PLEURITIS.

This young man tells a story of having been sick about a month. He has had some pain during that time along the border of the right ribs. Some days he has had it, and some days not. It hurt him to take a deep breath at times; but he was at first in fair general health. He went on with his occupation until about two weeks ago, when he was seized with severe pain in the right side of the chest, and had a good deal of fever, lost weight, and was obliged to quit work. The fever continued for a number of days, but is now about gone. He had profuse night-sweats during a part of this sickness, and expectorated a great deal,—about one teacupful a day. The expectoration contained no blood, but was mucoid in appearance, and some of it had a darkish color, not black; there was evidently no pus in it. The man has now about regained his normal temperature. He is now breathing over forty times a minute; his pulse is nearly eighty-four. He is short of breath, —that is, he has had dyspnœa since he got up. Vocal fremitus over the lower part of the right chest is nearly abolished. Percussion reveals flatness on the right side, up to a line on a level with the nipple; above that line or under the clavicle resonance is perfect, possibly better than normal. On the opposite side resonance is perfect and beautiful. Let us make a lower-pitched percussion with a hammer that is softer. That gives a sound as flat as percussion over a thick muscle would. That is evidently fluid. Let us see if the level of the fluid will change as the patient changes his posture. The line of the fluid seems to be, as he sits, an inch below the nipple. Yes, as he lies supine it is a little more resonant below the line than it was before, but it does not change greatly. The apex-beat of the heart is to the left of its normal place. Now we will listen over this region. Auscultation over a pleural cavity containing fluid is a very important matter. If it is very full of fluid, if the pleura is tense with fluid, sometimes the lung-sounds are transmitted from the opposite side and over compressed portions of the lungs on the same side that yet contain and receive some air, and an error of diagnosis is easy to be made by a careless study of the facts when those sounds occur. They are never like the sounds on the opposite side, which are puerile. But from a study of the facts of this patient we have been able to make a diagnosis before reaching this point and before making auscultation at all, and

satisfied ourselves that the man must have had a pleurisy and that he must almost certainly have fluid in his right pleural cavity. Percussion has substantially settled that question, but not absolutely. I hear on auscultation a very distant suggestion of the lung-sound. I hear on the left side distinct puerile and normal lung-sounds, and the normal sounds of the heart, which proves again that the apex must be displaced by a change in the position of the heart, and not by a disease of the heart itself with hypertrophy. Percussion over the back reveals flatness in the lower right lung region and resonance everywhere else. Fremitus is more marked on the left side. Now we know to a practical certainty that there is fluid there. Is there any other way to be sure of it? Yes; note the character of the intercostal spaces. They are less depressed on the right side, but they do not bulge. Another important question to settle is the character of the fluid; and he has been sick so long that it would be well for him to be relieved of it even if it is not purulent. We will explore with an instrument and get away a part of the fluid. If it is serum he would probably get rid of it by slow degrees by absorption in the course of a month or two, for if we can withdraw even half a pint—better, a pint and a half—it will start into more rapid activity the process of absorption. It is important to introduce the needle at about the centre of the mass of fluid, but at a distance from the lower part of it, so as not to plunge it into the curved portion of the diaphragm, for fear it might pass through into the liver. But we ought to remember, in this connection, that with some pints of fluid in the pleural cavity the diaphragm is always more or less depressed, and therefore less convex upon its upper surface. This needle has been antisepticized. I pass it through the seventh interspace. Fluid comes, but not very rapidly. The color is a demonstration that we have only serum. Now, having accomplished this much, the needle should be held perfectly still, and the hand should constantly have hold of it. It should not be intrusted to one who would not perceive any movement of the needle. Holding it yourself, you would perceive a motion should anything touch its point. Then if any motion takes place in the needle you must know that it is due to some disturbance at its point. You can then move it or withdraw it, so as to keep its point away from the lung. No considerable harm would come if the lung should be touched, but it is best not to puncture this organ. Various apparatuses have been invented for doing away with the point of the sharp instrument in the cavity while the fluid is flowing; for instance, a set of trocars with canulas somewhat like this used for drawing off fluid from the abdomen, but smaller. There is one

objection to them: it always hurts about tenfold more to plunge the instrument into the chest-cavity than it does to use this plain needle. That is because the canula has to be so much larger than the trocar. We shall continue this process as long as the fluid will flow, or until the patient complains of dyspnœa or commences to cough. The patient begins to cough, and I withdraw the needle. The cough and dyspnœa are the proper expressions of danger, and the aspiration should be stopped when they occur. The patient says he feels very well. I think another insertion of the needle will not be necessary, and it is not necessary to put a plaster over the puncture. [Patient expressed some pain when the needle was withdrawn, after about a quart of fluid had been removed.]

THE ETIOLOGY, PATHOLOGY, AND TREATMENT OF RHEUMATISM, AND ITS RELATION TO RENAL DISEASES.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL.

BY WILLIAM HENRY PORTER, M.D.,

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LECTURE II.

GENTLEMEN,—This patient, as you perceive, has acute articular rheumatism, and this is his fourth attack. The first one occurred when he was only eight years old. The present attack began about four months ago, taking the form of localized pain and swelling, which successively developed in the knees, ankles, and shoulders. His occupation is that of a waiter in a restaurant, and upon investigating the habits of this patient we find that he has always been a very hearty eater, and in the habit of drinking more or less freely of wine and beer. The recurrence of the rheumatic attacks is a noticeable feature in this case.

In reference to the etiology of rheumatic affections, the views here advanced will be found to differ somewhat from those that are commonly taught in connection with this disease. Not, however, that they are essentially new, but they differ in the definite relation which they hold to known physiological and chemical phenomena.

The first question to be solved is the cause of the rheumatic manifestations. Rheumatism is a separate and distinct disease, which has its origin in imperfect feeding, incomplete digestion, and a faulty oxidation and assimilation of the food-stuffs ingested, which finally results in the development of lactic acid as an incomplete or by-product of proteid oxidation. At one time in the study of this disease it seemed clear that rheumatism was the result simply of over-indulgence in the vegetable food-stuffs; because, in the majority of instances, we find that rheumatic subjects are largely vegetarians, or at least are commonly in the habit of indulging freely in vegetables and

fruits, and do not as a rule use much meat. In the vast majority of instances the vegetarian theory holds true; but, unfortunately for completeness in this view, there is found a marked minority in whom excessive meat-eating to the exclusion of the vegetable class of food-stuffs has preceded the rheumatic attack,—too large a number to form an exception to a common rule.

In endeavoring to find a logical explanation that would clearly elucidate this apparently contradictory clinical evidence, this information was obtained from an eminent chemist,—to wit, that when these CHO compounds, such as starch, sugar, and fat, began to be oxidized, the tendency was to have the process completed quickly, that they were rapidly transmuted into their final products of oxidation, carbon dioxide and water, and that they were not apt to stop short and develop into incomplete or by-products of oxidation, such as lactic acid. This information naturally interfered with the original theory that rheumatism was always the result of an incomplete oxidation of the starches and sugars. The clinical fact that many meat-eaters had rheumatism also opposed the theory of incomplete oxidation of the starches and sugars as the sole cause of rheumatism. Further investigation of the subject, and a more thorough study of the phenomena of oxidation of the starches, sugars, and fats, and also of the proteid molecules, showed that even where rheumatism is developed in vegetable feeders the lactic acid does not come from the incomplete oxidation of the starches, sugars, and fats. In all instances the lactic acid is a by-product developed from the incomplete oxidation of the proteid molecule contained in the food-stuffs introduced into the system, either as a meat albumin or as a vegetable proteid.

Rheumatism, nevertheless, occurs more frequently in those who use vegetables to excess than in the more exclusive meat-eaters. This is because the vegetable compounds are more difficult of digestion and require more oxygen and digestive energy than are required to utilize an equal equivalent of meat. When the starch, glucose, and fats are absorbed, they are more rapidly oxidized than a proteid element; therefore, when once within the system, they utilize a large amount of oxygen quickly, and consequently a deficient quantity of oxygen is left with which to accomplish the more difficult and slower task of transmuting the proteid substances into their complete and final products of oxidation,—urea, uric acid, kreatin, carbon dioxide, water, and sulphur salts. It is quite possible by eating an excessive quantity of meat to have the same incomplete metamorphosis of the proteid molecule, provided the amount ingested is greater than the oxygenating

capacity of the system. Thus we see that rheumatism can be produced from either a vegetable or a meat diet, but in both instances the exciting factor is developed out of the proteid molecules. Incomplete transformation of the proteid molecule, however, is less likely to occur when vegetables are largely excluded from the diet-list. This is because the system is more perfectly nourished when upon a meat diet and a moderate amount of vegetables.

In the patient before us to-day there does not appear to be a sharply-defined history of indulgence in any particular class of food-stuffs to the exclusion of other kinds. The history that he gives us is that of a man who has eaten to excess of all kinds of food-stuffs, together with a rather free use of alcoholic stimulants in the form of wine and beer. This patient also leads an in-door life, and takes but little exercise in the open air. The consequence has been that he has taken a much larger quantity of food-stuff than the oxygenating capacity of the system could fully utilize. As a result, every little while nature has rebelled at this imposition in the shape of increased work, until finally the nutritive vitality of the system has become unduly overtaxed, the protoplasmic tissue of the body has become impaired, and a slight exposure to cold or wet, or the excessive use of alcoholics, has excited localized congestions in and around the joint, in the intermuscular planes, and possibly in the membranes of the heart. As these cases advance, oxidation becomes still more imperfect, and the protoplasm of the tissues assumes a vicarious action, and induces at these local points the formation of lactic acid from the proteid molecule. The lactic acid so formed unites with the sodium, potassium, and calcium salts, and forms lactates of the same, and the neutral dibasic compounds, thus reducing the normal alkalinity of the system. These chemical mutations in the tissues intensify the irritation, and often excite a true inflammatory process, with all its attendant phenomena, both physiological and symptomatological; or these saline compounds may become encysted, and form the chalky deposits which are common to certain forms of rheumatism. This faulty transmutation of the proteid bodies, malnutrition, and vicarious action on the part of the protoplasmic elements of the body, together with the local formation of lactic acid and its consequent conversion into the somewhat insoluble lactates of soda, lime, and potassium, should always be kept in mind in the management of every rheumatic case.

Following these local disturbances in the physiological processes there is often a marked congestion of the part, followed by the production of new tissue without any inflammatory action, as seen in many

cases of chronic rheumatism. In other instances a truly inflammatory action is developed, with its secondary degenerations and formation of new tissue. In this way we have a full and satisfactory explanation of the pathological conditions and the symptoms developed in both the acute and the chronic forms of rheumatism of all kinds, whether the lesion be located in and around the joints, in the intermuscular planes, or implicating the heart and its membranes.

This theory also explains the recurring attacks which are so common in certain individuals. So soon as these patients recover from one attack, like the gouty subject, they at once return to their imperfect or excessive diet, which, in due time, is bound to be followed again by another of the so-called rheumatic attacks. In such subjects, at varying intervals, an excessive quantity of lactic acid is developed, precipitated often as the result of a little exposure to wet or cold, or an unusual excess in eating and drinking, until more work is imposed upon the kidneys than they are able to accomplish without damage to their protoplasmic elements, and the attack known by its symptoms as rheumatism is developed, but with an associated renal lesion. With this there are of necessity some renal symptoms, which have led some to consider rheumatism as one of the causes of nephritis. The truth, however, appears to be that the same condition that is causing the rheumatism will at the same time decrease the nutritive vitality of the renal cells and augment the excretory work of the kidneys, and in this way the renal lesion is developed in conjunction with the rheumatism. By bearing these facts in mind we can not only treat the primary rheumatic attack more successfully, but can also arrest the tendency to have such outbreaks, all of which, however, necessitates the patient's following directions to the minutest detail. Where this is done the tendency to rheumatism can be eradicated from the system and from the family.

When such a case comes before us for treatment, the first thing to be ordered is a cholagogue, preferably fifteen or twenty grains of calomel with ten grains of jalap, to be taken at bedtime, or twenty grains of blue mass in conjunction with a compound cathartic pill. Another favorite cholagogue of mine is the following tablet:

Acid. arsenic.,
Hydrarg. bichlor., āā gr. i;
Pulv. ipecac., gr. ii;
Hydrarg. chlor. mit., gr. xvi.
Misce et fiat in tabula no. xv.

Sig.—One of these may be given every three or four hours until free catharsis is established, or one can be given every night, every second night, or every third night, as the condition of the system may indicate.

The object in using any of these methods is to clear the system thoroughly of the old and deteriorated bile. This line of treatment first stimulates the hepatic cells, and then is followed by a truly cholagogue action. The final action of this combination is sedative in nature, during which period the liver-cells are enabled to rest and take on a more active nutritive condition, consequently the function of the liver, taken as a whole, is better performed, and assimilation and nutrition are improved as time progresses.

The next morning, after free catharsis, the following mixture is administered, which is a modification of what used to be known as Professor Alonzo Clark's formula:

Acid. salicylic., ʒiii;
Sodii bicarb., ʒii;
Elixir. gaultheriæ, ℥ss;
Glycerini, ʒiii;
Aquæ, q. s. ad ℥iv.—M.
Sig.—One drachm every two hours.

This makes a freshly precipitated sodium salicylate which is far superior in its action upon the system to any of the salts usually found in the market. This formula did not originate with Professor Clark, but was taken from some German journal, and used by him first in this country, when this drug was originally brought prominently to the attention of the profession as an anti-rheumatic agent. In the original formula there was no elixir of gaultheria, but it was composed of equal parts of glycerin and water. In its original form the mixture was found to be too sweet, and apt to disturb the stomach. Even the salicylic acid, or its compounds in any form, will in certain instances disturb an ordinary reliable and so-called "strong stomach." The addition of the gaultheria and the reduction of the glycerin make the mixture more palatable and more easily retained by an irritable organ. The above-noted remedy is pushed until a decided tinnitus aurium is produced, or until the pains are under control. If the stomach rebels, the salicylate must be given in smaller doses and at longer intervals. By some observers salicylic acid and the salicylates are considered as depressing to the heart, but the evidence obtained at this clinic is to the effect that this drug can be used with impunity without fear of depressing the heart.

So far as my experience goes, there are very few cases of acute articular or muscular rheumatism in which the symptoms will not abate or be entirely dispersed in three or four days, and often in twenty-four hours, when this plan of treatment is faithfully followed;

but without the vigorous action of the mercurial the salicylic compounds often appear to be absolutely worthless. There are a few people, however, who cannot take salicylic acid or its salts in any shape, myself being one of that number. For such oleum gaultheriæ can be substituted in the following shape:

Ol. gaultheriæ, ʒii.
Misce et fiat in cap. no. xii.
Sig.—One capsule every one or two hours.

Successful results have generally been attained at this clinic in the management of chronic cases, also in breaking up the tendency to have recurring attacks of rheumatism upon the slightest indiscretion. To begin with, these patients are placed for a week or ten days upon an exclusive diet of skimmed milk,—eight ounces every two or three hours. This particular form of diet yields the largest amount of nutrition with the least expenditure of oxygen, but it will not furnish the requisite amount of energy to the system; so that while on this diet the patients must not undertake to do a full day's work,—in fact, they are not inclined to attempt the undertaking. In all these cases the whole system gives abundant evidence of a general malnutrition of all the organs. You notice in this patient before you that he is not only rheumatic, but he is decidedly anæmic, and hence we see at once that one important step is first of all to improve this general nutritive state of the system. This, as I have just said, is accomplished first by a rigid plan of dieting; secondly, and to aid the digestive apparatus, we are in the habit of administering the capsules of ox-bile, nux vomica, and quinine, the formula of which I have previously given you.

As you have already been repeatedly told, ox-bile does more to improve the condition of the digestive system than any other remedy used for this purpose with which I am familiar. The urine in similar cases, as well as in this instance, has been carefully examined from day to day while the patients have been under the influence of various drugs, and in no instance has there been such a rapid change from the abnormal and by-products to those commonly regarded as the normal ingredients of the urine as when the patients are under the influence of the ox-bile. In many cases it is absolutely impossible to effect this change so long as the bile is withheld. After a few weeks of the exclusive skimmed milk, buttermilk, or milk diet, the patient is allowed some eggs, a little lean meat, and finally some bread and butter; all this time excluding every form of vegetables, cereals, fruits, sweets, and pastry. This form of diet will furnish the requisite amount of proteid matter to fully sustain the constructive work of the system,

and at the same time it can be utilized with the least expenditure of oxygen and the least loss of vital force. It also, when properly arranged, affords the full amount of heat and energy.

After securing immunity from the rheumatic symptoms, if the patient returns to the excessive use of the vegetable food-stuffs, and persists in eating large quantities of potatoes, or of starch, sugars, and fats, the nutrition of the system will again become so much reduced after a variable period of time that there will be another attack of acute or subacute rheumatism.

This second patient presents similar symptoms. Two days after he arrived in this city, which was about two weeks ago, he noticed that his ankles began to swell and to become painful; then the wrists became affected, and he began to suffer from headache. His appetite, however, has remained fairly good. The urine that he voids is very high-colored, hyper-acid, and contains albumin. Many writers state that rheumatism is one of the causes of nephritis. This opinion, as stated in connection with the first case, cannot be considered as correct in the light of our present knowledge; but, as already remarked, the same conditions that cause the rheumatism may in some instances impair the functional activity or even the histological integrity of the renal organs.

Instead of the four units of urine being produced, $C_{72}H_{112}N_{18}O_{22}S + 139(O) = 4(CH_4N_2O) + C_5H_4N_4O_3 + 2(C_4H_7N_3O) + 55(CO_2) + 38(H_2O) + H_2SO_4$, as from the transmutation of the proteid molecule, occurring in the normal state, only two may be formed, thus, $C_{72}H_{112}N_{18}O_{22}S + 136(O) = 2(CH_4N_2O) + 2(C_5H_4N_4O_3) + 2(C_4H_7N_3O) + 52(CO_2) + 40(H_2O) + H_2SO_4$. Out of the nitrogenous material left, which normally is converted into two more units of urea, there are formed to be eliminated some of the lower forms of proteid katabolins, such as uric acid, and also lactic acid, thus, $C_{72}H_{122}N_{18}O_{22}S + 94(O) = 2(CH_4N_2O) + 2(C_5H_4N_4O_3) + 2(C_4H_7N_3O) + 7(C_3H_6O_3) + 31(CO_2) + 19(H_2O) + H_2SO_4$. In rheumatism the latter transmutation, with the formation of lactic acid, is characteristic. The retrograde process may descend the scale of suboxidation still further until the renal cells are impaired and a form of derived albumin appears in the urine in place of the higher product, urea. This stage of the process, or albuminuria, we have perfectly illustrated in this second patient.

Rheumatism, then, does not produce nephritic lesions, but this general disturbance of the physiological metabolism which produces the rheumatism, if continued long enough, will finally cause a giving way of the protoplasmic vitality of the renal epithelium. This is first evidenced by the presence of albumin in the urine, and later by the presence of casts, and finally by many of the typical symptoms of a renal lesion being added to those of a rheumatic character. It may even go so far that the renal symptoms entirely displace those characteristic of rheumatism.

One of the modern theories regarding the etiology of rheumatism is that it is of bacterial origin, or a germ disease. If we accept this theory, the renal lesion is claimed to be the result of the kidneys having to eliminate from the system the germs or the substances produced by their presence. There is, however, very little, if any, good ground upon which to argue the bacterial origin of rheumatism; therefore it seems more tenable to regard rheumatism essentially as resulting from false feeding and a faulty digestion and assimilation.

This third patient is a porter by occupation, who presents symptoms of the so-called muscular rheumatism. Three weeks ago he developed a sudden pain in the lumbar region. This attack followed immediately upon an exposure to cold and wet. As you see, his tongue is soft, pale, and flabby. This condition of the tongue always indicates a poor nutritive state of the system. These cases, commonly called lumbago, or "crick in the back," are of rheumatic origin. Attacks of lumbago are more frequent in men than in women. This is due to greater muscular exertion in the former and to the construction of the clothing. Men are more likely to strain the lumbar muscles than women, and when they bend forward there is a gaping between the trousers and waistcoat which leaves this portion of the trunk poorly covered, as compared with the rest of the body, and it is, therefore, subject to the local action of cold draughts. This causes a localized congestion in the lumbar region, and in connection with this there is a state of systemic suboxidation. This engorgement of the vessels is often associated with a vicarious formation of lactic acid in the intermuscular planes, which, together with the distended condition of the blood-vessels, causes intense pain, due to pressure upon the peripheral nerve-endings, after which every movement of the muscles of the lumbar region intensifies the distress, and causes the characteristic symptoms of this affection.

These patients need just the same line of general treatment as is employed in the more active forms of rheumatism, if the recurrence of these attacks is to be prevented. First of all, a brisk mercurial purge, then the dietetic and constitutional treatment.

For the immediate relief of the painful symptoms many things can be used. As the pain is a pressure pain, due to congestion of the

vessels and pressure upon the nerve-endings in this region, anything that will relieve the engorgement will temporarily disperse the symptom. The sudden introduction of an acupuncture or long hypodermic needle deep into the lumbar muscles will often relieve the pain instantly. The sudden mechanical injury caused by the needle-thrust into the region in which the nerves are in a hyperæsthetic condition causes a reflex impulse to be carried up to the central nervous system. This is reflected back to the heart and blood-vessels, causing an increased enervation of the heart and vascular walls, by which means the sluggish circulation in the affected parts is set in motion, the congestion overcome, and the pressure removed, and with the removal of the compression there is a disappearance of the pain, often as if by magic. Local cauterization acts reflexly in a similar manner to the needle puncture, and is an exceedingly serviceable method for treating lumbago, and, in fact, all forms of muscular rheumatism. The Paquelin cautery is the most elegant apparatus to use for this purpose; but all of you who are familiar with this instrument know how liable it is to be out of order just when it is wanted most. Therefore by taking a glass rod, from three-eighths to one-half inch in diameter, and heating it in the flame of a Bunsen burner or that of an alcohol lamp, all difficulty is obviated, and cauterization successfully accomplished. When sufficiently hot, quickly and lightly touch the hot rod to the integument directly over the seat of the pain. If the area involved is large, it is well to make the applications along the line of the nerves distributed to the muscles implicated.

One patient out of many is recalled who came to my office from time to time with these attacks, involving the muscles of both shoulders and arms. When he entered the office the pain and stiffness of the muscles were so great that he could not remove his clothing without assistance; yet after a somewhat free cauterization over the affected parts he became quite active and supple, and had no difficulty in replacing his clothing. The relief from this cauterization is not transient, but often gives immunity from pain lasting for weeks at a time. Counter-irritants in the form of liniments can be used, but my experience teaches me that to be of service they must be applied daily, which is apt to be neglected as soon as the parts become a little tender. The following liniment is a very valuable one:

℞ Olei tiglii, ʒii;
Chloroform., ʒii;
Aquæ ammonii fortior., ʒi;
Olei sesami, ʒiii.—M.
Sig.—External use daily.

The application of a fly-blister is often of great service. It must be remembered, however, that unless it is carefully dressed the vesication will be followed by a troublesome and excoriated surface. By applying the ordinary fly-blister, three or four inches square, for a few hours only, being careful to remove it before vesication has actually taken place, and then applying a flaxseed poultice to complete the vesication, quite a thick layer of integument will be raised, which is not easily broken. The blister should not be opened for a day or two, during which time a partial integumental covering will be formed underneath the contained serum. By following this plan an excoriated surface is usually avoided, and the best possible results from the blister are obtained.

Another excellent method of employing continuous counter-irritation by vesication is to use Squibb's vesicating collodion. This is especially valuable around painful and rheumatic joints. It is applied with a camel's-hair pencil in narrow strips, some distance apart, over the affected parts. After two days another series of strips are painted in between the previous applications. In this way, by repeated brushings, continuous vesication can be kept up for days or weeks at a time without producing a raw surface, and yet the counter irritant effect of a blister is steadily continued.

Still another and a very good form of counter-irritation for the chronic cases of lumbago is by means of the ordinary thapsia plaster of French manufacture, that of American makers often containing only a little croton oil and gum, and none of the thapsia resin. So soon as the croton oil evaporates from these plasters they lose their virtue, while a plaster made with the genuine thapsia gum retains its irritating properties for years. Plasters made from the genuine thapsia gum act just as effectually when ten years old as when freshly manufactured. This assertion is based upon repeated experience. In using these old plasters, and even the freshly made, it is a good plan to moisten the gummed side with spirits of camphor until it will adhere to the integument. This thapsia gum acts as a poison to the integument, exciting a surface congestion and a vesiculo-pustular eruption with intense itching. The action spreads over an area three or four times as large as the original plaster applied. This irritation will continue one or two weeks after the application, irrespective of whether the patient removes the plaster or not, provided it remains in contact with the integument long enough to make the action possible. This continued reflex disturbance alters the nutritive condition of the deeper parts and aids in effecting a cure. In using these plasters care should

be taken to thoroughly cleanse the hands after handling the plaster, otherwise some of the gum may cling to the fingers and be rubbed upon the face, as frequently happens. Where this occurs it excites a facial erythema with œdematous swelling, which has often led to the erroneous diagnosis of facial erysipelas. If understood, and a soothing application is used, it does no harm, except the temporary inconvenience and possible scare, the facial erythema usually subsiding in from twenty-four to forty-eight hours.

SCIATICA.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF MARYLAND.

BY I. E. ATKINSON, M.D.,

Professor of Materia Medica and Therapeutics and of Clinical Medicine in the University of Maryland.

GENTLEMEN,—The patient before you is forty years of age, German, married, and a laborer by occupation. He has suffered irregularly since 1870 from rheumatism, which he contracted during the Franco-Prussian war. Four years ago he had an attack of rheumatic fever, and was ill for seven months. At that time he had some indeterminate trouble with his heart, the character of which he is not able to tell us. I may say, in passing, that there are no traces of it left. He has frequently had rheumatism in his leg since then. The present trouble began three weeks ago, while at work. The pain came on suddenly, shooting down the posterior aspect of the right thigh and leg. The pain is present both by day and by night, but is greater when he is quiet than when he is walking. He does not think he has had any fever during this attack. He does not remember to have had scarlatina or dropsy, neither has he had syphilis. His appetite is good. His bowels are regular, and he is free from cough. He is a moderate drinker. You can see that he is a fairly well nourished man, of medium size, and decidedly pale. His tongue is moderately coated. He has no sore throat now, nor is he subject to it. He has no pulmonary or cardiac trouble. His digestive functions are fairly well performed, and were it not for the pain that he has in the right lower extremity he would be able to work. This pain keeps him in almost constant distress. There is a slight increase—not important, however—in the area of liver-dulness. His splenic area is normal. He complains of pain in the neighborhood of the sacro-sciatic foramen, a pain which is increased upon pressure. There is also pain along the course of the sciatic nerve, over the head of the tibia, in the popliteal space, and pressure over this region gives pain. He has no pain, however,

be taken to thoroughly cleanse the hands after handling the plaster, otherwise some of the gum may cling to the fingers and be rubbed upon the face, as frequently happens. Where this occurs it excites a facial erythema with œdematous swelling, which has often led to the erroneous diagnosis of facial erysipelas. If understood, and a soothing application is used, it does no harm, except the temporary inconvenience and possible scare, the facial erythema usually subsiding in from twenty-four to forty-eight hours.

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BY I. E. ATKINSON, M.D.,

Professor of Materia Medica and Therapeutics and of Clinical Medicine in the University of Maryland.

GENTLEMEN,—The patient before you is forty years of age, German, married, and a laborer by occupation. He has suffered irregularly since 1870 from rheumatism, which he contracted during the Franco-Prussian war. Four years ago he had an attack of rheumatic fever, and was ill for seven months. At that time he had some indeterminate trouble with his heart, the character of which he is not able to tell us. I may say, in passing, that there are no traces of it left. He has frequently had rheumatism in his leg since then. The present trouble began three weeks ago, while at work. The pain came on suddenly, shooting down the posterior aspect of the right thigh and leg. The pain is present both by day and by night, but is greater when he is quiet than when he is walking. He does not think he has had any fever during this attack. He does not remember to have had scarlatina or dropsy, neither has he had syphilis. His appetite is good. His bowels are regular, and he is free from cough. He is a moderate drinker. You can see that he is a fairly well nourished man, of medium size, and decidedly pale. His tongue is moderately coated. He has no sore throat now, nor is he subject to it. He has no pulmonary or cardiac trouble. His digestive functions are fairly well performed, and were it not for the pain that he has in the right lower extremity he would be able to work. This pain keeps him in almost constant distress. There is a slight increase—not important, however—in the area of liver-dulness. His splenic area is normal. He complains of pain in the neighborhood of the sacro-sciatic foramen, a pain which is increased upon pressure. There is also pain along the course of the sciatic nerve, over the head of the tibia, in the popliteal space, and pressure over this region gives pain. He has no pain, however,

at the head of the fibula. His patellar reflexes are slightly exaggerated. There is no change in his cutaneous sensibility, but in walking he shows a decided limp. The pain he experiences while walking is similar to a cramp. It is always worse in damp weather, especially at night when he is lying still, so much so that he sleeps but little. The pain is constant, and does not occur in paroxysms. He has no albuminuria.

You will see that he is a muscular man, and there is no sign of atrophy in his extremities. As I press upon the space behind the trochanter major, he has pain. There is no pain in the neighborhood of the sacrum. You notice that the pain is upon the posterior and external surface of the thigh, that it runs down along the external surface of the limb, and that it stops at about the external malleolus. In other words, this pain follows quite accurately the course of the great sciatic nerve and its distribution. The chart shows practically no fever. Upon one occasion his temperature went up to 99⅗°. His pulse ranges from 72 to 90, without any regularity; some mornings it is 90, and some evenings 90. This may result from excitement, exercise, or similar causes.

This other patient is, as you see, also a stout man, a German, twenty-nine years of age, and a laborer by occupation. He has been in America since last March. He is a married man. His father died of phthisis, and his mother of some unknown trouble. He has one brother and three sisters, who are living and well. He has always been hearty until his present attack. He never had rheumatism, scarlatina, or syphilis. He is a moderate drinker. His present illness began last August with pain in the back so that he could not stoop. One or two weeks later the pain extended to the right lower extremity, and was confined to the gluteal and sciatic region, then slowly extended to the fibular side of the leg. It has appeared exclusively upon the posterior and external surface of the lower extremity. He has always been of a constipated habit, his bowels moving every three or four days. He has been in the hospital since September 19, without showing any satisfactory improvement until recently. He gives, as did the other man, the general history of good health. He has no febrile movement, his tongue is only slightly coated, his digestion is good, he is rather constipated, but generally seems to be healthy. I have a memorandum here that in 1881 he spat blood, but it was due to an injury to the back, and no bad effects followed. His heart, lungs, etc., are sound, the various reflexes are normal, and he has no albuminuria. His pain has been very severe. It appears generally along the line of

the sacro-iliac synchondrosis. He also complains of pain at the sacro-sciatic notch, in the popliteal space, over the head of the tibia, also over the head of the fibula and at the external malleolus, but there is no pain on the inner side of the ankle. Another very important modification of sensibility that I wish to call your attention to is that upon the cutaneous surface of his peroneal region there is almost an absence of sensation, very marked in contrast with that of the other side and the inner aspect.

Both these men suffer from sciatica,—a neuralgia of the sciatic nerve. This affection may show itself in limited localities, but very often one can map out very accurately the course of the sciatic nerve and its distributions. You know the sciatic nerve emerges through the sacro-ischiatic notch and then passes down the posterior aspect of the thigh, dividing into the internal and external popliteal nerves. Then the inner one merges into the posterior tibial nerve, passes down, and runs around the foot below the internal malleolus. The external popliteal or peroneal nerve and its branches pass to the external or fibular side of the leg. When the pain of sciatica makes its appearance, it will show itself in part or all along the course of the nerve. There are certain points at which tenderness is especially noticeable. The first is the sacro-iliac synarthrosis. (It is practically always unilateral, and we get these painful points, as a rule, on one side only.) The second point where one experiences pain is at the emergence of the nerve between the tuberosity of the ischium and the great trochanter. Usually the next most painful places are the popliteal space, and again over the head of the fibula, and often at the external border of the patella. Then, again, we have painful points along the external fibular region, but by far the most common of these is just on the outside of the ankle. Again, these painful points sometimes radiate over the dorsal and plantar surface of the foot. The pains that the patients complain of are cramp-like, or, again, like a sensation of great heat or of cold, or a dragging, drawing pain, with occasional exacerbations. Very often, during an examination of the patient, pushing against one of these painful points excites spasms of pain. Sometimes pain begins at one localized point, as, in this man, at the external malleolus. The pain is much exaggerated by walking. At first the man limps, and then walks along pretty well. Or the pain may occur when the patient is absolutely quiet in bed. Under ordinary circumstances there is no alteration in the volume of the muscles, no sign of paralysis, although at times we have other modifications of sensibility than those of pain. Our second patient shows a decided anæsthesia upon the

fibular side of his leg. Sometimes, instead of anæsthesia, hyperæsthesia is observed. Sometimes the patient feels as if the pains were pricking the skin, and at other times as if insects were crawling over it.

An attack of sciatica is often extremely persistent, and it may last for weeks or months. Most cases, however, ultimately entirely recover, although there may be returns of the affection from time to time. Our first patient, for example, states that he has had numerous attacks of rheumatism in his leg. These attacks were very probably sciatic in their nature, and not pure rheumatism. The electric excitability of the leg is often unchanged. In the second patient the muscles of the right side respond to both currents more rapidly than those of the left.

This disease, sciatica, may be a pure neuralgia, but is more often symptomatic of a neuritis, rheumatic or due to other causes. A pure sciatica is a pain without inflammation, a functional disorder of the nerve. It occurs much more frequently in men than in women, and very rarely in childhood and early adult life,—usually between the twenty-fifth and fortieth years. It is rare after the sixtieth year. The causes are many. It may be due to a prolonged contact with cold. A patient may sit upon a cold stone or metallic surface, or perhaps upon a lump of ice, for a protracted period; he may be exposed to the weather for a long time and his legs or feet become chilled. A man may sit upon a narrow fence-rail or a bar of iron for a long time, thus making circumscribed pressure upon the thigh. This may be sufficient to excite an attack of the disorder. Then, again, we have sciatica arising from other causes, within the body. There may be troubles about the pelvis, malignant growths, etc. In women pregnancy sometimes excites it. Sometimes a loaded condition of the bowels, impacted fæces in the sigmoid flexure, or pressure at any point upon the sciatic nerve may be sufficient. Rheumatism is no doubt a cause of the symptoms of sciatica, and occasionally decided symptoms follow that very interesting disease (by which we can always map out the distribution of spinal or cranial nerves) called "shingles." I must make some reservation here, however. Sciatica, as I have said, is purely neuralgia, but we use the clinical term "sciatica" to express the symptoms which correspond to this pure neuralgia, whatever may be its cause. It is hardly proper to call a rheumatic inflammation of the sciatic nerve pure neuralgia, yet the symptoms it excites may be the same. It is hardly fair to attribute the pain which follows shingles to pure sciatica, although it is undoubtedly due to an inflamed condition of the fibrous sheath of the nerve. When we use the term sciatica, therefore, in the strictest sense, we refer to pure neuralgia; but where we are unable to say positively what is the exact pathological change, we call it sciatica, because of the symptoms it presents. You will find, however, that some authorities speak of it as pure neuralgia and assert that the rheumatic sciatica is a disease much more rare than is usually supposed; but I am convinced that it is more frequent than many admit.

The course of the disease, as I have said, is protracted, lasting usually for a number of weeks, often for months, and sometimes for years. Many persons will have attacks which last a few weeks and then disappear. After a short interval there is a recurrence. Just as regularly as certain persons suffer from neuralgia of the trifacial nerve, so we have this recurrence of the neuralgia of the sciatic nerve in many individuals. However, there may be but one attack. The general prognosis is fairly good, although we often get very much discouraged. Of course, when a sciatica is systemic, or originates within the pelvis from malignant growths or otherwise, the gravity of the prognosis depends upon the character of the exciting cause.

The diagnosis is usually easy. There are, however, differences which it is necessary for us to consider. First, the difference between simply pure neuralgic sciatica and sciatica due to certain other influences. In these cases we make our general diagnosis of sciatica, but treat them according to the causes which bring them about. Now, for example, in the case of rheumatism. Patients with rheumatism and with a distinct rheumatic history suffer occasionally from distinct symptoms of sciatica. The etiological diagnosis in these cases is not important so far as the sciatica is concerned, but is important so far as concerns treatment. The recognition of sciatica that follows the various conditions of pregnancy or malignant tumors in the pelvis is, of course, important. The diagnosis so far as symptoms are concerned amounts to nothing, but the diagnosis so far as pathology and treatment are concerned amounts to a good deal. Ordinarily little need be said as regards the diagnosis of sciatica from the neuralgia of herpes zoster cruralis. In infancy the severity of the disease is shown by the eruption. After the eruption fades away, as it will in about two weeks, that is the end of it. In advanced age, however, the history is entirely different. The period of eruption forms a comparatively insignificant feature of the disease. After the eruption is gone, the neuralgia of herpes zoster asserts itself, and the patient may suffer for weeks or months from the most excruciating pain, when all signs of

eruption are gone. I have seen patients suffering from extremely painful neuralgia when all traces of the herpes zoster had gone for months. When we know that a patient has had herpes zoster we have no difficulty in assigning the neuralgia to the true cause; but when he comes to you having forgotten the nature of the trouble with which he was afflicted, we may be led astray and may not recognize the secondary character of the pain. This is another point which can be cleared up by careful examination into its history.

Now as to the diagnosis from troubles that are not sciatic in the strictest sense of the term; for example, the pain of ordinary neuritis. In ordinary neuritis the difficulty can soon be cleared up by modifications of sensibility, the atrophy and wasting of the muscles, and the general paresis and paralysis that are developed. It is a fact that in most cases called true sciatica certain modifications of sensibility show that there is undoubtedly a limited modification of the nerve-tract itself. Our patient here, for example, who shows decided anæsthesia upon the fibular side of his leg would not show it unless there had been some modifications of the nerve-fibre, and this is probably due to certain inflammatory changes involving the sheath of the nerve. In ordinary neuritis, where there is a recognized inflammatory condition, the pain which the patient has at first soon becomes distinguished by profound modifications of nutrition. Then, again, take locomotor ataxia. The patient complains of lightning-like pains in the lower extremities. This may be distinguished in a number of ways. First of all, it is usually bilateral, and the patient shows the concomitant symptoms of locomotor ataxia, with muscular incoördination, absent patellar reflexes, etc.

There is usually not much difficulty in making the diagnosis of sciatica from muscular rheumatism. It would generally be made from the fact that in muscular rheumatism there is muscular soreness. There are no sharply-limited painful points present, and the patient, usually, does not suffer when at rest as he does in sciatica. In ordinary rheumatism the trouble affects the joints rather than the course of the nerve. For example, here is another patient who complains of pain very similar to that of our first two patients. But it is on both sides, and especially about his heels and ankle-joints. He also complains of the knee-joint and back. It is a pain that is exaggerated upon movement, and is associated with redness and inflammation. In chronic rheumatism of the joints, therefore, we do not have much trouble in making our diagnosis.

The most important part for our consideration is the treatment of

these conditions. In many cases, rheumatism especially, the ordinary agents that are employed answer very well. Salicylic acid, the salicylates, etc., often cure rheumatic sciatica very speedily. Indeed, we are very often able to make our diagnosis of rheumatic sciatica by such treatment. Where we wish to treat a case of sciatica it is, of course, important for us to use our remedy cautiously, giving twenty-grain doses of salicylic acid every second hour as long as our patient can take it. By giving the acid in an insoluble form you avoid irritation of the stomach, for the reason that it is probably not dissolved until it is carried past the stomach into the intestines.

Another class of remedies, belonging to the aromatic series of carbon compounds, are antipyrin, antifebrin, phenacetin, etc. These remedies often exert a most decided influence in the direction of cure. This is attributable not only to their influence over rheumatism, but also to their marvellous influence over neuralgic pains. In some cases of sciatica very satisfactory results can be obtained by the proper administration of, say, twenty grains of antipyrin every third or fourth hour. Most cases, however, will resist this treatment.

Certain cases seem to do well under alkaline treatment. As a rule, however, I think you will be disappointed. Iodide of potassium is a remedy which enjoys a good reputation, but its use must be long continued. Where anæmia is present, appropriate tonic remedies are always indicated, such as iron, cod-liver oil, etc. We may go through the whole series without avail, and be compelled to resort to local treatment. Among the best is the ordinary cantharidal plaster. Very good results are often obtained by the application of such a blister to the seat of pain. If your patient complains of the most pain over the sacro-iliac synarthrosis, a series of blisters placed over that point often gives great relief. Or you can place a long blister, eight or ten inches by two or three, along the course of the sciatic nerve and repeat it at brief intervals. Keep up the irritation for a week, ten days, or two weeks. This is often followed by a very rapid amelioration. A very effective method is by hypodermic injection. In many cases the pain is so intense that nothing short of the strongest anodyne treatment will afford relief. The injection of chloroform or ether into the neighborhood of the nerve very often exerts satisfactory influences. Our second patient experienced no benefit until recently, though we had given him everything. Recently we have been injecting fifteen minims of sulphuric ether every other day along the course of the sciatic nerve. I have seen very satisfactory results from this. There may be a slight swelling for a day or two about the spot punctured, but

subcutaneous abscesses, as a rule, do not occur. It should not be forgotten that disastrous sloughing has been known to follow the hypodermic administration of these agents.

A procedure known as nerve-stretching has been resorted to. The scientific way in which to stretch a nerve is to cut down upon it, pass a hook around it, and give it a good pull. This nearly always gives relief, but does not always cure. Another way is to place the patient upon his back and flex the limb strongly until it touches his head. This is very painful, and may sometimes have to be done under the influence of an anæsthetic. In ordinary cases, by attention to the general health, by giving alteratives, iodides, salicylates, antiperiodics, etc., we can usually succeed after a while in curing our patients, and in many cases we can have the good fortune to cure them in a short time. It is a very common disease, one which you will frequently be called upon to treat, and one during which your patience will be sorely tried. Finally, let me caution you to be very careful in your administration of opiates in this affection. The practice has often proved the introduction to the morphine habit.

ACUTE CROUPOUS PNEUMONIA; RHEUMATISM WITH CARDIAC SEQUELÆ.

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY CHARLES CARY, M.D.,

Professor of Materia Medica and Therapeutics and of Clinical Medicine, University of Buffalo.

GENTLEMEN,—I present to you this morning a case of lobar, croupous, or acute pneumonia. This man entered the hospital day before yesterday with the same countenance which he presents to-day, and which is quite typical of the disease. There is intense coloring of the lips and a good deal of flushing of the cheeks, but it is not a healthy color; it borders a little on the mahogany. His breathing, you will notice, is exceedingly rapid, entirely out of keeping with the rate of his pulse, which is at present not far from eighty. He says his breathing is not painful. He is restless, and his manner is that of one in delirium. He has just expressed the desire to be suspended from this gas-fixture rather than to lie on the table, and he is constantly chatting in a way that while delirious is not entirely devoid of reason. Here is a collection of muco-purulent expectoration with a rusty color, due to staining with blood, and, as I pour it from the cup into the basin, you notice its remarkable tenacity. This sputum (about fifty cubic centimetres) has been raised since last night. Generally the sputum is more scanty than this, and you can invert the basin without its falling out.

On raising the patient into a sitting posture, which he says is more comfortable than the recumbent, and percussing over the back, you notice that there is much less resonance on the left side than on the right, although the right is not entirely normal. Notice the man's robust build and apparent vigor. His hands show that he has recently been working. I want you to observe the size of his chest and to mark the kind of man that can be affected with this disease.

The duration of the disease will be so short that after to-day not many of the characteristic signs will be demonstrable. The meagre

subcutaneous abscesses, as a rule, do not occur. It should not be forgotten that disastrous sloughing has been known to follow the hypodermic administration of these agents.

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history obtained is that the patient, a man of about twenty-six, has had a cough for some time, and on Monday night he had a hard chill and an increase in the cough and considerable shortness of breath. On Wednesday he entered the hospital with this flush, to which I have referred as being typical of, although it is not confined to, this disease. Sometimes it is unilateral, and then it occurs on the side in which the pulmonary disease exists. At the time of his entrance there was heard at the base of the left chest very loud tubular breathing. Upon percussion there was great dulness, and vocal transmission was much intensified, almost as if somebody were speaking into the end of the stethoscope. Vocal fremitus on palpation was also greatly increased. Dulness amounting almost to flatness at the lower part of the lung, increased vocal fremitus, increased vocal transmission upon auscultation, loud tubular breathing, are all evidences of one physical state, consolidation of the lung.

His temperature on admission was 102°, rising later to 103.2°, his breathing was nearly forty, and his pulse eighty. There are very few forms of disease in which we get that peculiar relation between the respiration and the cardiac pulsation. It is not so in phthisis, for in that disease the pulse is also rapid. In severe cases of pneumonia sometimes the patient will breathe sixty or eighty times to the minute, the respiration keeping pace with the heart-beat. What is the nature of this kind of consolidation? This we have to determine entirely by post-mortem examination, and when those anatomical lesions are witnessed they are unique. There is in the early stage of the disease harsh breathing over the entire chest, harsher breathing over the lower part of the affected lung, which is usually the right, and accompanying the breathing a very fine crackling sound, which is called the crepitant râle. It is heard generally at the completion of inspiration. It is sometimes not heard unless you invite the patient to take a prolonged forced inspiration. It is not a pathognomonic sign of the disease, although it has been so booked in the past. The crepitation is likened to the sound elicited by rubbing hair between the fingers near the ear, although the comparison is not very good. The crepitant râle is due to the forcing apart of the air-cells by inspiration. An absolute infiltration of the walls of the air-vesicles occurs, and there is in the vesicles a slight exudate, which, being sticky, causes a little sound when the vesicle walls are drawn apart by inspiration. As the exudation increases, however, all breathing ceases. You simply get transmitted sounds from regions where breathing is going on, as, for example, the larynx and the larger patulous bronchial tubes. As the

air blows over the end of a bronchus it produces a hollow, tubular sound, in character not unlike that which I get from blowing across the end of an uncorked bottle. The peculiar exudate in the air-cells is such as to give the disease the name of *croupous* pneumonia. Other forms of pneumonia may be set up by traumatism to the lung or by simple inflammation, but croupous pneumonia is caused only by infection. The material in the lung is just like that which you saw on the patient's tongue, and microscopically I do not believe you could distinguish them. The disease is due to a special micro-organism, the pneumococcus. The disease seems to be a local manifestation of a general infection, just as diphtheria or scarlet fever is a general disease with local manifestations in the throat and skin respectively.

As soon as the bloody exudate has been poured out into the air-vesicles to any extent, the lung becomes heavy, sinking in water, and is as solid as liver and is called hepatized. During this stage of the disease the patient is, as a rule, more comfortable than in the earlier stage, for the disease is, so to speak, satisfied. This stage may last a few hours or a few days. Then one of two things occurs,—either a very rapid absorption, or a tendency to breaking down, which is called gray softening. When gray softening occurs, we find on pressure oozing from the lung of blood-corpuscles, which seem to have undergone a whitish softening, and the blood comes out grayish and a little like pus, and it is asserted that an absolute purulent change occurs. It seems to be, at all events, a degeneration of the primary products of exudation. After purulent softening has occurred, the patient will never regain a perfect lung. If resolution occurs before this change sets in, the lung is not damaged. In spite of the fact that this material is purulent in its appearance even when resolution takes place, abscess rarely occurs in this form of pneumonia. Absorption is quite rapid, or else the sinking of the patient is quite rapid, so that the patient usually either recovers entirely or dies. If gray softening once sets in, you may expect that the patient will be taken from you. The disease seems to terminate by resolution in a crisis, and after a duration of from two to six days suddenly there is an improvement. Tubular breathing disappears, and the so-called crepitant râle redux occurs. This râle is coarser and moister than the first râle heard, and is formed in the smaller bronchial tubes, not in the vesicles. In two or three days normal breathing is restored in the lung, and recovery has virtually occurred. Sometimes, however, there is a stationary period, dulness continues, tubular breathing ceases, expectoration stops. This means that the bronchus has become filled up so that there is no orifice for the air entering the other lung to blow over,

and hence the tubular breathing disappears. You will get increased vocal fremitus just as before, and probably the vocal transmission is greater than normal, but not as great as when the bronchus is open, since the bronchus itself is the medium in which vibrations occur before they are brought to the consolidated lung.

The cerebral symptoms which this man presents are not uncommon. Meningitis may occur with pneumonia, although it is rare. This general disease seems to attack fibrous structures, and hence it involves occasionally the meninges and the pericardium as well as the pleura, which is typically inflamed at the same time as the lung. As in any disease of an infectious character and general nature, not infrequently we find evidences of irritation of the kidneys during pneumonia. There may be albuminuria, and the urine is almost always scanty and high-colored.

The difference between lobar or croupous and lobular or catarrhal pneumonia, aside from the pathological and anatomical differences indicated by their names, is that a catarrhal pneumonia is always secondary to a catarrhal inflammation of the bronchial tubes, whereas a croupous pneumonia is primary. Last winter, on account of the presence of the grippe, we had a good many cases of pneumonia, but they were mostly of the catarrhal type.

I do not think we can describe any routine treatment of croupous pneumonia. In many cases there is an extremely high arterial pressure, with hard, bounding pulse, and in such cases blood-letting would do good. I have never practised this, however, believing that the same result can be obtained by reducing the volume of blood through purgation. Along towards the close of the disease the patient seems to die of absolute heart-failure, and on that account I should recommend expectant rather than radical treatment. In any disease in which there is blood-poisoning, including infectious colds or influenzas, I call on the bowels to remove the poison whenever I can. In pneumonia there is a certain amount of pleurisy, causing a stitchy pain. The use of opium for this as well as to slow down the breathing is of the utmost importance. Poulticing the chest also detracts from the blood in the pleura and relieves the pain. This man has had no opium, however. I put him upon ammonium carbonate, repeated every three hours. The antipyretic plan of treatment has not been indicated. The Germans in following out this plan of treatment even go so far as to plunge pneumonia patients into ice-cold water; but I consider such treatment dangerous. If antipyretic treatment is needed, acetanilide, antipyrin, etc., are to be preferred to the cold-water treatment. I have every expectation that this man will recover.

Case II.—This is a case of rheumatism occurring in a boy of sixteen. The disease is now rather subacute, but it has left behind grave cardiac lesions, whose evidences you see in the pallor of the lad's countenance and in his general malnutrition. This knee has had quite a synovial effusion, but the only relic which you see of it now is the slight amount of fluid, which becomes noticeable when I force the fluid from one side of the patella to the other, or as I press the patella downward, causing the fluid to appear at either side.

With my fingers over his left clavicle I can distinctly feel a murmur or a thrill, and yet the boy is not conscious of his heart. When I place the finger in the fifth interspace outside the nipple-line, I find the apex, and the beat is so forcible that my hand rises and falls with the cardiac impulse. The apex is at least two inches downward and outward from its normal position. On percussion I find that the area of dulness is considerably enlarged outward. Considering the amount of back pressure and extra strain thrown upon the heart by the valvular lesion, which is a mitral regurgitation, the heart is doing its work quite well. We cannot treat the heart directly for the organic valvular lesion, that being something which lies outside the domain of medicine, nor does the heart need tonic treatment. Do not think that digitalis and heart disease are so intimately associated that the presence of the latter is a uniform indication for the administration of the former. Digitalis and other heart tonics of similar action are not specifics for heart disease in general, but are called for only when the circulation of the blood is imperfect and when the heart needs something to spur it on to greater activity. The boy needs general treatment, however, which will tend to enrich his blood. An altered and thinned state of the blood may cause valvular leakage, and by improving the condition of the blood the valvular leakage may be lessened, and the nourishment of the heart will thus be improved. I propose to keep the boy in bed so as to enable the heart to contract and lose whatever element of dilatation there may be in its condition. The balance whose turning in cases of heart-disease tends to make the patient grow worse or better is usually very even, and I think that in this case it is turning downward. If I can throw the balance the other way but a trifle by resting and improving the nutrition of the heart, I may set the vital process going towards improvement. In this way we may improve his condition so much that he will be able to have some mild sort of occupation. He has been a bell-boy in a cheap hotel, and I scarcely know any business that would be more contra-indicated in such a case.

Neurology.

EPILEPSY TREATED BY ANTIPYRIN; ANGINOID ATTACKS DUE TO SUBACUTE INFLAMMATION OF THE AORTA.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL, PHILADELPHIA

BY WILLIAM PEPPER, M.D., LL.D.,

Professor of Medicine and of Clinical Medicine in the University of Pennsylvania.

GENTLEMEN,—I bring before you this morning a young woman who has been under observation for two years and four months. She came here with a distinct history of epilepsy, from which she has suffered for three or four years. The attacks, however, were not frequent at first, occurring only at intervals of seven or eight months. They always occurred at night, and the paroxysms were evidently severe. She was twenty-four years of age when the spells first began. When she came here she had been under medical treatment for some time and had taken a great deal of the bromides. She was so much depressed, anæmic, and weak from the effects of these agents that, as the spells occurred only once in eight months, it seemed unwise to continue this drug, and we asked ourselves if there was not some way of eradicating this tendency without keeping the woman for long periods of time under a remedy so depressing. This is a point of great importance, to which I have alluded on other occasions. We therefore carefully regulated her diet and the hygiene of her daily life. We insisted that she should eat slowly and chew properly, and we regulated her rest and work. In addition we gave her antipyrin in five-grain doses at night, in the hope that it would exert such an influence upon the peripheral vessels as to prevent them from passing into the state of spasm which appears to constitute the initial stage of a convulsion.

Our belief is that the first stage of a convulsion is associated with a spasmodic condition of the minute arterioles and peripheral vessels, as is shown by pallor of the surface. When the physiological action of antipyrin was discovered, it at once suggested itself as a preventive of convulsions, and it has been used in many diseases presenting such conditions. Phenacetine has been used in the same way. I am forced to say that, like all other drugs, it has proved itself most irregular, sometimes doing a great deal of good and at other times exerting no apparent effect, and where its use is associated with a thorough regulation of the details of the patient's life it is impossible to determine how much is due to the latter and how much to the use of the drug. This patient has never had an attack since she was put under this treatment. For a little while after she came here she continued to have what she had before and what she described as "little things in her head," which appeared to be attacks of *petit mal*. Her general health has improved, and she has gained in weight and flesh. She also took a pill of proto-carbonate of iron and carbonate of potassium after each meal. This she has continued. The antipyrin was taken steadily for two years. For the past two and a half months it has been stopped, and she has had no spell. The disease is evidently checked, but how far this can be considered a cure I cannot say. How far this woman could overwork herself, be imprudent in her diet and disturb her digestion, without bringing on a spell, I neither know nor want to know. I want her to go on carrying out the same course of life she has been doing and gradually to drop all medicine. I prefer to depend more upon hygiene than upon drugs. We shall have her continue to report to us every two months.

ANGINOID ATTACKS.

This patient, T. J. R., forty-five years old, comes from a neighboring town. He was a letter-carrier for five years on a long suburban route, carrying a heavy bag. For the past six years, that is, since a year before he was appointed to the position of letter-carrier, he has had attacks of distress when walking. He has been in the habit of taking his meals irregularly, eating rapidly, without chewing the food properly. Six years ago he began to notice that on walking he had a sense of pressure in the pit of the stomach, and that if he continued it went into the arms and down to the wrists and sometimes to the fingers. It was worse on the left side. He would then have to stop walking for a minute or so, when the feeling would pass off. After he began work as a letter-carrier his business tended to make it worse,

and he suffered more or less every day, sometimes having to stop one or two dozen times on his route. As soon as he rested for a while it went away, and when he is quiet he has no attacks. He raises a more or less bitter liquid, especially after meals. He has been temperate in regard to the use of alcohol, but has used tobacco to a certain extent. The pain continued to grow worse, and three months ago he gave up his business on account of it.

A case of this kind of course raises the question of diagnosis as between some vascular lesions, some gastric lesions, and a pure neurosis. The symptoms are anginoid in character. The spells are not true spells of angina pectoris. They begin in the epigastrium and extend upward to the shoulder and down the arm. True angina comes on in the præcordial region and extends chiefly to the left arm, but it may extend down both arms. The attacks here do not come on suddenly, like the spells of true angina pectoris. They are not attended with the same sense of constriction and inability to breathe, and often sense of impending death, that mark true angina; but these attacks are brought on distinctly by exertion. He can bring them on at will; when he wills to be quiet he has no attack. You meet with a great many more cases like this than of true angina pectoris. These cases last almost indefinitely and are capable of great relief, while true angina pectoris is attended with great danger of sudden death.

The first question that you wish to settle in a case of this kind is whether or not there is organic disease of the heart. You meet with many cases like this where the examination shows lesions, especially about the aorta. You find an aortic murmur, and with this you may find hard radials, arcus senilis, and accentuation of the second aortic sound, and all the evidences of a high degree of atheroma of the aortic arch. This man has a good deal of arcus senilis for a man forty-five years of age. There is quite a crescent at the upper part of the cornea. This is due to fatty degeneration of the layers of the cornea, and it is an indication of degeneration of the tunics of the arteries, which is often associated with atheroma. When this is found with hard radials and hard carotids it is suggestive of atheroma of the aorta. You, however, meet with many people with arcus senilis who enjoy such excellent health that it is evident that the aorta is not diseased. When you have hard radials and resisting carotids you may conclude that the aorta is involved, for the disease usually affects the aorta before the radials. In this case the radials do not seem to be hard. It is especially where there are evidences of disease of the aortic valve and stiffness of the aortic arch that these symptoms are present. The explanation is easy.

The patient attempts to walk or exercise; this stimulates the circulation, and the heart acts more energetically; but when it tries to drive a larger amount of blood into the aorta, the walls of which are stiff and unyielding, the heart beats against a resisting wall, and a cramp of the heart or a cramp of the aortic wall results. This causes pain, and the patient is compelled to rest until the circulation quiets down. As soon as he exerts himself beyond a certain point the pain returns. This pain is evidently due to the inability of the aorta to expand in response to the vigorous action of the heart due to exertion. You find these symptoms in all degrees. Some can walk but not run. Others cannot walk a few steps without having this anginoid pain. In this case the heart-sounds are clear. There is a little accentuation of the second sound over the aortic area, as though the valve was a little stiff. The radials are not distinctly hardened. The carotids are not resisting. There is no cardiac murmur. There is no evidence of organic heart-disease or of marked atheroma of the arch of the aorta. We find these cases often with lesions of the aortic valve and arch of the aorta; but we find them also in people who have no such lesions or in whom we cannot demonstrate them. In such cases I am sure there exists at times simply a slight degree of arteritis, and that the aortic walls are irritable and are tender on account of a low grade of irritation. I have seen patients who after a mountain trip or a fatiguing hunting expedition have begun to present such symptoms as we have here and who have become so bad as to have to be confined to bed. Under proper treatment they have recovered and remained well. I have seen patients who presented alarming conditions and for months at a time have had such symptoms in an aggravated form, but who for ten or fifteen years have been well, with no return of the symptoms. There is then a condition of subacute arteritis, affecting the tunics of the aorta principally, possibly due to rheumatism, gout, syphilis, or over-exertion, where the artery becomes tender, and when the heart drives more blood into it, forcing it to expand, it passes into a painful state, compelling the patient to rest until the heart quiets down. When you meet with a case of this kind in a comparatively young man, and when the disease is distinctly of this mechanical anginoid type brought on by exertion, passing away when the exertion ceases, and when the examination fails to show organic valvular disease or evidences of atheroma of the aorta, such a case is hopeful in its prognosis, and should be treated on the supposition that there exists a subacute irritative condition of the root of the aorta. Such a case should be treated by graduated exercise, and if it is very bad you may be obliged to put

the patient at absolute rest. I have been forced to keep patients in bed for sixty days. The diet should be carefully regulated. You do not want the patient to have too much blood, and you want no flabby tissue to oppose resistance to the action of the heart. Counter-irritation over the aorta by blisters or the light cautery should be employed. The patient should be put on long courses of iodide of potassium with small doses of mercuric bichloride, or of iodide of potassium with sodium salicylate. Under the hygienic treatment, which, after all, is as efficacious and as important as the medicinal treatment, and with counter-irritation, we may hope to see such patients recover. We would not begin the use of nitrite of amyl, nitro-glycerin, and such agents. It is not a neurosis, as angina pectoris sometimes is. This is dependent upon a definite physical change; it is a definite mechanical condition; and we do not want to accustom the patient to these drugs, to which he would quickly become addicted, but we want to get rid of the local condition that is present. This patient should be urged to take gentle exercise, but never sufficient to bring on the pain. He should try to tone down in regard to his flesh, take proper food and chew it thoroughly. Such cases as this are numerous, and you will usually find that if you treat the patient properly you will be able to give him a great deal of relief, if not entirely cure him.

OBSTETRICAL PARALYSIS; MULTIPLE NEURITIS AND ITS RELATION TO DISEASES OF THE SPINAL CORD.

CLINICAL LECTURE DELIVERED AT THE HARVARD MEDICAL SCHOOL.

BY JAMES J. PUTNAM, M.D.,

Instructor in Diseases of the Nervous System, Harvard Medical School, Cambridge, Mass.

GENTLEMEN,—In a previous lecture[1] I spoke of the so-called obstetrical paralysis of infants, due to injury of the nerves of the brachial plexus, especially the fifth and sixth, and I referred to the important suggestion made by my colleague, Dr. C. F. Carter, that this form of paralysis is not due to pressure, as has been heretofore supposed, but to the stretching of the cervical nerve-roots on account of the position of the head during labor, and especially under traction. I refer to the subject again now merely to introduce the photographs of a striking case which has recently come under my care in the Massachusetts General Hospital.

It will be remembered that the muscles usually affected are those which rotate the arm outward, abduct the arm at the shoulder, and flex the forearm, frequently also the extensors of the forearm, and sometimes the extensors of the hand and fingers. Usually the child's arm hangs close to the side, extended, and rotated more or less strongly inward.

In the case illustrated by these photographs the arm is drawn up, as you will see, by a strong contraction of the upper portion of the pectoralis major and probably the anterior part of the deltoid, though it is difficult to determine this by the finger. The contraction is so strong that some effort is required to overcome it. The arm can be drawn down into its normal position, but quickly returns to that represented in Figs. 1 and 2, evidently because the antagonistic muscles are para-

[1] Published in the second volume of the first series.

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lyzed. This contraction is likely to be an important element in the case, because if it is allowed to persist adaptive shortening will undoubtedly take place. The pectoralis major receives its nerves from the sixth cervical root, together with most of the other paralyzed muscles, and it must happen very rarely that a few fibres should escape in this way while the rest are injured.

MULTIPLE NEURITIS, AND ITS RELATIONS TO DISEASES OF THE SPINAL CORD.

I do not propose to give in this lecture anything like a complete account of multiple neuritis, but rather to call attention to some of the more important developments of the last few years. The whole subject of multiple neuritis is one of immense importance, partly from its intrinsic interest and partly because the neuritis may be associated with serious diseases of the spinal cord and brain, while, on the other hand, these diseases may be simulated without really existing.

The peripheral nervous system in the aggregate represents, diffused over the whole cutaneous and muscular area of the body, the functions which in the central nervous system are concentrated into a small space. It seemed easy enough to understand how a limited lesion of this small space, occupied, for example, by the spinal cord, should give rise to symptoms affecting a large peripheral area of the body, but it was harder to believe, and until less than twenty years ago it was not believed, that the peripheral nerves representing this large area or large segment of the muscular system could become primarily and simultaneously affected so as to give rise to symptoms closely simulating those which the lesions of the central nervous system would have produced. Yet this turns out to be the fact, and inasmuch as the prognosis in a case of peripheral neuritis is usually far better than it is in disease of the central nervous system, and as the treatment is to a considerable extent different, it becomes our duty to learn to distinguish, so far as this is possible, the cases in which the peripheral nerves alone are affected, and, furthermore, to recognize under what circumstances the central and peripheral nervous systems are liable to be affected together.

The position of the nerve-fibre, as compared with the rest of the nervous system, is a little peculiar, and it is evident that we have still a good deal to learn about it. The natural way of regarding it is as an appendage of a nerve-cell, a prolongation of a nerve-cell process. Cut off from the nerve-cell it degenerates, but while in connection with the nerve-cell it may be called into activity by faradization for pro-

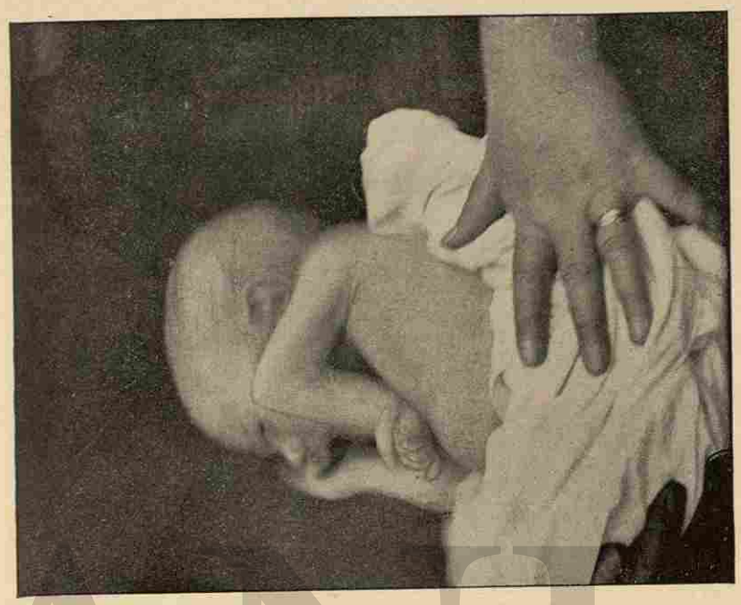


FIG. 2.—Lateral view of the same infant. The arm is drawn up by the action of the pectoralis major.

FIG. 1.—Paralysis of left arm, due to injury of the brachial plexus.

longed periods without showing material signs of fatigue,[1] while at the same time its independent nutritive changes appear to be but slight. In view of these facts it was hard to believe that a nerve-fibre possessed the delicate chemical affinities which seemed necessary in order that it should fall prey to any considerable variety of diseases. The brilliant investigations of the last twenty years have, however, brought to light an immense number of important facts, of which the following are perhaps the chief. The nerve-trunk contains a number of fibres which, for convenience' sake, we will speak of as having different functions, centripetal, or motor, or vaso-motor, the conduction of different sorts of cutaneous sensibility and of muscular and arterial sensibility, possibly special trophic fibres, and the like. It now appears that under the selective influence of disease any one of these systems of fibres may become affected almost alone, though usually several are affected at once. It should be said, however, that in view of the fact that nerve-fibres seem to be conductors merely, and, so far as we know, are capable of conducting equally well in either direction, and even of conducting impulses of varying kinds (though it may be thought on this point our generalizations run ahead of our knowledge), some pathologists cling to the view that when a nerve becomes diseased otherwise than from injury we should not lose sight of the possibility that this disease may be secondary to changes, slight and as yet indiscoverable, in its trophic centres. The number of agents which may thus attack the peripheral nervous system is large, comprising the mineral poisons, arsenic, phosphorus, copper, lead, mercury, ptomaines generated by specific organisms, as those of typhoid, tuberculosis, syphilis, variola, and so on, the poisons generated by gout, rheumatism, Bright's disease, severe forms of anæmia, and many other conditions, besides atmospheric states of which we know little.

The most familiar type of multiple neuritis is the acute, with which every one is now familiar, such as is developed most often by alcoholic excess and arsenical poisoning, but occurs also after typhoid fever and the grippe, and sometimes under conditions pointing to infection, as in beriberi, and among fishermen on our northern shores, as has been shown by Dr. F. C. Shattuck and others and by myself. In this acute form all the functions of the nerve are generally involved, so that the patients suffer from progressive paralysis, beginning usually in the hands and feet, but sometimes beginning in other muscular groups, such as the deltoid; from paræsthesia, hyperæsthesia, anæs-

[1] See first lecture.

thesia, degeneration of the muscular masses, and sometimes of the nerve-trunks.

Besides the acute type of multiple neuritis, in which all the fibres of the nerve are usually more or less involved, there are a number of other types which are equally important. In the first place, the disease may be excessively chronic and the symptoms so indefinite that the diagnosis is difficult or impossible. Then, even in moderately acute cases one set of nerve-fibres are sometimes involved more than the rest; thus we may have well-marked incoördination of all four extremities attended with comparatively little paralysis or pain, and this has even led to the erroneous diagnosis of locomotor ataxia. These ataxic cases have been most frequently observed after poisoning with arsenic, but they are met with also after other cases. I have this winter seen two typical cases of this sort, in one of which the attack followed immediately after a severe cold, and so, very likely, may have been a symptom of grippe-poisoning. In the other case both lead and arsenic were found in the urine. It is not certain, however, that in this latter case the ataxia and neuritis were purely plumbic or arsenical in origin, for it is not uncommon to find lead and arsenic in the urine. On the other hand, it is highly probable that the presence of the arsenic in particular added to the patient's susceptibility, if indeed it was not the whole cause of the attack. In both these cases the diagnosis of neuritis was made certain by the presence of other characteristic signs, especially impaired electrical reactions in one case, and muscular atrophy in the other. In both cases the ocular muscles were affected, and this has been true of other cases of ataxia from neuritis which I have seen. One of these cases had been actually diagnosticated as an acute form of locomotor ataxia, greatly to the alarm of the patient. In the other the persistence of the symptoms indicates that the spinal cord may actually be involved. Another type of multiple neuritis is that which occurs in the ordinary form of lead-poisoning. Here, as a rule, the muscular system alone is affected, although anæsthesias are sometimes met with. In the majority of cases, as is well known, instead of having a paralysis of all four extremities, as happens in most cases of acute and even chronic neuritis, the disease is confined to the arms. On the other hand, a study which I have made of lead-paralysis as seen in children would seem to indicate that in them the legs are as likely to be involved as the arms, though the number of reported cases is but small. Sometimes also, even with adults, forms of neuritis are seen in cases of lead-poisoning which resemble in all essential respects the generalized varieties hitherto considered.

The type of polyneuritis met with after diphtheritic poisoning has its own characteristics, which are too well known to need special mention here.

Is it always an easy matter when we meet with a case of well-marked multiple neuritis to satisfy ourselves as to its origin? To my mind this is far from being true. It sometimes happens that we are able to rule out lead, arsenic, diphtheria, and alcohol, and then are obliged to fall back on the assumption of poison carried in the atmosphere, the nature of which we are as yet unacquainted with. I have already alluded to the fact that occasionally multiple neuritis occurs in an epidemic form among fishermen, not only in the tropics where they are exposed to beriberi, but even in northern latitudes and on our own shores. Again, I have seen an acute fatal case of neuritis where no cause except exposure to wet could be discovered, and I have seen other cases of great interest where even this cause was not present. Thus, an elderly gentleman of exemplary habits and excellent previous health, not gouty or rheumatic, who had been travelling in Europe for pleasure, began to suffer, just before he took the steamer for home, with prickling of the feet. The typical symptoms developed rapidly, and in the course of a few weeks he became so severely affected that he could not walk alone. I feared that he was suffering from a diffuse form of myelitis; yet after a time the symptoms began to mend, and in the end he made a perfect recovery.

The difficulty as regards etiological diagnosis is especially marked in the more chronic cases, which are, after all, the most numerous. In the acute cases we have for those of plumbic origin, as a rule, the characteristic distribution of the paralysis; for the arsenical cases, the occurrence of severe pain, often gastro-intestinal disturbances, and brown or yellow discoloration of the skin; for the diphtheritic cases, the typical course of the disease; for the alcoholic cases, the greater persistency of the delirium and usually a well-marked history. But in the subacute and chronic cases these guides often fail us.

I wish now to turn to the relations between multiple neuritis and diseases of the spinal cord. I have already spoken of the important fact that we are now often able to assure patients, who would formerly have been considered victims of an incurable poliomyelitis or locomotor ataxia, that their disease is one from which they will recover. Yet we should not forget the fact that, even if we have before us a case of polyneuritis, this should not blind us to the possibility that we may have to deal with a myelitis as well. In the first place, in cases of polyneuritis the spinal cord is liable to be secondarily involved. Thus,

in the acute fatal case of my own, where the patient died at the end of a week, enlarged axis cylinders were found in the lateral columns and in the nerve-roots, and collections of leucocytes were found about the vessels in the medulla oblongata. Then, poliomyelitis, especially the adult form, is liable to be complicated from the outset with polyneuritis. I have had an opportunity of making a post-mortem examination in a severe case of this kind, where the gray matter was found threaded with a line of destructive inflammation through the entire length of the spinal cord on both sides. The peripheral nerves were not examined, but the intense pain which the patient suffered, besides the extreme tenderness of the limbs and pain on handling, rendered it certain that an extensive neuritis would have been found. In the next place, the same cause which gives rise to polyneuritis is liable also to cause a poliomyelitis, and perhaps degeneration of the long spinal tracts. Thus, lead-poisoning is liable to give rise to these forms of myelitis as well as to polyneuritis, and the same may probably be said of arsenic and alcohol and syphilis. There is, furthermore, a form of chronic or subacute spinal degeneration, which has been described by Dana and myself in this country, and by Lichtheim, Bennett, and others abroad, which is especially prone to occur in connection with anæmia and other constitutional diseases, and seems pre-eminently to attack feeble persons and women, generally of advanced life.

Now, in some of the cases of this kind which I have observed, well-marked signs of neuritis of the chronic type have seemed to be present, though it must be admitted that in the presence of a chronic myelitis the diagnosis of chronic neuritis is not always to be made with certainty. In one case, however, some filaments taken from the perineal nerve showed evidences of degeneration, and this is not surprising in view of the fact that the anterior gray matter was more or less involved as well as the anterior and posterior roots. It is difficult to say whether in this case the neuritis formed an original part of the process or whether it was secondary to the myelitis, but the studies in the pathology of locomotor ataxia have made it certain that neuritis of the cutaneous nerves may occur at a very early stage.

I have already alluded to the fact that cerebral symptoms are also found in connection with those of polyneuritis. In one case of this sort, observed by myself, but of unknown origin, several foci of encephalitis were found along the walls of the third ventricle. At the time this observation was made but little was known of polyneuritis, and unfortunately the peripheral nerves were not examined, but the spinal cord was free from any marked disease, and the symptoms of

acute polyneuritis were absolutely unmistakable. Besides the cerebral and spinal relations of neuritis, it is also to be noted that cases are occasionally met with where the symptoms to which we should be tempted to give the name of polyneuritis seem to begin in the muscular system. This has been specially referred to by Professor Senator, of Berlin. Again, a few cases have been observed where a progressive form of muscular atrophy, resembling in some respects the typical progressive muscular atrophy of spinal origin, seems to have been due to progressive degeneration of the peripheral nerves.

As regards the treatment of polyneuritis I have but little that is of especial importance to say, but can warmly commend the opinions offered by Professor C. K. Mills, of Philadelphia, in a recent number of the *University Magazine*, of Philadelphia, and I cannot do better than to give you a brief summary of them. After speaking of the extreme care with which acute cases should be mechanically handled, he says that where alcohol has been the cause it should usually be withdrawn at once, but if this causes absolutely dangerous symptoms a little milk-punch may be allowed. Dr. Mills thinks that there are sthenic congestive cases, where inflammation of the brain and spinal cord threaten, which may be treated to advantage by venesection. To relieve the pain and tenderness he recommends hot fomentations or poultices or repeated alternating applications of hot and cold water. Besides stimulants and cardiac tonics he believes in the use of the salicylates or gaultheria in the early stages. In the subchronic or subacute stages more energetic local measures may be used, as local and general baths, with precautions against exposure; stroking, kneading, percussion, massage, and galvanism. As a matter of fact, each acute case is a study by itself, and offers a large field for perseverance and ingenuity, and the physician will have to turn to all the authorities for detailed instruction. In chronic cases the essential thing is to improve the general and local nutrition by every means in our power.

FORMS OF CEREBRAL TUBERCLE.

CLINICAL LECTURE DELIVERED IN THE ROYAL INFIRMARY OF EDINBURGH BEFORE THE UNIVERSITY CLASS OF CLINICAL MEDICINE.

BY G. A. GIBSON, M.D., D.Sc., F.R.C.P. EDIN.,

Assistant Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine at Minto House, Edinburgh.

GENTLEMEN,—Cerebral tubercle, regarded from the stand-point of morbid anatomy, presents itself, as is no doubt within the knowledge of you all, in two forms, which in regard to their structural characters are quite distinct from each other. The symptoms produced by the two forms of the disease cannot, however, be so easily differentiated, and the exact diagnosis in consequence is frequently a matter of some difficulty. The clinical features offered by the two main types referred to are nevertheless in many points dissimilar, and a determination as to the probable nature of the lesion may be attained during life with a considerable degree of certainty. The subject has been much before us in connection with a case lately in ward 25, and, with the hope of being able to place before you the various difficulties surrounding cases of cerebral tubercle, as well as the means of solving the problems which they present, it will form the subject of lecture to-day.

D. J., a little school-girl, ten years of age, was admitted to ward 25 on January 4, 1892, complaining of headache and giddiness, with dimness of sight. The little patient's father and mother are both alive; the former is in robust health, but the latter is weakly. She is the thirteenth of fourteen children, of whom all are alive except two sisters, who died of bronchitis in infancy, and a brother, who died in his fifteenth year of some pulmonary affection. She has never been a strong child. She has suffered from scarlet fever and measles, and at the age of sixteen months she broke the bone of one of her thighs in consequence of a fall. Her social surroundings are by no means good. Her father's work usually keeps him in the west of Scotland, and her mother is employed away from home most of the day, during which time the house is under the charge of a little girl of twelve.

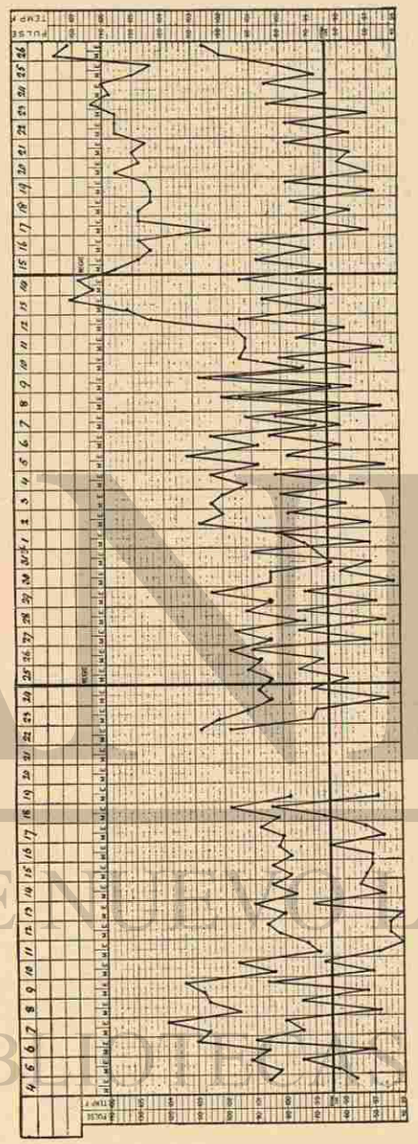


FIG. 1.—The pulse and temperature are recorded on the "Graphic Clinical Chart" of my friend Dr. Handford, of Nottingham. The upper tracing represents the pulse, and the lower the temperature variations.

The present illness appears to have begun during last summer, when the little patient was often feverish and perspired profusely, losing at the same time a good deal of weight. Notwithstanding these symptoms, she attended school with comparative regularity, and seems to have enjoyed a holiday of two weeks which she spent in the country during August. Early in October, when she was standing in the street, the head of a boy who was running past came violently into collision with her forehead. She was stunned by the blow, but did not fall, and, after resting a short time, was able to walk home. From this date, however, she always suffered from headache, and very frequently complained of giddiness; she attended school, nevertheless, with regularity until her sight became so bad that she could not see. The feverish attacks were at the same time more marked, and they were accompanied by great flushing of the skin and considerable breathlessness.

On admission it was noted that the patient, a slenderly-built and imperfectly-developed child, persistently lay on her back in a sleepy state, with her brows knitted. The temperature was then nearly normal, but, as will be seen from the accompanying chart (Fig. 1), it afterwards showed a rise in the evenings, with a remission on the following mornings. The tongue was furred, there was little appetite, and persistent constipation. The pulse varied considerably in frequency, but was always regular and of low tension. Examination of the heart revealed no morbid phenomena. The respiration was somewhat jerky, but on physical examination nothing abnormal could be detected, with the exception of a few scattered crepitations. No symptoms of disease were present in connection with the urinary and integumentary systems.

With regard to the nervous system it was observed that ordinary sensibility was unimpaired. The senses of smell, hearing, and taste were in no way modified. The acuity of vision was diminished, and the visual fields were lessened in both eyes. Ophthalmoscopic investigation showed that both retinæ were hyperæmic, and that there was early optic neuritis. There was internal strabismus of the left eye, pointing to paralysis of the abducent nerve of that side; but, although the movements of the arms and legs were very much enfeebled, there was no evidence of complete motor paralysis elsewhere. The organic and superficial reflexes had undergone no change. The knee-jerk was increased on both sides, and ankle-clonus was elicited easily. The co-ordinating functions were quite intact; the trophic and vaso-motor functions were but little affected at the time of her admission, the

flushing and perspirations being probably accompaniments of the febrile condition. The mental functions were tolerably good.

The patient was sent to us as a case of tubercular meningitis, and at the time of her admission the symptoms seemed so far in accordance with the opinion arrived at by the physician who recommended her that this was the provisional diagnosis. The course of the disease, however, during the two weeks following the admission of the little girl, led us to modify our views as to the nature of the cerebral affection, and to conclude, as you will hear, that the case was one of tubercular tumor.

After the lapse of a week the condition of the poor child changed for the worse; she became listless, and gradually sank into a soporose state. Her mother, being dissatisfied with the course of the illness, removed the patient on January 19, but, finding that it was impossible to give her due care and attention at home, brought her back to the ward three days afterwards. During the next fortnight a gradual increase was observed in the gravity of the symptoms. The little patient became more comatose. Sensibility was lost on the left side of the face, and the mouth was drawn to the right side, showing that the trigeminal and portio dura were implicated. The arms and legs became almost completely paralyzed, pointing to some interference with both motor tracts. The pupils became unequal, the breathing assumed the characteristic cerebral type, bed-sores supervened, and on the 26th of February the poor little child passed quietly away. Up to the time of her death the pupils reacted perfectly to light; there was no paralysis of the third or fourth nerves, and there was no tendency to ptosis. The patient was never absolutely comatose, and even during her last hours could be roused up.

Before proceeding to make any comments on the case you will perhaps allow me, as briefly as possible, to lay before you the recognized pathological and clinical facts as to tubercle of the brain. Tuberculosis affecting the brain presents, as already remarked, two different kinds of lesion,—on the one hand, tubercular meningitis; on the other, tubercular tumor. We shall, in the first place, glance at the anatomical characters of the former.

In *tubercular meningitis* the lesions found after death are most pronounced at the base of the brain. Lymph, usually glairy, but sometimes purulent, is seen between the optic tracts in front and the crura cerebri behind. The inflammatory change is usually seen also over the pons and bulb; it extends along the great vessels, and often spreads by means of the Sylvian fissure so that the convexity of the brain may

undergo changes. The velum, as a rule, is thickened, and the same change almost invariably occurs in the plexuses. The fluid in the ventricles is increased in quantity, and is usually somewhat turbid, or even distinctly purulent. In consequence of the increased amount of fluid in the cerebral cavities, the convexity of the brain is flattened, while as a result of the inflammation the base becomes soft.

Such changes are not in themselves characteristic of tubercular meningitis; they are to be seen also in other forms of cerebral inflammation. The distinctive lesions in the special variety of meningitis at present under our consideration are tubercles, which in the vast majority of cases are of the gray miliary type, but sometimes, although rarely, present the features of the yellow caseating kind. These tubercles are commonly isolated, but in some cases are aggregated in little masses. They are to be seen most frequently on the pia mater in the sulci, on the blood-vessels, and on the plexuses. The great feature of the lesions in tubercular meningitis is therefore the tendency to spread over a considerable area, and to produce extensive changes in the brain.

We must in the next place turn our attention for a short time to *tubercular tumors*. These are usually seen to be rounded masses of caseous aspect and firm consistence. The central part of such a tumor is yellower and harder than the outer portion, which is always grayer and softer. The growth of such tumors appears to proceed by extension along the lymphatic sheath of the blood-vessels, which are obliterated, and this is probably the reason why the inner portion is more solid than the outer layers. Tubercular tumors may become encapsuled or calcified. Tubercular disease of the brain, when it assumes the form of tumor, has, therefore, as might be expected, a more limited distribution than when it is seen as meningitis; but the fact that in a certain proportion of cases tumors and meningitis are found associated together must not be overlooked.

Let us now glance very briefly at the clinical features presented by the two principal forms of cerebral tubercle. In cases of tubercular meningitis a *premonitory phase* may sometimes be seen. More commonly, however, after the disease has progressed to such a stage as will not allow its nature to fail of recognition, it is remembered that, before the development of its pronounced characteristics, many less definite symptoms had been present without attracting special attention. Such a premonitory stage is marked by irregular pyrexia, attended by flushing, pallor, and sweating; digestive disturbances, such as loss of appetite and constipation; nervous troubles, more especially lack of energy,

irritability of temper, and interference with sleep, shown by grinding of the teeth, crying aloud, and lying with open eyes during slumber; and, along with these symptoms, decrease of weight, depending mostly upon loss of the flesh of the trunk and limbs, is rarely absent.

It is still customary, when considering the symptomatology of tubercular meningitis in its fully declared condition, to divide the clinical history into three classical, although conventional, stages.

The *primary stage* is frequently ushered in by rigors, followed by pyrexia; the pyrexia, however, is inconstant. It is of importance to observe that during this phase the pulse is infrequent, full, and regular. Another point, not without considerable significance, is that the skin at this period is almost always dry, even in those cases which have been marked by profuse perspirations during the prodromal phase. Headache is a prominent symptom, and may be detected, even when the patient is unconscious, by the sufferer raising the hand to the head. Giddiness is often complained of, yet it is obvious that it may escape notice. Vomiting is one of the most constant features of the disease. Convulsions may be present, or rigidity of the neck and back may be seen, and the boat-shaped abdomen, produced by tonic contraction of the recti with falling in of the flanks, is often to be observed; vasomotor disturbances are shown by a tendency to flushing of the skin, and by the tache cérébrale, which, although not pathognomonic of this disease, is more common in it than in any other. In addition to these more general symptoms, there may be some with a greater tendency to be focal, such as contortions of the face and squinting. Hyperæsthesia is commonly found, and it may affect the nerves of ordinary sensibility, or of the special senses, producing such symptoms as photophobia. The great features of this phase may be summed up by terming it "the stage of excitement."

The *secondary stage* is marked by a deepening of most of the symptoms, but some of these undergo considerable modification. During this stage the pulse is usually irregular on account of interference with the centres in the base of the brain. The respiration also assumes an irregular rhythm in most cases, so that it has some resemblance to, but is not identical with, the form of breathing known as Cheyne-Stokes respiration. It is probable that this cerebral breathing, as it is usually called, is in part caused by the loss of the influences of the higher tracts of the brain, but it seems likely that in this, as opposed to true Cheyne-Stokes breathing, there must also be some local irritation of the centres in the bulb leading to irregular discharges of nerve energy. Sensory changes, mostly in the direction of diminished

activity, are common, and may affect the nerves of ordinary or of spinal sensibility. Dimness of sight is often observed, and on examining the fundus of the eye the disk is found to be hyperæmic or œdematous. Alterations in the motor functions are even more frequent than sensory disturbances, and, like them, are almost always paralytic. The motor oculi and patheticus are especially liable to suffer, the latter, so far as is known, never being affected alone. When the third nerve is paralyzed alone there is ptosis, with divergent squint and dilated pupil; if the fourth is also implicated the eyeball is turned upward as well as outward. Other cranial nerves may be interfered with in the course of the disease, and different results are produced according to the lesions which are present.

The superficial reflexes present no constant condition; they are sometimes increased, at other times diminished. The knee-jerk, as a rule, is but little altered, and, if changed, the modification is as commonly in the direction of diminution as of exaggeration; ankle-clonus is very rare indeed, even in cases of complete paralysis. This seems to favor the view that the paralysis is dependent on rapid disintegration of the cerebral tissues without time having elapsed for the development of the secondary changes in the motor tracts, upon which increase of the knee-jerk and the presence of ankle-clonus probably depend.

Among symptoms produced by changes in the higher centres may be mentioned aphasia, delirium,—taking the form of illusions, hallucinations, or delusions,—and coma. During profound unconsciousness the patient often utters a loud shriek,—the hydrocephalic cry,—which is probably caused by irritation of the motor apparatus for vocalization, and has certainly nothing to do with volitional impulses. The general tendencies of this phase may be given by terming it, as is commonly done, the "stage of compression."

The *tertiary stage* is characterized by exceedingly variable pyrexia, very frequent but usually quite regular pulse, and extremely irregular breathing. Profound coma is present, with wide-spread sensory and motor paralysis. The optic disks are swollen and œdematous. There is great wasting of the trunk and limbs, but no reaction of degeneration. The main characters of this phase are embraced by the term usually applied to it,—the "stage of paralysis."

The clinical features of tubercular tumors of the brain are for the most part similar to those produced by other kinds of new formations. It must not be forgotten, however, that these are found in patients presenting some of the characteristics of the particular diathesis with which they are associated, and, as tubercular tumors are most com-

monly found in the base of the brain, the symptoms which result are referable to lesions of the structures in that region.

The temperature in cases of tubercular masses in the brain may present many different kinds of curve. As you might expect from the underlying diathesis, there is a well-marked tendency to diurnal variations, but this may be quite overshadowed by the pyrexia produced by the meningitis or encephalitis which may be present along with the tumor. The pulse may have no special characters during the earlier period of tumor formation, but towards the end there is a great tendency towards extreme frequency. (See chart, Fig. 1.) The rhythm of the respiration is usually disturbed in the later stages, presenting the features of cerebral breathing, or even assuming the characters of the Cheyne-Stokes type.

Headache, giddiness, and vomiting are, along with optic neuritis, the most frequent symptoms of cerebral tumor. The optic neuritis may be combined with dimness of vision, but this is not always the case. The other special senses may be affected, but, as with amblyopia, so in their case it is often impossible to attach any special significance to such changes. Such vague *general symptoms* point to the probability that a tumor is present in the brain, but they do not prove it.

The *focal symptoms*, which are in some cases of brain tumor entirely absent, consist of localized sensory and motor changes, which by their distribution give evidence as to the tracts involved, or of alterations in the special functions belonging to particular regions, by which the position of the lesion may be determined. One great law, often shown to you in the work of the wards, is that lesions of the cortex have a tendency to produce, in the first place, spasmodic effects, while changes in the deeper layers, if they give rise to any local symptoms at all, are more likely to cause paralytic effects from the outset. When there is paralysis of a limb there is almost always increased myotatic irritability,—an exaggeration of the knee-jerk and the development of ankle-clonus being rarely absent.

From the brief sketch laid before you of the clinical features of tubercular meningitis and tubercular tumors, it must be obvious to you that in many respects the symptoms have more than a superficial resemblance. In both conditions there may be headache, vomiting, and optic neuritis, and more or less pyrexia is usually present in each. The main differences between the clinical effects of the two lesions are that in tubercular meningitis the course of the disease is shorter, there is more tendency to the development of distinct stages, the symptoms are more extensive, there is an implication of the cranial nerves one

after another, and there is not such an intense degree of optic neuritis. When paralysis of the limbs occurs, it is a sudden process, without increase of the myotatic irritability, in most cases. In short, the symptoms produced by the cerebral lesions are not so definitely localized as in the case of tubercular tumors. It may be added, moreover, that, while both lesions may be associated with pyrexia, the course of the temperature is more definite in meningitis, and the elevation is attended in most cases by a dry skin.

Now, in the clinical history of the patient whose case we have just been considering, the prodromal symptoms resembled those of tubercular meningitis, and the gradual development of the soporose state pointed in the same direction. The presence of profuse perspirations, however, caused us to hesitate, for, even in cases of phthisis pulmonalis, if meningitis supervenes the sweating ceases. The course of the nervous symptoms finally negatived the first impression that the case was one of meningitis. The comparatively slight degree of headache, the absence of any hydrocephalic cry, the limitation of the paralytic symptoms,—particularly in regard to the restriction of these to the fifth, sixth, and part of the seventh nerves of one side,—all these points led us finally to conclude that we were dealing with a tubercular tumor involving the roots or pressing upon the trunks of the fifth, sixth, and seventh nerves. The facts that the arms and legs were paralyzed, that both knee-jerks were exaggerated, and that there was ankle-clonus on both sides, seemed to indicate either that the mass implicated both motor tracts or that more than one tumor was present.

The post-mortem examination was performed the day after death by my colleague Dr. Russell. The heart weighed four ounces; there was hypertrophy of the left ventricle, which was firmly contracted, but no other change.

The lungs were firmly adherent to the thoracic walls; the right weighed ten ounces, and the left six ounces. The right lung had a mass of enlarged caseous glands at its root, and at the lower part of the posterior edge there was a caseous area separated from the lung-tissue. Above this the lung was somewhat fibroid, and presented projecting tubercles, some of which were perivascular and peribronchial. Probably most of these tubercles were in the lymphatics. Throughout the rest of the lung there were sparsely-scattered tubercles. The left lung presented numerous tubercles scattered throughout the pulmonary tissue.

The liver weighed twenty-nine ounces. It had some tubercles on its surface, and was adherent in patches to the under surface of the

diaphragm in consequence of old perihepatitis. On section, it showed a few tubercles. The spleen weighed three and a half ounces, and, like the liver, had tubercles on the surface and adhesions to the neighboring viscera. The pulp was firm, the Malpighian bodies prominent, and there were scattered tubercles throughout the organ.

The kidneys each weighed two and a half ounces, and were healthy, although somewhat congested.

The intestines showed extensive tubercular disease. At the junction of the large and small intestine there was a large transverse tubercular ulcer, and a similar ulcer was observed in the first few inches of the colon. There were numerous small ulcers in the small intestine as far up as the duodenum, and the lymphoid follicles were enlarged throughout.

A prevertebral gland in front of the sixth dorsal vertebra was found to be in a purulent condition, and under this abscess the bone was bare. In other words, there was vertebral caries.

The brain weighed forty-eight ounces. The membranes were healthy, but there was some milkiness about the optic chiasma. No tubercular structures could be seen upon the surface of the brain, but on making sections at different levels it was found that there was a tubercular mass extending from the upper part of the pons as far as the upper part of the bulb. This new formation showed in its typical form the hard yellow structure in the central and older part, and the softer gray texture in the outer and younger part of the mass. The sections made at different levels of the pons and bulb showed, as may be seen in the accompanying figures (2, 3, 4, 5, and 6), which are copies of rough tracings obtained by means of transparent paper, that the mass in the course of its growth had spread into widely different regions of the pons.

Dr. Russell expressed the opinion that the tubercular process had taken its origin in the intestines, and had extended, by means of the bronchial glands, to the lungs, whence it had spread to the brain.

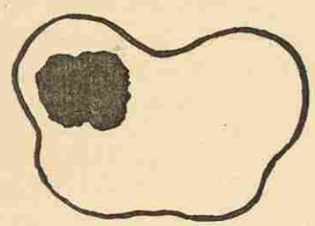
The result of the post-mortem examination in this case fully justified in most particulars the diagnosis to which we had been led, and made clear why there had been paralysis of both arms and legs, with increase of the myotatic irritability. The paralysis of the fifth, sixth, and seventh nerves on the left side was produced by the implication of the nuclei of these nerves by the mass in the lower region of the pons, which at this level extended far into the left half.

It was no part of my intention to-day to add anything with regard to prognosis or treatment; but it may be advisable very shortly to refer

to these aspects of the subject. In cases of tubercular meningitis, while the prognosis is in all stages very unfavorable, and, in those cases which have developed coma, almost hopeless, it is to be remembered that recoveries have not infrequently taken place even in patients whose condition seemed desperate.

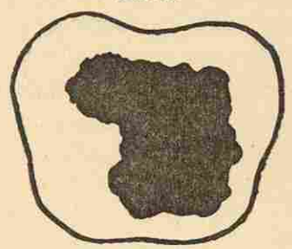
The prognosis in all cases of cerebral tumor must be guarded, and

FIG. 2.



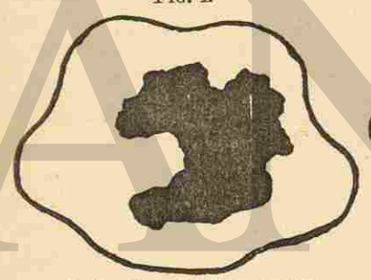
Section through the upper end of pons.

FIG. 3.



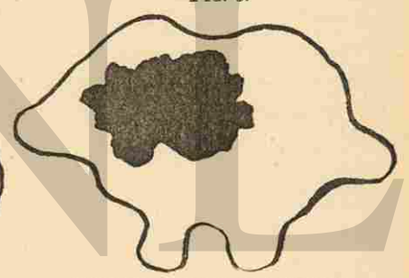
Section through upper half of pons.

FIG. 4.



Section through middle of pons.

FIG. 5.



Section through lower half of pons.

FIG. 6.

Section through upper end of bulb.

THE TRACINGS ARE PLACED WITH THE DORSAL SURFACE UPPERMOST. THE PARTS COLORED BLACK ARE THE DISEASED PORTIONS.

the most important principle to be laid before you is that, next to tumors of syphilitic origin, those of a tubercular nature are the most likely to undergo favorable changes. Occasionally the progress of a tubercular tumor becomes arrested, and a regression may even occur.

In regard to the treatment of tubercle of the brain little need be said at this time. For tubercular meningitis, absolute rest, the promotion of sleep, the application of ice to the head, the internal use of mercurials or iodides, and careful attention to every symptom as it arises, are the principal means at our disposal.

In cases of tubercular tumor the question of surgical interference does not often present itself, since the lesion, as we have seen, is most commonly in the base of the brain. The method of treatment most likely to cause an arrest of the morbid process is the use of tonic remedies along with cod-liver oil and iodide of iron, together with careful attention to every hygienic means.

VERTIGO: ITS TYPES AND TREATMENT.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL.

BY C. L. DANA, M.D.,

Professor of Nervous Diseases in the New York Post-Graduate Medical School; Physician to Bellevue Hospital.

THESE three patients are suffering from vertigo. The first one is seventy years of age, and was well up to a short time ago, when she suffered for the first time from vertigo. The vertigo was of the form we call "objective," or that form in which things around the patient seem to be moving. Shortly afterwards she developed a tremor of her hands and some weakness of the extremities. The arms are extensively involved in this, and there is slight tremor of the head, and slight nystagmus. After suffering from this tremor for two or three months, the vertigo became troublesome whenever she moved around much. It was accompanied by a peculiar humming noise, not in the ears, but in the *head*, a symptom which we call "tinnitus cerebri." She also complains of considerable disturbance of the stomach.

The second patient is forty-seven years of age, is married, and has had several children. She enjoyed good health up to last November, when she had an attack of acute Bright's disease, lasting two months. After recovering from this, she continued to be pretty well until two or three weeks ago, when she began to suffer from sudden and paroxysmal attacks of vertigo. The stomach is not much disturbed, she is not particularly anæmic, and she has none of the ordinary symptoms of uræmia. It is difficult to make some patients understand just what you mean by dizziness. She describes her condition as a "sudden coldness in the head, and then a sudden heat."

The third patient is thirty-three years old, is married, and has had several children. She is of a nervous temperament. She was well up to a year and a half ago, when she had a miscarriage, since which time

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The third patient is thirty-three years old, is married, and has had several children. She is of a nervous temperament. She was well up to a year and a half ago, when she had a miscarriage, since which time

she has been very nervous, irritable, suffers from insomnia, has feelings of pressure on the top of the head, numbness of the hands, pain in the neck, creeping sensations about the body, and all the classical symptoms of neurasthenia. In addition to these symptoms, she tells us now that she suffers from dizziness, from objective vertigo.

Vertigo is, of course, a very common symptom, and one which, like headache, is very difficult to classify, but for practical purposes we can divide these cases upon an etiological basis as follows:

(1) There is a vertigo dependent upon hæmic and cardiac causes, the *vascular* form. This form of vertigo is due sometimes to simple anæmia, and sometimes to a poison in the blood, and hence it is often spoken of as "anæmic" or "toxic."

(2) There is a large class of vertigoes which we call "auditory," because they are due to disturbance of the space-sense branch of the eighth nerve. The auditory nerve is really composed of two nerves,—one the nerve of hearing, which starts from the cochlea and goes to the medulla, then to the posterior tubercles and to the first and second convolutions of the temporo-sphenoidal lobe, and the other a nerve from the semicircular canals, which passes up into the middle lobe of the cerebellum and then passes forward to the cerebrum. This nerve, when injured, causes disturbance of the space-sense, producing vertigo. The most typical form of auditory vertigo is what is called Ménière's vertigo, and is generally due to organic disease of the internal ear, often of syphilitic origin. There are many milder forms of aural disease, anæmias or hyperæmias of that organ, which cause vertigo, so that Gowers considers that one-half of the vertigoes are of auditory origin.

(3) There is a class of vertigoes which we call "ocular," because they arise from disturbance in refraction, or in the muscles of the eye. These vertigoes are often found in cases of astigmatism and of muscular asthenopia, but I consider that a very small proportion are of this origin.

(4) There is a large class due to reflex irritation, as from the stomach, liver, intestines, and the pelvic viscera. These are perhaps the more common forms met with.

(5) There is a vertigo of purely neurotic origin,—*e.g.*, the epileptic and neurasthenic vertigoes. Many persons after studying very hard will begin to have dizziness in the morning, which is promptly relieved by cessation of the work. It is a form of neurasthenic vertigo.

Now, gentlemen, you have heard the histories of these three patients with vertigo, and I shall be pleased to know how you would classify them. Of course it is quite common to find that you cannot

place a case entirely in one category. It is suggested that this first patient has a vertigo due to old age; and it is true that there is a vertigo from obliterating endarteritis which comes on somewhat in this way. The vertigoes which are preliminary to acute softening or hemorrhage are usually temporary, coming on several times in a day, but only for a short time. I think if you had this patient in your office you would not hesitate to say that her vertigo was due to "biliousness" or something of that kind, and there seems to be no doubt that it is of toxic or reflex origin, and due largely to the condition of her stomach. She has hard arteries and a poorly-nourished brain, and hence, if you add to this a dyspeptic condition, there is abundant cause for her vertigo. She has also a gradually developing tremor, chiefly of the hands. The tremor is almost constantly present, is increased upon motion, and is accompanied by sweating and a feeling of fever and weakness. There are three common conditions in which there is tremor,—viz., paralysis agitans, senility, and multiple sclerosis. The tremor in the last-named condition is increased by movement, and is due to small spots of connective tissue in the brain and cord. This patient's tremor either is of toxic origin or is due to paralysis agitans or to senility, for she is too old for multiple sclerosis. In my opinion, she is developing a form of paralysis agitans, and in the course of a few months she will probably show the typical symptoms of shaking palsy.

I shall give her a calomel purge, and follow this with fifteen drops of nitro-muriatic acid three times a day. To secure her *prompt* relief, it would be advisable to give her a powder consisting of twenty grains of bromide of sodium with a little pepsin and ginger, for you know that bromide of sodium is the best symptomatic remedy for vertigo.

Our second patient with vertigo gives us only a history of an attack of Bright's disease. One week ago her pulse was weak and tense, and yet she does not give the ordinary symptoms of uræmia. Her vertigo was produced by a mild uræmic poisoning, and it belongs to the class of vascular vertigoes. The proof of this is to be found in the fact that on giving her one one-hundredth of a grain of nitro-glycerin three times a day the vertigo was promptly controlled, and two days after stopping its use the vertigo returned. I shall again put her on nitro-glycerin, for her pulse is again hard, and I shall also give her iron and spirit of chloroform as a general tonic.

In regard to the third patient, who was neurasthenic, I may say that she is a type very frequently met with in general practice. The vertigoes which occur in women of this class are simply the expression

of the excessive nervous irritability of the subject, combined with the ordinary dyspeptic symptoms associated with this condition. These vertigoes belong primarily to the neurotic group; but dyspepsia, constipation, anæmia, and various reflex causes, such as ocular and auditory troubles, all help to produce it. The treatment is, of course, the treatment of neurasthenia, and this is a subject into which I have not time now to enter. I shall begin her treatment by giving one drachm of fluid extract of valerian and twenty grains of bromide of sodium, three times a day, until the nervous irritibility is quieted, and then I shall prescribe hydro-therapy, electricity, tonics, and proper diet.

DELIRIUM TREMENS AND OTHER FORMS OF SURGICAL DELIRIUM.

CLINICAL LECTURE DELIVERED AT ST. GEORGE'S HOSPITAL.

BY WARRINGTON HAWARD, M.D., F.R.S.C.,
Surgeon to St. George's Hospital, etc.

GENTLEMEN,—There has lately been an opportunity of observing in the wards of the hospital several different kinds of delirium occurring in connection with surgical cases: so that it may be useful to devote a clinical lecture to the consideration of this subject.

In the first place we may notice two cases of delirium tremens, both furnishing illustrations of some characteristic points concerning this disease.

Both of these were men; and it is remarkable with regard to delirium tremens that it is very seldom seen in women. Yet the causes which are usually, and I believe rightly, considered conducive to this disease in men are largely operative also upon women. I am afraid there can be no doubt that there is a good deal of drinking among women, and that their intemperance is often combined with mental anxiety, physical fatigue, lack of rest, and insufficient or inappropriate food. We also see a good many cases of injury among intemperate women. Yet in spite of all this, and of the greater mobility of their nervous system, we rarely meet with a case of delirium tremens among them. I suppose the fact is that though many women are intemperate, yet the actual amount of stimulant taken by them is generally less than in the case of men. Among the poorer classes the money usually has to filter through the pocket of the man before it reaches the wife, by which time there is mostly but little margin for her to spend on drink; and probably the needs of the children act through her maternal instinct as a material restraint on much expenditure in that direction: so that she has to satisfy her craving with less than would content the man.

These men were both strong men in the prime of life,—one a stable-

of the excessive nervous irritability of the subject, combined with the ordinary dyspeptic symptoms associated with this condition. These vertigoes belong primarily to the neurotic group; but dyspepsia, constipation, anæmia, and various reflex causes, such as ocular and auditory troubles, all help to produce it. The treatment is, of course, the treatment of neurasthenia, and this is a subject into which I have not time now to enter. I shall begin her treatment by giving one drachm of fluid extract of valerian and twenty grains of bromide of sodium, three times a day, until the nervous irritibility is quieted, and then I shall prescribe hydro-therapy, electricity, tonics, and proper diet.

DELIRIUM TREMENS AND OTHER FORMS OF SURGICAL DELIRIUM.

CLINICAL LECTURE DELIVERED AT ST. GEORGE'S HOSPITAL.

BY WARRINGTON HAWARD, M.D., F.R.S.C.,
Surgeon to St. George's Hospital, etc.

GENTLEMEN,—There has lately been an opportunity of observing in the wards of the hospital several different kinds of delirium occurring in connection with surgical cases: so that it may be useful to devote a clinical lecture to the consideration of this subject.

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These men were both strong men in the prime of life,—one a stable-

man, the other a mason. Both had been accustomed for several years to drink largely of various kinds of stimulants,—beer and spirits. In both an injury was the immediate cause of the attack of delirium.

Case I.—In the case of the stableman the injury was a fractured leg, and when he was admitted to the hospital he was talkative, and evidently excited by drink. When, however, the fracture had been reduced and placed in splints, and the man put comfortably to bed, he became quiet, and showed no tremor even of the tongue (the organ often most difficult in this, as in other conditions, to control). The night after admission he slept for short intervals after a morphine injection; it was not until the next evening that symptoms of nervous disturbance began to appear. He then became restless, wanting to sit up, his movements and speech were jerky, he refused his food, and later in the evening insisted that he was quite well and must go home. The bowels had been freely opened by a purgative, and a subcutaneous injection of morphine was given, and repeated during the night. He did not sleep, however, but spent a restless and talkative night, and next morning had well-marked delirium tremens. His tongue and certain muscles were tremulous, he perspired profusely, he tried to leave his bed, said he must go home, and addressed incoherent remarks to imaginary comrades. His tongue was moist and coated with a thick white fur; his pulse 110, soft and compressible; temperature 100.2°; urine loaded with lithates, free from albumin; he objected to food. If asked a direct question, he replied rationally, but in a sudden, jerky manner, and then wandered off again into incoherent talk. It was ascertained that he had lately been often out of work, and had been living badly for some months past, and thus, in addition to his drinking, there was impaired nutrition, due to insufficient food, as another predisposing cause of the disease.

You will observe that the immediate effect of the rest and food obtained by his admission to the hospital was a temporary quiet, but that as the second night came on he became fidgety and restless, and, failing to obtain sleep, then became delirious. That is a sequence of events very commonly to be noticed. These cases usually become worse towards night, and when the habitual time for sleep comes, if the needed rest is not obtained, delirium begins. It is therefore desirable to see such patients late in the evening, when the need for sedatives can be best appreciated.

This man, having been freely purged, was given a draught of bromide of potassium and chloral every three hours. He was after some perseverance persuaded to take some strong beef-tea, and subsequently a

sandwich of pounded meat and bread. A screen was arranged round his bed, and a nurse placed in attendance upon him, who by judicious persuasion kept him in bed and fed him and thus prevented the need of apparatus of restraint. Under this treatment he gradually quieted, and the following night slept soundly, and the delirium was at an end.

Case II.—The other man, the mason, was admitted on account of a rather severe and contused scalp-wound, due to a blow from a piece of iron which fell upon him. He was somewhat stunned by the blow, and lost a considerable quantity of blood. So that here we had the complication of an injury to the head, and it was necessary to differentiate the ensuing delirium from such as might be due to intracranial inflammation. In this case the delirium set in the night after the injury, and the following day the man was very noisy and troublesome, so that it was necessary to place him in a separate ward. The symptoms were very similar to those which I have described as displayed by the first patient. The delirium was what Sir Thomas Watson so well named, in his admirable description of the disease, a "busy delirium." The man was constantly talking and fidgeting, throwing off the bedclothes, sitting up and wanting to leave his bed, and every now and then shouting noisily to some imaginary person who annoyed him. The tremor was very slight and only occasionally noticeable; and you must remember that this symptom, though it gives the name to the disease, is not always present or well marked. This man was in a better nourished condition than the first patient, and I think the tremor is more apt to be displayed by the ill fed and badly nourished. So that although the tremulous tongue or hands may often help you to anticipate the onset of delirium tremens, or to diagnose it when developed, yet the absence of this symptom must not lead you to doubt the nature of the attack if the other symptoms are characteristic of it. The man was given a senna draught, which he vomited, and vomiting continued after food during the next twelve hours, but ceased after a dose of calomel. He received an injection of morphine, but did not sleep. It was difficult to count the pulse, because it was very rapid, as one would expect from his active and excited condition. He perspired profusely, his face was flushed, his tongue thickly coated with a moist white fur. The scalp-wound, in spite of frequent disturbance of the dressings, went on fairly well, but suppurated slightly. His bowels acted freely, and after the vomiting ceased he took the greater part of the fluid food offered him. He was then given bromide and chloral in frequently-repeated doses, but the delirium continued as actively as ever, until forty drops of laudanum were administered, followed after

two hours by twenty drops, after which he slept soundly for about eight hours, and awoke quiet and sane.

Now, these two cases are fairly typical specimens of delirium tremens, and, having thus briefly described them to you, I will add a few comments, and then pass to the consideration of some other forms of delirium with which these may be usefully compared and contrasted. I told you at the commencement of the lecture that both these men whose cases are before you had been accustomed to drink largely, and it is in the habitually intemperate that delirium tremens is chiefly met with. A single indulgence in large quantities of alcohol may produce various other harmful effects, such as may result from any other poison, but it does not produce delirium tremens. The nervous system must have undergone some change due to prolonged alcoholic poisoning which renders the drunkard predisposed to the attack. But something more than this is usually needful for the production of the disease, and most commonly this is want of sleep or want of food; and with these is often combined fatigue or anxiety. You will observe that these are all depressing influences, and you will see how easily these may all occur together with intemperance. A person suffering from severe anxiety or grief is apt to sleep badly and to lose appetite; if he is intemperate he will have the less moral power to combat his trouble, and feeling depressed and unable to take food, he is tempted the more easily to drink, and has thus entered a vicious circle.

I saw recently a very bad case of delirium tremens in a delicate young man who had been for some years drinking a good deal, and in whom the fatigue of dissipation had been aggravated by loss of sleep caused by cystitis and irritability of the bladder.

And one of the few cases I have seen among women was that of a lady whose sleep had been prevented by the terrible itching of acute eczema, and who had, moreover, been taking large quantities of alcohol. In other cases nervous shock is the immediate cause of the attack, as was the case in the two men in whom it followed an injury. The shock, however, may not be traumatic, but mental, as from the sudden communication of bad news. Loss of blood, too, may be the depressing influence which develops the disease, and this cause probably contributed something to its production in the second case I have related.

The diagnosis does not usually present any material difficulty. The history is, of course, very helpful when it can be obtained and is reliable; but the true history is often concealed from us or is purposely

distorted. Remember, however, that the sequence of events is first restlessness, then disturbed sleep, then no sleep, then, lastly, delirium: not delirium and therefore loss of sleep, but loss of sleep and therefore delirium.

In the second case, as may not infrequently happen, an injury of the head preceded the delirium, so that the question arose whether the delirium was due to the injury or to drink. But there cannot often be any real difficulty in deciding this. The history, the mode of onset, the character of the delirium, and the concomitant symptoms are entirely different. The delirium of inflammation of the brain or its membranes is more acute, violent, and constant, without the intervals of more or less coherence seen in the drunkard, who will often be recalled for a moment from his irrational jabbering by a question put to him in a decided manner, which he will answer in a jerky, sudden way, but it may be quite reasonably. Moreover, in the case of inflammation, pain in the head precedes delirium, the pulse is not only quick but hard, the tongue is parched and dry, the temperature is high, the skin dry, the pupils contracted, and though there may be muscular paralysis there is no tremor. These conditions are in marked contrast to the soft, weak pulse, the moist tongue, the sweating skin, and the comparative absence of fever seen in delirium tremens.

The prognosis of delirium tremens depends very much upon the ability of the patient to take and digest food. Vomiting is therefore a serious symptom, for, besides preventing the administration of food, it is in itself exhausting. If the delirium is violent, the case is the more serious because of the exhaustion which violent delirium causes. The heart and blood-vessels in drunkards are apt to be deteriorated, and thus are the less able to stand the increased strain upon them. Exhaustion, which is the usual cause of death, thus the more easily ensues, and is often indicated by the change of the delirium from a violent to a low, muttering form. Very severe tremor is a bad sign, especially if tremors occur during sleep. Epileptiform attacks are also of serious import. A serious and not uncommon complication is pneumonia. On the other hand, if food is readily taken and digested, if the patient is young, if the organs are fairly sound and the delirium easily restrained, recovery is to be expected.

And now let us consider the treatment of this disease. You will have observed that the immediate causes which, acting upon the predisposed, bring on delirium tremens, are of a depressing character,—fatigue, loss of sleep, privation, want of food, shock, physical or mental, anxiety, grief. So, again, the symptoms show depression,—the

compressible pulse, the sweating skin, the tremors. The most common complication is a low, spreading pneumonia; the usual mode of death, exhaustion. Obviously, then, the treatment must not be of a lowering character: recovery must be obtained by food and sleep. Food is more important than physic, for if food is freely taken, and the patient is kept quiet, sleep will probably ensue. Of course the more nourishing and easily assimilated the food is, the better; but the patient's inclination must often be to some extent considered. Besides the ordinary forms of nourishment, such as strong soup, milk, eggs, and butter, a very useful food will be found to be pounded raw meat. This should be reduced to a pulp and passed through a fine wire sieve, and may be given on buttered toast or in a sandwich; it will often be acceptable when the fluid foods are refused. Oysters, of course, are also useful. Stimulants should certainly be avoided if possible, though sometimes their administration may be necessary. But if necessary, they are a necessary evil, which should be dispensed with as soon as possible, and used with the greatest caution. A free purge at the outset is almost always useful; and although purgation should not be carried to an exhausting extent, its repetition at intervals will be generally beneficial. You will have noticed that although we do not usually wish to disturb patients suffering with recent fractures by the administration of aperients, yet in the case of the man with delirium tremens and fractured leg we gave aperients with evident benefit. With regard to other medicines, it must be admitted that no rule is universally applicable, and that the drug which has produced sleep in one patient may fail with the next. But, speaking generally, I should say that the most useful medicine is a combination of chloral and bromide of potassium given every two or three hours till sleep is obtained. If, however, the pulse is very feeble, the bromide is not desirable, and then chloral may be given alone, or sulphonal. In some cases, however, full doses of opium answer better than anything, and when the stomach will not retain the drug, morphine may be subcutaneously injected. But when the presence of albumin in the urine shows the kidneys to be damaged, or if there be any pneumonia, opium had better be avoided. If nausea is troublesome, small pieces of ice may be given; but it must be remembered that the continued swallowing of iced water is seriously depressing. It is of the utmost importance that the surroundings of the patient should be as quiet as possible. Light should be subdued and noise excluded, as well as any sources of bodily or mental irritation. A good nurse who will treat the patient with a judicious combination of firmness and gentleness is

a most desirable aid, and will usually render any apparatus of restraint unnecessary.

Before leaving this subject I would remind you that delirium tremens is occasionally met with in persons who are not drunkards, but who have been subjected to a long-continued nervous strain, with loss of appetite and sleep, to which has been added some injury, loss of blood, or other nerve-shock. But there is another kind of delirium, met with in intemperate persons whose blood-vessels are degenerated, which must be distinguished from delirium tremens. This comes on in connection with some febrile disturbance or exhausting disease. We recently had an example of this in a man who was admitted on account of cellulitis of the foot, which rapidly led to extensive sloughing. He had been intemperate, and had atheromatous arteries and impaired digestion. When the discharge became profuse and exhausting, he grew delirious, and continued so for several weeks. But the delirium was quite unlike delirium tremens. He was constantly chattering and throwing his arms about, but he slept frequently for short intervals; he had a high temperature and dry skin, a dry, brown tongue, and a quick, feeble pulse. He took food freely, though he was sometimes nauseated, and was benefited by a moderate amount of stimulant. For this form of delirium opium in small doses frequently repeated is the best treatment in addition to judicious feeding and stimulation. Another form of delirium, of which we have recently had two examples in the wards, is what may be called "senile delirium," for it is seen only in old people, who are subjected to some sudden shock or injury. It is important to recognize this delirium, for it sounds a serious note of warning to which we should at once take heed. The two patients of whom I spoke as exhibiting this form of delirium were both old men, one with fracture of the neck of the thigh-bone, the other with an injury to the ankle and tarsal bones, with severe general bruising. Both of them were healthy-looking old men, and when admitted seemed to suffer comparatively little from their injuries. But after being in bed a few days they both began to babble in a feeble manner. If asked a question they would answer in a fairly coherent manner, but at once relapsed into a senseless chatter. In this condition there is no violence, and though the patient may try to do something undesirable, such as leaving his bed, he is at once and easily controlled. The chief characteristic of this delirium is a subdued but constant talkativeness, generally in a low voice, the words being often imperfectly uttered and even unrecognizable. Sleep is disturbed, and lasts only for short periods. The bowels are confined, and the appetite is impaired.

Now, when an old person exhibits these symptoms under such circumstances as I have described, he must be taken out of his bed at almost any risk, or he will die. Even to place him in a chair and wheel him about his room is a great aid to recovery; but if the nature of the injury permits it, it is still better to let him walk about for short periods at a time. In the two cases in the hospital the injuries were in both of them of the lower extremity; nevertheless, a leather hip-splint for the fractured femur, and a silicate of potash bandage applied to the damaged foot, enabled them to leave their beds and to be wheeled about the ward, with the result that the delirium ceased, and they made good recoveries.

I recently saw, in consultation, an old gentleman who had been so unfortunate as to rupture simultaneously the quadriceps extensor in both thighs, a condition which would almost seem to necessitate confinement to the bed or couch. But at the end of a week of such confinement the patient began to babble nonsense and to sleep badly. I therefore urged that he should be enabled to get up at any risk, and by having a leather splint moulded to each limb this was rendered possible, and he was wheeled about his room, and after a few days about his garden. The delirium ceased at once, and he made a good recovery.

I might relate other cases to the same purpose, but what I wish to impress upon you is that in this condition old people who were becoming rapidly worse while lying in bed, and whom reasonable doses of opium failed to quiet, were immediately benefited by leaving their bed and moving about, and that almost directly the delirium ceased and they slept well under the influence of small doses of the same drug. Let me remind you, in passing, that old people are very apt to be constipated, especially if kept in bed or unable to take exercise; that constipation materially impairs their nutrition and favors the occurrence of senile delirium; and that it is often overlooked because the patient is said to have frequent movements of the bowels, which may only be the expulsion of a little mucus tinged with fæces, while the rectum may be blocked with a hard mass. It is often worth while in such cases to inspect the evacuation, or to pass the finger into the rectum to ascertain the amount and character of its contents.

Since the introduction of antiseptics we have seen less than formerly of traumatic fever and its sometimes accompanying delirium, but the diagnosis of traumatic delirium is not usually difficult. Perhaps the injury in which this form of delirium is most frequently met with is a severe burn. But here we have the high temperature, the dry skin,

and the character of the delirium, which is active in proportion to the elevation of the temperature, to distinguish it from the other forms of delirium of which I have been speaking. Traumatic delirium is best treated by frequent doses of quinine and opium.

Sometimes the case is complicated by the fact that we have to deal with two causes of delirium acting in the same patient. For example, I saw a short time since a young gentleman who had been under my care for syphilis, and who had suffered from great pain in the head and sleeplessness, and who had cranial nodes. He came a long journey to London, and the day after arriving at his hotel he became actively delirious. I was called to him in this condition, and expected to find that the syphilitic disease of the skull had spread to the coverings of his brain. But I found him sweating profusely, and with a moist, coated, tremulous tongue, a soft weak pulse, and natural temperature. It was found, on inquiry, that he had been drinking heavily; and in fact he had delirium tremens, for which he was treated with satisfactory result.

I will mention only one other kind of delirium of traumatic origin, —namely, that which sometimes sets in after a head-injury which has produced laceration of the brain or its membranes. This is an accompaniment of inflammation of which we have the other signs, —nausea or vomiting, rigors, pain in the head, high temperature, quick pulse, dry skin, contracted pupils, intolerance of light and noise. Here the treatment must be directed against the inflammation of which the delirium is only a symptom. But when, in such a case, delirium is a prominent symptom, a free bleeding is often of the greatest benefit. Of course you would also use free purgation, cold, or other appropriate treatment.

I will close this lecture by reminding you that occasionally a surgical operation is followed by an outbreak of acute mania.

NEURITIS.

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY JAMES W. PUTNAM, M.D.,

Clinical Professor of Nervous Diseases, University of Buffalo, New York.

CASE I.—The history of the first patient presented to you to-day is as follows: "Male, laborer, thirty-eight years old. Family history negative. Has always had good health until present trouble began. Last September, being out of work, he tramped over a considerable portion of the country, sleeping sometimes on the ground, occasionally under shelter. One night, after a hard day's tramping, he threw himself upon the ground in damp clothing. He awoke the next morning feeling cold and benumbed, and, as he expressed it, stiff in his legs and arms. He walked that day with difficulty. The stiffness and loss of power increased gradually until, at the end of a few days, he found himself unable to stand." He was brought to the hospital a month ago, when he was examined and the following condition found. There was loss of power in the anterior group of muscles of the leg, loss of patellar reflexes, diminution of sensibility. The muscles of the calf and thigh were normal. Examination of the upper extremities showed paralysis of the forearm muscles, both anterior and posterior, with a tendency to contracture of the flexors. Sensation was diminished. A diagnosis was made of multiple neuritis, due to cold and exposure. At no time does he remember having had fever. There was atrophy of the muscles of the legs, feet, and hands, but not of the forearm.

The differential diagnosis should be made in this case between multiple neuritis and progressive muscular atrophy. In the latter we expect to find a slow progression, the atrophy usually beginning in the hands, the loss of power coming on subsequent to the atrophy. The history of this case, as we get it imperfectly from the patient, is that he lost power first, and that the muscles did not waste until some time afterwards. In progressive muscular atrophy the reflexes are not lost until very late in the disease, whereas in neuritis they are lost in the early stages. Some of you may suppose that this is a case of rheumatism, especially as neuritis due to exposure is often spoken of as rheu-

matic neuritis. The differential diagnosis here is easy, in that there has been no swelling or pain of the joints, no real stiffness or soreness of the muscles themselves, and the tenderness, which is slight, is along the affected nerve-trunks.

An examination of the nails of the hand should always be made in such cases. In this patient you will observe that instead of being flexible the nails are brittle and reedy, which is an evidence that their nutrition is interfered with and that trophic changes have taken place. We next examine the skin. In place of the rough, reddish, hairy hand of a working-man, you will notice that the skin has a peculiar, smooth, glossy appearance, especially along the fingers. This is technically known as Glossy Skin, and is quite characteristic of the trophic changes which occur in this tissue in the later stages of neuritis.

An electrical examination shows loss of faradic irritability to the strong current in both arms and legs. The galvanic current shows contraction to the anodal closure to be stronger than to the cathodal closure. This is known as the Degeneration Reaction, since normally the response of the muscle to the cathodal closure should be the stronger. The degeneration reaction suggests that the nerve-fibres are degenerated, but, as the cathodal contracture is present, we are enabled to say that the degeneration is not complete.

CASE II.—The second patient is a female, aged twenty-seven, who was brought into the hospital two weeks ago with the following history. On Monday morning she commenced a hard day's washing, having her hands alternately in hot and in cold water, and using her hands at times with forcible contraction in wringing out clothes. The next morning she was unable to raise the left arm. There was considerable pain about the shoulder-joint, the pain radiating down the arm. Examination at the hospital three days later demonstrated complete loss of power of all the muscles of the arm and forearm, great tenderness along the trunks of all the nerves coming from the brachial plexus, and consequently the diagnosis was made of polyneuritis.

This case is a peculiar one, and it illustrates the fact that the same cause does not operate equally upon similarly exposed parts of the body, for we learn that the patient used both arms equally, working as hard with the right as with the left, that the patient had not had a previous injury of the left arm, and that it was apparently accidental that one arm was affected instead of both. In this patient, although the disease is only of short duration, we notice that the nails of the left hand are more brittle than those of the right. The skin-changes are not so marked.

The treatment of these cases has differed, because we received the one in the acute stage, the other in the chronic. The indications for the male patient point clearly to an attempt to restore the lost muscular power and to cause the resorption of the products of inflammation. This is best accomplished by systematic massage of the affected extremities by the interrupted galvanic current in order to cause muscular contractions. The joints should be moved daily, in order to prevent false ankylosis. The necessity for this is at once apparent to you when you observe the difficulty which I have in extending and flexing the wrist. This should have been done long ago, but owing to lack of medical attendance it was neglected, and we have, as a result, limited loss of function in that joint as well as in the fingers. Motion of the joints is too often neglected, the physician directing his attention to the affected muscles and nerves, forgetting that the stiffening process is continually going on.

As an illustration of how carelessly such cases are treated, I will relate the history of a case of syphilitic hemiplegia with gradual recovery of the muscles and partial ankylosis of the shoulder-joint. A brakeman recently came to my office with power in all the muscles of the arm, but unable to move the arm away from his side. It had been supposed by his physician that this limitation of motion was due to paralysis of the deltoid and other muscles moving the shoulder. The mistake was easily demonstrated by an attempt at passive motion. It took weeks of persistent effort, which consisted of massage and manipulation, to restore this cured arm to usefulness.

The internal treatment should consist in the administration of potassium iodide and iron. We will substitute the faradic current for the galvanic as soon as we discover the return of faradic irritability, which we will look for once a week.

The treatment of the second case should be directed on entirely different principles, because the conditions are very different. Here we have acutely inflamed tissues, and, following the law laid down by Hilton, we will prescribe rest. The arm should be placed in the position which the girl finds gives the greatest relief; it should be wrapped in hot wet bandages. We will give her, internally, sufficient morphine to quiet the pain. She will be placed on light diet and the following prescription:

℞ Sodii bicarb., 25;
Potassii nitratis, 25;
Potassii et sodii tart., 50.
Sig.—One teaspoonful dissolved in water, three times daily.

We will also give her a wineglassful of Hunyadi water before breakfast if there is still any tendency to constipation. This treatment will be continued till the acute symptoms subside; then we will give her the mild galvanic current, not exceeding three milliampères, along the course of the affected nerves. We will choose for our negative electrode the large six- by four-inch copper plate covered with spongio-pyline. The positive electrode will be of the size of a half-dollar, covered with the same material, and will be applied over the affected nerves. As the symptoms of pain disappear we will increase the strength of the current. We will commence using massage with very gentle friction within a week, gradually increasing its force as the acute symptoms subside. The subsequent treatment will be the continuance of massage and manipulation of the joints.

The prognosis in the first case is unfavorable, as far as complete recovery is concerned, owing to the duration of the disease, although we may hope for partial restoration of some of the muscles. In the second patient we look for complete recovery, as the case is taken in time, and our experience has taught us that the majority of properly-treated cases of acute neuritis are restored to health.

Now, a word with regard to the pathology of neuritis. As its name indicates, it is an inflammation of a nerve-trunk. The causes of this disease are, first, traumatic, which may include the direct wounding of a nerve by a sharp instrument, and pressure upon a nerve, as from a crutch or a blow, or even the compression of a nerve-trunk by a strong muscular contraction. Nerves affected by this cause may be those of the brachial plexus, the musculo-spiral especially, and the great sciatic. I have one interesting case of neuritis of the nerves in the palm of the hand, which was caused by firmly grasping a small rope in attempting to raise a weight. A second cause of neuritis may be an inflammation of the nerve-trunk from adjacent inflammation, as from an ulcer or a bed-sore. Intercostal neuritis has been observed following pleurisy. As already illustrated by the first case shown you this morning, cold is another cause of neuritis. According to some authorities, gouty persons and those who suffer from muscular rheumatism are more apt to have neuritis from this cause than those who are affected with acute articular rheumatism. Neuritis occurs also from many general diseases, chief among which are syphilis and cancer. It also occurs after small-pox, typhoid fever, scarlet fever, measles, diphtheria, and in constitutional states such as that induced by alcoholism.

Neuritis due to traumatism, either direct or indirect, is apt to be limited to one extremity, and even to one nerve-trunk of that ex-

tremity. Neuritis due to alcoholism and to constitutional diseases is apt to be multiple. As an illustration, diphtheritic paralysis usually affects many different and widely-separated nerve-trunks. Alcoholism produces neuritis of the nerves supplying the extensor muscles of the legs, and, less frequently, the extensors of the forearm.

The differential diagnosis between neuralgia and neuritis should also be made. The severe neuritis causes pain which is intense, and which is greatest at the seat of inflammation. This is so because the sheath of the nerve is greatly inflamed, and the nerves to the sheath, or the nervi nervorum, are first affected. In a very short time the other nerve-fibres become affected, and then we have pain in the entire area of distribution of the nerve. Neuritis of mixed nerves interferes with the afferent and efferent conducting functions, causing first persistent hyperæsthesia, then plaques of anæsthesia, the muscles at the same time becoming weak and soft, atrophying rapidly, and then showing a loss of electrical irritability. In slight cases the sheath is mainly involved, and the conduction is not interfered with. In this case the differentiation from neuralgia requires close observation of the character of the pain. In neuritis the pains are continuous, and if the nerve is superficial it can be felt to be swollen. In neuralgia we sometimes have remissions of pain and there is no fever, whereas in neuritis we usually have, at any rate local, elevation of temperature.

In the treatment of neuritis we should be governed by the principles of relieving pain and fever in the acute stage, and if we suspect syphilis we should use potassium iodide. If we suspect rheumatism, salicylate of sodium or salol should be used. In the chronic cases we should direct our attention to repairing nutrition. In old cases which have been neglected and in which we find contractures, it is rare that you will ever have to do more than I have indicated in the way of massage and manipulation of joints, though you may come across instances in which nothing short of tenotomy will rectify the errors of those who preceded you in the management of the cases.

PARALYSIS AGITANS.

CLINICAL LECTURE DELIVERED AT ST. LUKE'S HOSPITAL, CHICAGO.

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THIS is a case of not very well pronounced paralysis agitans, or the shaking palsy of old age. The patient is seventy-five years old. If you will watch the hand you will see every now and then that there is a paralytic trembling of the fingers. It began about ten years ago. He was then injured by a falling building. He was perfectly well up to that time, but the accident produced paralysis of the fingers and hands, and also fracture involving the ankle-joint. The patient is a printer. About six months ago the trembling began to increase, and kept increasing until he could not pursue his vocation any longer. It never gets so bad that he cannot hold his hand still for a short time if he wills to do so. It was quite marked to-day when I first came into the ward. He has used tobacco for the last sixty years; has taken no whiskey for the last seven years; has been in the habit of drinking a little beer after work; never was intoxicated from beer, except perhaps in his young days he says he might have been. The disease is now in its first stage; when it develops into a well-marked case the tremulousness will increase. It generally begins in one finger,—the index or forefinger,—and comes on, apparently, without any immediate cause. It is one of the indications and consequences of old age. It is frequently accompanied by well-marked cramps in the muscle affected; then there commences a tremulousness of some or all the fingers; then it will go on and affect the whole arm; then the muscles of the face, right or left side; then it almost always affects the leg before it crosses over; but at last, in old people, it comes to affect the muscles of the whole body.

One of its characteristics is its rhythmical action; so many muscular contractions in a given time. The implicated muscles contract and

relax with wearisome, indeed exhausting, regularity, and cases have been known where death occurred as a consequence of this unceasing rhythmical pulsation of the muscles. It is tiresome to watch a patient so afflicted; hour after hour, for days or even weeks, sometimes even during the hours of sleep, this regular jerking of the muscles goes on, exhausting the patient and wearying those who are compelled to be witnesses of his affliction. On account of its perfect regularity, or of its perfect rhythm, the peculiar muscular contraction of shaking palsy cannot be imitated. It shows most curiously in the writing of the patient, and I once saw and helped expose an attempt to counterfeit the signature of a magistrate afflicted with paralysis agitans. It was a case involving the validity of a marriage, and the magistrate who, it was claimed, performed the ceremony and signed the certificate, was dead. The "expert" question involved the genuineness of the magistrate's signature. It was a question very easily decided. The attempt to imitate the symmetrically tremulous handwriting of the dead magistrate was very easily seen when the genuine signature was compared with the counterfeit.

With reference to the pathology of this disease, I have only to say that there is no well-marked characteristic lesion. In different cases a variety of lesions have been observed, but they are uniform in only one particular,—namely, that they always partake of some form of degeneration. While the fatty degeneration is the most common, calcareous deposits around or upon the blood-vessels of the brain have been found, and pigmentary degeneration of the brain-substance has been noted, and in some cases all these changes occur. These changes, you will observe, are not characteristic of any one disease: they simply show that nutrition is gradually being undermined by advancing age or other causes. In other words, paralysis agitans is nothing more nor less than the symptom which indicates a gradual interference with the processes of nutrition.

As to the treatment of this disease, of course you do not need to be told that it is incurable; but I think our authors generally say too little about the treatment, because a great deal can be done to ameliorate a patient's condition. In the first place, a patient is generally anæmic, and this indicates some one of the chalybeate tonics, and they are generally more useful if combined with arsenic and quinine. The well-known elixir of iron, quinine, and arsenic answers a very good purpose.

Of course it should be continued for a long time in such doses as are well borne by the patient. Now and then you will find a patient

to whom arsenic seems to be a poison,—an active poison in the smallest doses. In such cases of course the arsenic must be omitted, but I always regard it as unfortunate, because arsenic is so generally useful in neurotic conditions.

Another matter which always needs attention is the obstinate constipation which is almost invariably present. It arises partly from inertia of the muscular layer of the bowels, and partly from the mental dulness which is conspicuously present. It is best remedied by some combination of strychnine and aloïn.

The aloïn, belladonna, and strychnine pill, now so much in use, answers the purpose very well. Frequently the patient is troubled with insomnia, and we should always make careful inquiries about this.

If the patient does not sleep, a dose of chloral or sulphonal, or some one of the recently-discovered hypnotics, should be given without hesitation. Do not, however, fall into the error of prescribing opium or any of its preparations. The diet should have careful attention. I think it is quite too commonly the fact that the aged are not properly fed. The idea prevails generally that a working-man's diet is suitable for an old person, with poor teeth or no teeth and feeble gastric digestion, and so it not infrequently happens that an aged person slowly starves in the midst of plenty.

Aged people, and especially those with paralysis agitans, require a nutritious diet which is easily masticated and easily assimilated. Let me urge you, therefore, in conclusion, to give some attention to the treatment of paralysis agitans. Do not pass it by as unworthy your attention because it is theoretically incurable. If the patient cannot be cured, his condition should be ameliorated as far as possible.

TWO TYPES OF DISEASE OF THE SPINAL CORD IN ADULTS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY LANDON CARTER GRAY, M.D.,

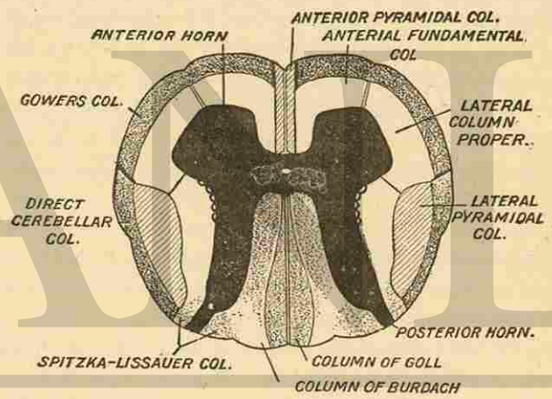
Professor of Nervous and Mental Disease in the New York Polyclinic.

GENTLEMEN,—I have several cases here to illustrate two of the commoner forms of disease of the spinal cord, but before going into the details of them I want to say a few words to you about what we know nowadays of the pathological anatomy of this organ. I presume that most of you are thoroughly acquainted with the elementary matters, and upon them therefore I shall not dwell.

You know that the gray matter of the cord is in the form of horns, so-called *cornua*, anterior and posterior, and that these horns are almost surrounded by gray matter; that the anterior of these is more club-shaped and does not come up entirely to the periphery, whilst the posterior one tapers to a point which reaches the periphery. Our knowledge of the different strands of the white matter has been derived partly from embryological observations, partly from pathological ones, and very little indeed from physiological experimentation. The different strands of the spinal cord in the embryo take on their covering of myelin at various periods of intra-uterine life, so that it is very easy with the naked eye, or at any rate with the microscope, to distinguish those strands which have the myelinic covering from those which have not, because of the great difference in the refraction. In this way Flechsig was able to map out a number of columns, and he published his results some eighteen years ago. I will map them out for you on this blackboard. Dividing the old posterior column into two distinct strands, the one nearest the posterior median fissure is known as the column of Goll, while that which adjoins the posterior gray horn is the column of Burdach. At the side and on each side of the anterior median fissure are found the anterior and lateral pyramids, or, as they are sometimes called, the anterior and lateral pyramidal

columns. On the extreme edge of the posterior lateral column is marked out a little narrow rim of fibres which Flechsig alleged to be in direct connection with the cerebellum, and to which he gave the name of the direct cerebellar column. What was left over of the lateral tract he called the anterior fundamental column. Some of these columns which he outlined had been outlined before, with the exception of the anterior fundamental column, which he never claimed to be one distinct strand, but rather held to be composed of a number of separate strands. Every one of these observations of Flechsig's has been confirmed by pathologists and embryologists in the seventeen or eighteen years that have since elapsed. But we have gone much further than Flechsig went. Part of the column of Burdach has been separated into a column abutting immediately upon the posterior root

FIG. 1.



and described simultaneously by Spitzka of this city and Lissauer of Berlin, to which I have given the name of the Spitzka-Lissauer column. Then it has been demonstrated that there is a distinct column running around from the anterior edge of the posterior cornu to the anterior median fissure, embracing the direct cerebellar column, but elsewhere hugging the periphery, its boundaries being somewhat uncertain, and this has been shown to take on a covering of myelin at a distinct period of fœtal life, and to degenerate separately. It has been named after the gentleman who first called attention to it, Gowers, of London. I have no doubt that this seems to you to be quite a maze, but it is all in reality very distinct and very simple. I will draw a continuous line around all the columns which are sensory,—*i.e.* the columns of Goll,

Burdach, Spitzka-Lissauer, and Gowers. Then I will draw a dotted line around the columns which are motor,—*i.e.*, the lateral and anterior pyramidal, and probably the fundamental lateral column of Flechsig. We know the function of some of these columns, while of others we cannot say as much, and yet we can make a pretty accurate diagnosis and localization of spinal disease; indeed, when you consider that the spinal cord is so very small, not much larger than the piece of chalk which I hold in my hand, it should be a matter of surprise and congratulation that medical science is so exact as to tell us that a disease is located in the centre, or in the periphery, or in some part of the individual columns, as we can do in a large proportion of cases, and as I propose to show you clinically to some extent to-day.

Locomotor ataxia is a disease starting in the central portion of the cord as a subacute inflammation in the connective tissue, and thence extending by means of secondary degeneration through all or nearly all the sensory columns,—*i.e.*, the columns of Goll, Burdach, Spitzka-Lissauer, and Gowers, and the direct cerebellar. We do not know the functions of these sensory columns sufficiently well to say what individual symptoms are due to implication of this column or that, but we can tell you certainly that locomotor ataxia means, in pathological terms, a subacute myelitis starting in the central portion of the cord and thence spreading by secondary degeneration through the sensory strands. This is a man forty-nine years of age. He had a chancre twenty-three years ago. At the outset you should take notice that he had a syphilitic history, because most of these forms of subacute myelitis with secondary degeneration of the sensory tracts, to which we give the name of locomotor ataxiæ, are syphilitic sequelæ. They do not especially belong to the tertiary state, or to the secondary, or to any stage, because they can follow the syphilitic infection at any time. They are not syphilis, and are not necessarily to be cured as syphilis, but they are in the vast majority of cases true syphilitic sequelæ; and therein lies the importance of the fact that this man had a chancre twenty-three years ago. Five years ago he noticed a numbness in the feet. Two years ago he noticed a girdle or cincture feeling extending two-thirds around his body at the level of the mid-dorsal vertebræ. He has had headaches, insomnia, and boring pains in the legs. The latter came on one year ago, and the sensation was, as he says, as if something were boring into him. He has had incontinence of urine for the last two and a half years. His bowels have been much constipated. The facts that are relevant to the diagnosis are that he has had boring pains in the legs, and obstinate insomnia, and that the

headaches come on generally in the morning and leave him toward the latter part of the day, having a periodicity which is almost as regular as that of malaria. This peculiar headache with a quasi-periodicity, and accompanied by insomnia, is very significant, because every headache, contrary to what you might think if you had had no clinical experience, is not accompanied by insomnia. The headache of Bright's disease is not so attended. The headache of meningitis not only does not produce insomnia, but causes somnolence, and though the individual suffering from either of these two kinds of headache may complain acutely during the daytime, he will generally sleep well at night. So that the fact that the man has a headache which is accompanied by insomnia separates this headache from other forms of headache, and when it is also quasi-periodical let me assure you that you are perfectly safe in assuming in the vast majority of cases that it is due to intracranial syphilis. This headache, therefore, means this: that, in addition to the syphilitic sequelæ affecting the spinal cord and giving rise to the symptoms of locomotor ataxia, he has an intracranial syphilis,—*i.e.*, he has both intracranial and spinal syphilis, which makes a great difference in the therapeutics and in the prognosis, because it is possible for you to relieve an intracranial syphilis, and in relieving this you may possibly check the syphilitic extension in the cord. Bear in mind, however, this possibility is not a certainty, for the damage done to the cord may be irreparable, although you may be able to check the cause of it. In addition to these symptoms he has an ataxia which I want you to study. Let us draw this chalk-line upon the floor and see him attempt to walk along it, putting one foot in front of the other. You see that he fails to do so, that he totters, that his feet make irregular and spasmodic movements, that he cannot bring a foot to the point that he aims at. You might think that this disorder of gait was due to the fact that he had some muscular weakness. When I put his leg out straight, however, and direct him to keep it straight, you see that I am unable to overcome the muscular resistance which he offers in that position, for his leg is like a bar of iron, and if I were to attempt to use my full strength I might actually fracture his patella from muscular action. Now I will give him the aid of the sense of touch, or tact, as it is technically called. I will support his little finger upon my little finger, not lending him enough support to be of any muscular aid to him, but simply putting in contact the surface of his finger with the surface of mine. You see that he now walks better, although he does not lean upon my finger at all. He has got the aid of the sense of touch, and through that he is

getting the benefit of my steady muscles. Let us watch him walk with his eyes shut. You see how immediately his gait becomes impaired, the weak movements of the legs become more marked, and he is actually in danger of falling. Let us observe his walk again when he has his eyes open. You perceive that he is unable to co-ordinate his legs, notwithstanding the strength of the individual muscles which I have shown you. This inability to co-ordinate unless with the aid of something else than the motor and sensory apparatus of the legs, constitutes the characteristic ataxia of that disease which we know especially as locomotor ataxia, and is distinguished from other ataxias or kinds of incoördination arising from many different causes. Ataxia may result from anything that interferes with the proper co-ordination or harmonious action of the muscles. If the muscles themselves are impaired, if the different senses which are necessary to the muscular act are impaired, if the joints which are necessary to the movement of the segments of a limb are impaired, if the spinal cord is diseased in other portions than the site which I have indicated in locomotor ataxia, if there is a lesion of the higher brain-centres, we may have what is in one sense an ataxia, or a disorder or incoördination of movement, but the incoördination of locomotor ataxia which you see in this man is as beautifully shown as I have ever seen it in any case in my life. This characteristic ataxia is one of the most valuable of diagnostic factors. By means of it I will make a diagnosis of locomotor ataxia, even if every other symptom in the case is eliminated. But when in addition you have the history of syphilis, certain severe pains, and atrophy of the optic nerve, which you will have to take my word for his having, you may be perfectly sure of your diagnosis of locomotor ataxia. If, furthermore, you have the peculiar pupil which I demonstrated to you in this man, the so-called Argyll-Robertson pupil, you add to the evidence. This peculiar pupil, when typical, is contracted, does not respond to light, but does respond in movements of accommodation,—*i.e.*, it is small, does not dilate or contract with the light or removal of light, but does dilate and contract sluggishly when looking successively at near or distant objects. With these symptoms in your possession—the ataxia, certain severe pains, atrophy of the optic nerve, and the Argyll-Robertson pupil—you can put everything else out of the case as immaterial. The incontinence of urine, the impairment of sensation around the body (the so-called girdle or cincture feeling), the aching pain, are symptoms which are common to other diseases of the spinal cord and to certain diseases of the peripheral nerves, but their presence would not help

the diagnosis of locomotor ataxia any more than their absence would vitiate it.

Here is another individual, twenty-two years of age. His history was taken about a year ago. Some six months previous to this he had been struck on the back in the lower lumbar region by a gas-generator weighing several hundred pounds. Two ribs were broken. He was told at the hospital that his back was broken, and he was in the hospital seven weeks. He could not walk or sit up when he came out of the hospital. He tells us that he had a large bed-sore on the back. He used the catheter three months, and his bowels moved only by the constant use of cathartics. He lost all sensation in defecation. His sexual power is almost extinct. The pain sense is gone in the perineal region, as well as the tactile sense. He had the so-called girdle-feeling, as of a band encircling the lower part of his abdomen. It was evident to us when he came here a year ago, as it is now, that he had had a severe fracture of the lumbar vertebræ. We tried electrical treatment and internal medication, but he did not improve. We advised him to have the fractured vertebræ removed, but he was very much averse to having this done. Finally he did have it done by a very competent surgeon outside of this clinic, and he comes here to-day, several months after the operation, somewhat improved. It is probable that this man's trauma implicated the spinal cord in the lower dorsal region, and that the cauda equina was also injured. I want to call your attention to a matter of medico-legal interest in this case. The absurd dictum has been laid down by some of the earlier writers upon surgery that a man receiving a blow that fractures his vertebræ will not have his cord injured, and, conversely, when the blow does not fracture the vertebræ, the cord will be injured. This sounds very well in physics, but it is as arrant nonsense as ever was talked in medicine. This man, for instance, received a blow with a heavy gas-generator sufficient to injure both the vertebræ and the cord, as the symptoms indubitably indicate, for we could see that the vertebræ were distorted; he tells us that a portion of two was removed, and his symptoms could not have arisen except from injury of the cord and the cauda equina. The central portion of the cord, which I mark out here, was the portion that was injured. You will perceive that this central portion of the cord which I have indicated is in the gray matter, and is anterior to that portion of the posterior columns in which I have mapped out the subacute myelitis of locomotor ataxia. I make my diagnosis of this central portion of the cord having been injured by means of the paralysis of the bladder and rectum, the loss of sensation in the penis and peri-

neum, the bed-sore, the motor and sensory paralysis of the lower limbs. Some central point must have been affected that would implicate all these nerves, and the only central point is just that portion of the central gray matter which I have mapped out. We might suppose, of course, that myelitis running around the periphery of the cord and catching all these different nerves on their way out to the anterior and posterior roots would give us the same symptoms; but a peripheral myelitis of this kind could not result from a trauma, because it is not conceivable that a trauma would implicate only the periphery of a small body like the spinal cord, and leave untouched the central portions. This man has therefore had a central myelitis which may have extended transversely across the whole cord. We must now ascertain the result of the surgical operation. The incontinence of urine, he tells us, has not been affected. The bowels are no more under his control than they were. The sexual sense is just as much in abeyance as it ever was. The girdle-feeling, however, is gone. He can now walk better. The sensory impairment is otherwise just the same as before the operation. He has the same area of anæsthesia in the sacral region, and the same absence of sensation in defecation. You see that when I tap upon his knee the knee-jerk, or the so-called tendon reflex, is very much exaggerated. You see that when I test his flexor muscles they are weak, but his extensor muscles are fairly strong. You can also see the weakness of his flexor muscles by his manner of walking with a loose flop of the legs, because there is no steady, strong action of the flexor muscles acting antagonistically to the fairly strong extensor ones. This peculiar walk is often mistaken for the gait of locomotor ataxia, but you can easily make the distinction. This case of traumatic myelitis has therefore been somewhat improved by the surgical operation which was done three months ago. I think he will improve still more, but whether he will entirely recover is a matter of grave doubt, because he waited eighteen months after he had the accident before the operation was performed, and during that time a chronic myelitis was extending in the cord, setting up secondary degeneration and inducing irreparable changes of the connective tissues and of the membranes, and probably also extending along the peripheral nerves, so that the only thing the operation could do was to remove the pressure of these fractured vertebræ, and the removal of that pressure could not re-convert these organically changed structures into healthy ones, which might have been done if he had had the operation performed immediately after the accident.

In these two cases you have before you, gentlemen, two types of the

most common diseases of the spinal cord,—viz., locomotor ataxia and central myelitis. In reality, both of them are central myelitis, but locomotor ataxia was diagnosed clinically long before we knew much about the pathology of the cord, and central myelitis came under observation at a much later period, so that time has sanctioned the use of these terms, which, moreover, it is desirable to retain because of the differences in the clinical symptoms. One is a myelitis of very subacute onset and very chronic extension. The other is a myelitis that is usually, even when idiopathic, of acute origin and rapid extension within a few hours or a few days. The locomotor ataxia is a myelitis that is prone to extend throughout the cord, and even travel up into the upper and large portion of the cord which we know as the *medulla oblongata* or oblong spinal cord, and it is also, you should bear in mind, in the vast majority of cases, one of the syphilitic sequelæ. The central myelitis, on the other hand, is much more prone to remain localized in the portion of the cord in which it starts, and is caused either by trauma or by causes whose exact nature we do not know, but not by syphilis, tuberculosis, or other diathetic affections. Locomotor ataxia is much more relievable than is generally supposed; and in some cases, where the syphilitic infection has been very recent and implicated the whole cerebro-spinal tract, there is reason to believe that relative cures have been effected: at least cases have been put on record in which all the symptoms have disappeared. But the disappearance of all the symptoms, unfortunately, does not mean that the pathological alterations of the cord have been repaired, for in a notable case recorded by Erb an autopsy made fifteen years after the disappearance of the symptoms showed the characteristic spinal lesion. Nevertheless, it is practically of little account to a man to know what the pathological alterations may be in his cord, provided that he is able to do his work in life as well and as comfortably as he ever did; and that this result has been attained in several cases where the syphilitic infection has been recent is undoubted, and I have also seen it in some few cases in which the syphilitic infection had dated back some time.

I regret very much, gentlemen, that the time at my disposal will not permit me to go into the treatment of these cases, or to show you other types of spinal disease.

FACIAL PARALYSIS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL.

BY GRAEME M. HAMMOND, M.D.,

Professor of Mental and Nervous Diseases in the New York Post-Graduate Medical School.

THE three patients whom I present to you to-day illustrate three different forms of paralysis of the muscles of one-half of the face. If we study the course of the tract through which motor impulses travel from the cortex of the brain to the facial muscles, we will ascertain that the conducting fibres, springing from the facial centre at the lower extremity of the anterior central convolution, pass downward through the internal capsule, decussate at the upper border of the pons, and join the facial nucleus in the medulla. From this nucleus the facial nerve arises. It passes through the internal auditory canal with the auditory nerve, then through the aqueduct of Fallopius, and, emerging from the stylo-mastoid foramen just beneath the lobe of the ear, supplies most of the muscles of the face. The knowledge of the course of the nerve after it leaves the facial nucleus is important, as it enables us in many instances to locate the situation of the lesion with a considerable degree of accuracy. It is possible, in the great majority of instances, to differentiate clearly between facial paralysis due to lesions of the facial motor tract in the brain above the facial nucleus, and lesions affecting the nucleus itself or the facial nerve. It is also possible, if the lesion involves the facial nerve or its nucleus, to determine whether the nerve is affected after it leaves the skull or within the bony canal through which it passes, or where it lies on the base of the brain just after its superficial origin from the border of the pons.

CASE I.—The first case is a man, fifty years of age. He has suffered from two attacks of inflammatory rheumatism during the past eight years. On October 26, 1891, he awoke about six o'clock in

CASE II.—Facial paralysis due to a lesion of the facial nerve, showing inability of the patient to close the eye on the paralyzed side.

CASE I.—Facial paralysis from a cerebral lesion above the facial nucleus, showing ability of the patient to close both eyes.

the morning. He was then perfectly well, so far as he knew. He went to sleep again and slept perhaps an hour. This time when he woke up his face was paralyzed on the right side, and his speech was thick and indistinct, but there was no evidence of aphasia.

Comparing him with Case II., the woman who sits by his side, you will observe that the faces of both patients, while in repose, are perceptibly drawn to the left. Notice particularly that when I make a motion as if I intended striking him in the face he winks with both eyes, while a similar demonstration made before the woman's face is followed by winking in the left eye only. When he attempts to close the eyelids voluntarily you will observe that the lids of both eyes close completely and simultaneously; on the contrary, when the woman attempts to accomplish the same act the lids of the left eye close naturally, while those of the right eye do not close at all. With this single exception in the man's case, neither of them can voluntarily move the facial muscles on the right side. You will also note another point of difference. When I make a remark to him which he considers humorous he smiles distinctly with both sides of his face, thereby greatly diminishing the facial asymmetry. When the woman smiles only the left side of the face contracts, the right side remaining perfectly immobile, and thus the facial asymmetry is increased. Let me now call your attention to the difference in the electrical reactions of the muscles in the two cases. In the man's case, even after six months have elapsed since the advent of the paralysis, the muscles react readily to the faradic current and also normally to galvanism, while in the woman's case, though I am using as strong a current of faradism as she can bear, the muscles do not contract at all. You will observe, however, that they do respond to galvanism.

These three conditions in the man's case—the ability to close voluntarily the lids of both eyes simultaneously; the response of the paralyzed muscles to reflex action, such as laughter, when they cannot be made to move by will-power; and the ready reaction which results from faradic stimulation, even after the paralysis has lasted a long time—are characteristic features of facial paralysis due to a lesion in the facial motor tract above the facial nucleus, while in the woman's case the absence of these conditions shows conclusively that her paralysis is due to a lesion affecting the facial nerve.

I will now explain why the symptoms of facial paralysis due to an intracerebral lesion differ so materially from the paralysis associated with a lesion of a facial nerve. A cerebral lesion above the pons very seldom destroys all the motor fibres which supply the opposite side

of the body. Even in very severe cases of hemiplegia some of the muscles of the paralyzed arm and leg either escape altogether or else are very slightly affected. All the fibres going to join the facial nucleus are seldom destroyed by an intracerebral lesion, and it seems, from an anatomical arrangement of the fibres, that those which supply the upper portion of the face are the ones to escape injury. When the lesion is extensive, so that all the fibres of the cerebral facial motor tract are either injured or subjected to pressure by the hemorrhage, the resulting facial paralysis is complete for the time being, but in a few days, as the pressure is diminished by the absorption of the fluid portion of the clot, the paralysis of the orbicularis palpebrarum gradually disappears, and the individual will then regain the power to close the lids of both eyes, although the rest of the muscles on the affected side may be completely paralyzed.

When an individual receives a pleasurable sensation through the medium of his senses his facial muscles are apt to contract into what is termed a smile. This is an emotional involuntary movement. The impulses which govern it reach the facial nucleus through different channels, and may be transmitted even if the cerebral facial centre is cut off from the facial nucleus. But the integrity of the nucleus itself and of the facial nerve must remain unimpaired. Absolute freedom of these organs from disease is essential for the proper performance of any kind of muscular movement, either voluntary, emotional, or reflex. Hence, when facial paralysis is the result of injury of the facial nerve or its nucleus, a smile is represented only by the contraction of the facial muscles on the sound side, while in facial paralysis due to a lesion of the facial motor tract above the facial nucleus both sides of the face may smile, and yet the paralyzed muscles fail utterly to respond to the will.

The muscles contract properly to the electrical currents when the lesion is in the brain above the facial nucleus, because the facial muscles receive their nutrition from the cells in the facial nucleus, which, being uninjured, performs its nutritive functions properly, and hence the electrical reactions are normal. On the other hand, when the facial nerve is affected its power to conduct nutrition to the muscles is seriously interfered with. The muscles therefore degenerate, and, like all similarly degenerated muscles, they soon show the characteristic electrical reactions of degeneration.

Comparing these two cases of facial paralysis, therefore, we observe that Case I., in whom paralysis was caused by a lesion in the facial motor tract above the nucleus, can close the lids of both eyes voluntarily, and simultaneously smiles on both sides of his face when he sees or hears anything that amuses him; and that the paralyzed muscles respond normally to both faradism and galvanism. Case II., on the other hand, in whom the facial paralysis is caused by injury of the facial nerve, cannot close the eyelid on the paralyzed side; when she smiles the muscles on the sound side contract, while those on the opposite side are absolutely motionless. It is also observed that the muscles fail to respond to as strong a current of faradism as the patient can bear, although they will respond to galvanism. These three differential points will always enable you to distinguish facial paralysis due to injury of the facial nerve from the paralysis due to a lesion of the facial motor tract in the brain above the facial nucleus.

Let us now compare Case II. and Case III., both of whom are suffering from facial paralysis due to injury of the facial nerve, and let us see if we cannot locate the positions of the lesions in both cases.

Case II. is forty-two years of age. She has been a widow for fifteen years. She has had four children, all of whom died in infancy from contagious diseases. On January 1, 1891, her face suddenly became paralyzed on the right side, and sensibility on that side of the face was greatly diminished, but not entirely abolished. She observed for about a month previous to her paralysis that she had gradually been getting deaf in the right ear. After the paralysis the deafness increased rapidly, and in about two weeks hearing in the right ear was lost entirely. If you will examine the uvula you will observe it deviates decidedly to the left. I have already shown you, in comparing her with Case I., that the entire side of the face is paralyzed. She is totally unable to close the eyelid; she cannot smile with the paralyzed muscles, and the muscles show typical electrical reactions of degeneration.

Case III. presents exactly the same appearance as Case II. She cannot close the eyelid, cannot smile on the paralyzed side, and the muscles will not respond to the faradic current. She is about fifty years of age. Two months ago she got out of bed at two o'clock in the morning, and for about a quarter of an hour sat with her head out of an open window, watching something going on in the street. She then went to bed, and when she awoke again the right side of her face was completely paralyzed. There is no disturbance of hearing, no anæsthesia, the uvula hangs perfectly straight, and there is no loss of the sense of taste. In fact, there is no symptom whatever except the complete paralysis of the facial muscles on the right side of the face.

In analyzing the symptoms of Case II. so as to locate the lesion, we will begin from the periphery and work inward. The muscular

paralysis might be due to a lesion affecting any part of the facial nerve but not the facial nucleus. It is probable that the fibres which supply the orbicularis oris arise from the hypoglossal nucleus and join the facial nerve after its exit from the medulla. Hence a lesion of the facial nucleus would cause paralysis of all the facial muscles *except the mouth*, which is not the case in the present instance. Hence the facial nucleus must be intact. The paralysis of the right half of the uvula would indicate a lesion of the nerve either in the aqueduct of Fallopius, where the petrosal nerves which supply the palate are given off, or else back of this point. We are warranted in locating the lesion back of this point because loss of hearing has accompanied the paralysis. The auditory nerve accompanies the facial nerve through the auditory canal. Disease of the auditory canal would cause both facial paralysis and deafness. In this case there is no history of any such disease, and, besides, a lesion in the auditory canal would not cause facial anæsthesia, which is, of course, in this instance due to an affection of the fifth nerve. Farther back than the auditory canal on the base of the brain the auditory and facial nerves lie side by side, and but a short distance from them is the root of the fifth nerve. It is in this situation, and in this situation only, that a single lesion could produce complete facial paralysis, facial anæsthesia, deafness, and paralysis of one-half of the uvula. Meningitis and growths of various kinds are the usual lesions which cause this form of facial paralysis.

In Case III., on the other hand, there are no symptoms, as I have just pointed out, except the paralysis of the facial muscles. This is the most common form of facial paralysis we meet with. It is caused by an injury to the facial nerve, usually inflammation, which occurs just within the Fallopian canal, but not far enough back to implicate the petrosal nerves, otherwise there would be paralysis of the palate, as I have just shown. Exposure to cold is the usual cause of the paralysis. It is so in this case. The patient sat for some time with the side of the face exposed to a draught of cold air. Inflammation of the sheath of the nerve followed, the sheath became swollen, and, as the nerve at this point is situated within a canal with firm walls, pressure must necessarily have been exerted inward against the nerve, which was thereby compressed, and consequently incapacitated from transmitting motor impulses.

The prognosis of facial paralysis is influenced by the situation and nature of the lesion and by the severity of the injury and the consequent neural and muscular degeneration. Above the facial nucleus in the cerebral hemispheres the facial motor tract is liable to be damaged,

principally by hemorrhages and tumors. In either case, if the motor path is simply incapacitated by pressure from transmitting motor impulses, the resulting paralysis will disappear if the removal of the pressure can be accomplished. Thus the prognosis is more favorable when the paralysis is due to a syphilitic growth, which it is possible to absorb, or to a small hemorrhage, the fluid part of which soon becomes absorbed.

In ordinary neuritis of the facial nerve the prognosis is excellent. Sometimes the pressure from the inflamed sheath is so great that extensive degeneration of the nerve occurs, and consequent degeneration of the facial muscles follows. In such cases the disease may last for several months, and may in rare instances become permanent. The electrical reactions of the muscles are of great assistance in determining the prognosis when the paralysis is due to nerve-injury. If the muscles respond to faradism, the injury is slight, and will probably be recovered from in a few days. If faradism fails to cause muscular contraction, while galvanism is quite successful, the case is more severe, and will probably last from one to three months. If the muscles will not respond to either current, the prognosis is bad: the disease may last from four to eight months, and in some instances will be permanent.

The treatment likewise varies with the nature and seat of the lesion. Facial paralysis due to a hemorrhage within the hemispheres is not materially affected by treatment. So much of the paralysis as depends upon pressure from the hemorrhage will disappear as the fluid part of the clot becomes absorbed. It is believed by many that the iodide of potassium accelerates the resolution of the clot and hastens the absorption of its fluid part, but there is little or no evidence to substantiate this theory. If there is any likelihood that the lesion is syphilitic, the iodide of potassium and the bichloride of mercury are both indicated. Case II., in which we have diagnosed a lesion at the base of the brain, involving the facial, auditory, and trifacial nerves, is probably suffering from a syphilitic growth. There is no history of a primary lesion, but secondary symptoms are plentiful. She has been taking iodide of potassium in increasing doses, and bichloride of mercury, for the past ten days. Improvement has already begun. If you will observe closely you will see that when she attempts to close the right eye a slight but distinct muscular contraction follows. This was not apparent when she first came under observation.

Electricity applied to the muscles in all forms of facial paralysis is beneficial. Not that it exerts any curative effect upon the lesion, but simply on account of its nutritive influence on the paralyzed mus-

cles. When the nerve is injured, the nutrition of the muscles is interfered with, and they undergo atrophy.

Electricity in the form of faradism, if that current will contract the muscles, or galvanism, if it will not, unquestionably delays this atrophic change, so that when the nerve recovers from its injury the muscles will be in such a good state of preservation that complete recovery soon follows. On the other hand, if electricity is not used the muscles atrophy rapidly, and in severe cases may almost entirely disappear. In such cases it will be months before recovery takes place. The positive electrode should be placed just in front of the lobe of the ear, and the negative electrode applied successively to the paralyzed muscles, care being taken to interrupt the current so as to make the muscles contract with each interruption. Strong currents should not be used, on account of the likelihood of inducing severe vertigo from the action of the electricity upon the brain. As a rule, the current from six or eight cells will be sufficient. Application should be begun as soon after the advent of the paralysis as possible, and should be made certainly daily, and twice a day if possible, and from ten to fifteen minutes at each *séance*. A small blister should be applied behind the lobe of the ear, or the skin in the same location may be lightly cauterized. Counter-irritation is quite serviceable when applied directly over the seat of the inflammation. This may be accomplished by touching the skin behind the lobe of the ear lightly with a cautery, or else by applying a small blister to the same locality.

The paralyzed muscles are further weakened by being continually pulled and stretched by the contraction of the muscles on the sound side of the face. To overcome this I use a simple apparatus consisting of an S-shaped hook, one end of which fits in the angle of the mouth on the paralyzed side. To the other end of the hook an elastic band is attached which can also be fastened to the ear by means of a loop of cord. By regulating the length of the elastic the tension on the muscles is greatly diminished. A very good hook can be made by putting a piece of whalebone into hot water for a few minutes, then bending it to the desired shape, and tying it. When cold and dry it will retain its form. This apparatus should be worn all night, and also during the day when practicable. Medicinal treatment, except when syphilis is the cause, is ineffectual. We are obliged to wait for nature to remedy the injury. But we can render some assistance by applying counter-irritation over the seat of the lesion, keeping up the nutrition of the muscles by electricity, and relieving the strain on the paralyzed muscles by the apparatus just described.

Pediatrics.

THE SURGICAL TREATMENT OF PLEURISY AND EMPYEMA IN CHILDREN.

CLINICAL LECTURE DELIVERED AT THE MEETING OF THE BRITISH MEDICAL ASSOCIATION, BOURNEMOUTH.

BY RICKMAN JOHN GODLEE, M S., F.R.C.S.,

Surgeon to University College Hospital, and to the Hospital for Consumption and Diseases of the Chest, Brompton, London.

PLEURISY.

GENTLEMEN,—The first part of my subject may be passed over in a very few words; because in dealing with the surgical treatment of pleurisy in children it is only necessary to refer to the operation of aspiration, an operation so simple that it is even included in the practice of the most orthodox Fellows of the College of Physicians. And for this reason it happens that a surgeon has, with exceptional opportunities, but little familiarity with it. Still, he is not unfrequently asked to do it, and sometimes to express an opinion as to the advisability of its being done. To this I always answer that I have never seen any harm result from it, and that I consider it not only advisable but indicated:

1st. When the fluid—whether in large or small amount—shows no tendency to become absorbed, and especially if the temperature keeps above the normal; and

2d. When the fluid is in sufficient quantity to interfere with the action of the heart or the respiration. It must be remembered that the extent of the dulness and the displacement of the heart give a very imperfect indication of the amount of fluid in the chest, for, if the lung on the affected side be consolidated, a small amount of fluid may cause more displacement than a large quantity where the lung is healthy enough to be capable of considerable compression; but, fortunately, under these circumstances the withdrawal of a small amount of fluid gives as much relief as the evacuation of a large quantity would if the lung were much collapsed.

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I can suggest no improvement on the usual method of performing the operation. The bottle aspirator, or a siphon arrangement, may be used: personally I prefer the former, perhaps because I have had greater experience with it. The child is to be placed on the back, close to the edge of the bed, and the skin and needles are to be well purified by the use of a carbolic acid lotion, one to twenty. A fine but rather long needle (three or four inches) should be used, and it may be introduced just in front of the posterior fold of the axilla in the sixth or seventh interspace. The intercostal space is felt with the index finger of the left hand, and the needle is introduced with a firm thrust into the cavity of the pleura. It should be passed far enough to enable the operator to manipulate it freely, so that he can push it back to the angle of the ribs if necessary. The bottle should not be completely exhausted, but should be gradually filled by a slow process of pumping, which can be stopped at a moment's notice if pain or coughing be set up. The danger of cardiac failure owing to the sudden withdrawal of a large amount of fluid is thus avoided. It is not necessary to attempt the complete evacuation of the pleura; indeed, unless the lung is capable of complete expansion—a rather unlikely contingency—it is physically impossible. The indications for stopping are, first, the arrest of the flow of fluid; second, the appearance of blood in the fluid; third, persistent pain or coughing.

Sometimes it is impossible to obtain any fluid from a chest which obviously contains plenty, or the flow may suddenly stop. This is usually caused by a piece of lymph becoming sucked against the canula. The difficulty may be overcome by introducing a plunger smaller but longer than the canula, and keeping it in position during the process of exhaustion. This is often of the greatest service also in the aspiration of an abdomen for ascites, the contact of a piece of intestine stopping the flow in the same way as a flake of lymph in the cavity of the pleura. The same thing may be done if the siphon be used instead of the aspirator.

When the operation is completed, a little manipulation of the part will prevent the escape of any fluid through the puncture. A scrap of antiseptic wool is then luted down over the wound with some collodion. After this I prefer to leave things alone, or, at most, to apply mild counter-irritation by means of some tincture of iodine applied just sufficiently often to make the part hot but not sore; and I am not quite convinced of the advisability of even this mild application.

EMPYEMA.

The surgical treatment of empyema in children can only be by means of an operation, except: first, in those rare cases complicating phthisis, when the same rule applies as for adults,—namely, that, unless the breathing or the heart's action is materially interfered with, the case had better be left alone, because, while a cure is very unlikely, it appears to be almost certain that the tubercular process is accelerated if the pleural cavity be opened. Secondly, if the empyema has ruptured into the bronchus, in which case a *certain* length of time must be allowed to see if nature will effect a cure by this means. I say a "certain" time advisedly, because I do not believe it is possible to fix a definite limit in days or weeks. If the expectoration is obviously diminishing and the child is gaining flesh, whilst the temperature remains normal, the surgeon need not be in a hurry to interfere; but it must be remembered that it is very difficult to estimate the amount of expectoration in children, as they are so often in the habit of swallowing it. The result of a free opening into the pleura is, however, now almost invariably successful, and therefore if there be any doubt about the satisfactory progress of the case it is far better not to delay, but to operate at once; for it must not be forgotten that if a large amount of pus is being coughed up, and especially if it be coughed up with difficulty, some of it may easily be drawn into the bronchi of both the affected and the healthy lung and may set up destructive processes in them. Some may maintain that if spontaneous rupture have taken place externally, nature should be allowed the chance of effecting a cure, but with this I do not agree. The surgeon's help is, I think, always desirable, if not actually necessary.

In dealing with an empyema which is not being expectorated, whether it be a localized or a complete one, the question of aspiration versus free incision is the first to be considered. And I would say at once that I do not believe—taking the average of a considerable number of cases—that aspiration, even if successful, saves time. That it is successful in a few cases has been amply proved by experience; though seldom until the process has been repeated two or three times. It must be remembered that this method of cure involves the absorption of a certain amount of pus, and probably the leaving behind of flakes of lymph in the pleural cavity. It is evidently impossible to evacuate the whole of the contents of the pleura by means of the aspirator, unless the lung can expand completely, or unless the thoracic parietes can contract to such an extent as to come into perfect

apposition with it,—a very unlikely condition of things. It is pretty certain that a considerable number of empyemata in children are never diagnosed, and recover spontaneously; but this is no argument in favor of neglecting a process which must leave behind what theoretically, at all events, may be the source of mischief later on. Therefore, taking all these points into consideration, I am strongly in favor of free incision at the earliest possible opportunity. Every day that is allowed to pass after the presence of pus has been ascertained is a day wasted. The lung becomes more compressed and less able to expand, whilst the walls of the thorax are rendered more rigid by the inflammatory processes taking place in them.

The question of the anæsthetic hardly needs discussion, because one is not tempted to give ether to a child. For adults, chloroform is, in my opinion, better, if the respiration be in any way interfered with, and I certainly should not think of giving anything except chloroform to a child with empyema. It is not necessary to push the anæsthesia far, and it is essential to avoid doing so if rupture into the bronchus has occurred, for an escape of matter into the trachea of a patient deeply under the influence of chloroform may put him into very great peril.

In dealing with a general empyema, I believe the most favorable position for the opening is opposite the ninth rib, a little outside the line of the angle of the scapula. I have made the incision here for several years, and experience confirms the opinion that it affords the most satisfactory drainage, both when the patient is lying down and when he is standing up, and that it is not so low as to involve an awkward obliquity of the drainage-tube when the diaphragm becomes drawn out towards the chest-walls. If a lower position be selected behind, this obliquity of the drainage-tube is often a source of much inconvenience. If the so-called seat of election—namely, the sixth or seventh interspace in front of the posterior axillary fold—be chosen, a pocket not unfrequently remains behind, which necessitates a subsequent posterior opening. In a child I almost always take out a portion of rib about two inches long, because it is seldom, if ever, possible to explore the cavity with the finger without doing so, to introduce a drainage-tube of sufficient size to be of any value, or to evacuate those large flakes of lymph which are often abundant, especially in acute cases. If the patient be very ill, and it appears dangerous to turn him, even a little, on to the sound side, the incision at the spot indicated may easily be made by turning him well over on to the affected side, and operating from behind.

It seems almost unnecessary to discuss again the much-debated question of the advisability or not of removing a portion of rib, because I believe most surgeons at the present day adopt this plan. It is to some extent a question of expediency, and no doubt many long series of recoveries after simple incision of an intercostal space might be produced. But it may be fairly claimed that the operation is not rendered materially more severe or more difficult, that it affords better drainage and a better means of diagnosis, and that the objection urged against it that a weak spot may be left in the chest is not a valid one, for the rib is reproduced in from six to eight weeks in a young child. It is only in very putrid cases that I feel shy of removing a portion of rib; and that for two reasons. First, there is the objection —perhaps only a theoretical one—that septic absorption is more likely to follow the flow of pus over a cut surface of bone than over freshly-divided soft parts. Secondly, by removal of the rib the vessels are exposed to the pressure of the drainage-tube, from which they would otherwise have been protected. This risk is, however, obviated by cutting the artery across and tying it before introducing the tube. But that the danger is a real one is shown by the case of a young lady who had a very putrid empyema of some weeks' standing, by which her health had been very much reduced. As the ribs were very close together, I removed a portion of one of them. A few days afterwards free hemorrhage took place into the pleura, as the result of which she ultimately died. I do not know for certain that it was the intercostal artery which gave way, for I had no opportunity of seeing, but it seems the likeliest explanation of the disaster.

I know of no better drainage-tube to employ than the simple flanged one, which any one can make for himself by splitting the end of the tube into four parts, introducing them through a hole cut in a piece of rubber, and stitching them down with silver wire. But I say this without prejudice, as I believe there are other forms which find favor with other surgeons. Its length should be just sufficient to enter well into the cavity of the pleura, and its size about that of the little finger. No hole should be cut in it, or at most one at a point corresponding exactly to the inner surface of the ribs. This tube is not intended to be shortened, but it may be replaced if necessary at a later date by one of equal length but smaller bore, and when the time comes for it to be dispensed with it may be taken out altogether.

The length of time that the tube should be kept in varies very much, and depends upon the amount of discharge. In young children, I have found on the average (taking long and short cases together)

that a week or ten days is sufficient. But it would be absurd to remove the tube if there were much discharge, and especially if it were purulent. It is not always necessary to wait until the cavity is almost or quite obliterated, but if the cavity remains large whilst the discharge from it is very small, I have frequently found the following method of great use, both in children and in adults. The tube is removed, and the next day—or two days after—a gum-elastic railroad catheter is introduced into the wound, and the contents of the pleura are allowed to escape through it. Very likely when it is introduced a slight whiff of air may be heard, and in some cases—not in all—it may be found by holding a lighted match at the end of the tube that this is being sucked into the chest, not expelled from it. If this be the case, it shows that another means of bringing about expansion of the lung is at work,—namely, the absorption of the air of a pneumothorax. If the fluid which escapes be serous, and each time diminishing in quantity, the tapping may be repeated at intervals of three or four days, and stopped when it has become very slight. In this way it has often been found possible to bring cases to a rapid conclusion that have promised to be very tedious. But if the discharge remains purulent notwithstanding the adoption of this plan, it will be found better to continue the use of the drainage-tube.

Case I.—I employed this method first in the year 1887 in a lady, aged about twenty-five, who had suffered from a right empyema for a year, which was discharging through an opening opposite the fifth interspace in front, which led upward and inward obliquely to an opening in the fourth interspace behind the mamma. I made a large opening opposite the ninth rib, outside the scapular line, and the discharge rapidly diminished and became serous. But, owing to the rigidity of the walls of the cavity, closure was taking place very slowly until I adopted this method, which in a few weeks effected a permanent cure.

Case II.—The next case was that of a gentleman, aged sixty, who had an old localized empyema on the left side which had been treated by poulticing from the commencement. The result was a cavity beneath the scapula containing about six ounces of stinking pus and discharging itself through an oblique opening near the posterior fold about opposite the eighth rib. I removed about four inches of this rib, and so obtained free access to the anterior part of the cavity. The effect upon his general health was wonderful. Before the operation he was feeble, pale, blue, with clubbed fingers and weak pulse, so that I was disinclined to operate upon him at his age. Afterwards the circulation

improved, the numbness and clubbing of the fingers disappeared, and the cavity, though it remained of some size, stopped discharging almost completely. There appears to have been great difficulty in keeping the tube in, and I had the opportunity of seeing him at intervals of several days and ascertaining by means of a catheter that the cavity had almost ceased to secrete, although the opening was practically closed. Thus, though I did not methodically adopt the plan I am describing here, it was a great encouragement to continue its use in other cases; which I have frequently done since, and generally with most satisfactory results.

Case III.—One particular case may be mentioned,—that of a young man, aged twenty-four, who had a left empyema with an acute onset, in which after the first evacuation the temperature came down for only a few days, and then assumed a hectic type, whilst the discharge remained very copious and purulent. After a prolonged period of irrigation with a weak solution of iodine and a one-per-cent. emulsion of creolin, the discharge diminished, but remained purulent. The tube, however, caused so much discomfort that it was removed about six weeks after the operation, and the periodical tapping was begun. In a short time the discharge became serous, and in a little more than three weeks we were able to discontinue the process altogether.

Injecting the pleura at the time of the operation is, I think, never advisable. It is a process not free from danger, as it is well known that many cases of sudden death have occurred during its performance, both in old and in recent empyemata. For this no sufficient explanation has been given, though many have been suggested. The only case I ever saw was many years ago in a child with a small old cavity with dense fibrous walls. If during the subsequent treatment it seems advisable to employ injections, care should be taken that no pressure is caused inside the chest, which may be avoided by using a tube much smaller than the opening, and by introducing the fluid through an irrigator held at a height not greater than eighteen inches above the level of the bed. The tube employed may be either an india-rubber drainage-tube or a gum-elastic catheter. The latter has the advantage that it can be directed to any part of the cavity, and, if desired, it may be provided at the end with a rose, which will allow the fluid to escape in a spray instead of in a single jet. I am in the habit of employing either tincture of iodine added to water until a pale sherry color is produced, or a one-per-cent. emulsion of creolin. Sometimes it is useful to blow finely-powdered iodoform into the cavity. The temperature of the solutions employed should be about 100°.

The spray has now practically gone out of use, but it is as essential as ever to prevent the entrance of those coarser fragments of dust into the chest which are now known to be the principal vehicles of the organisms that produce septic changes. It is therefore well, at the time of the operation, to allow the matter to escape, and to perform the subsequent manipulations as far as possible beneath a veil of muslin or lint soaked in some antiseptic solution. The same method may be adopted in changing the dressing. When the deeper part of the dressing is to be removed, the patient is told to hold his breath, the tube is removed, and the veil is placed over the wound. When the tube is to be reintroduced, the same manœuvre is repeated. It is thus easy for any one to keep an empyema perfectly aseptic without the use of the spray. The double cyanide gauze (of mercury and zinc), now employed by Sir Joseph Lister, forms an excellent dressing for these cases. It is far superior to the alembroth gauze, because this double salt is very insoluble. When the alembroth gauze was used, if the discharge was copious, a stronger and stronger solution was produced as it gradually soaked its way towards the edge of the dressing. This not infrequently caused an eczema near the margin of the dressings, which was not only troublesome to the patient, but often a source of putrefaction spreading to the wound. If the pus be already offensive, it will not be necessary to employ so expensive a material. A piece of boric lint soaked in one to two-thousand sublimate lotion beneath a pad of oakum or wood-wool makes a very excellent application, and there are many other suitable dressings, which I need not mention. If the discharge be copious in a very chronic or incurable case, the dried and compressed moss introduced some years ago from Germany will be found very useful. Nothing further need be said with regard to the treatment of a *localized empyema*, except this: that the opening must be, if possible, in the most dependent situation.

If an empyema has already ruptured externally, it is right to consider whether the opening is in a sufficiently favorable situation for good drainage. If it be, it may be utilized after it has been enlarged. If it be not, the surgeon must not hesitate to make a second opening in the most favorable situation. It is bad practice to delay the cure for the sake of avoiding a second scar which will never be seen. If a second opening be made, the first may probably be left without a drainage-tube, but sometimes the presence of the second opening appears to favor the escape of the fluid, perhaps in the same way as the flow of beer from a cask is rendered possible by the removal of the spigot.

I have previously referred to cases which have ruptured through the lung, and hinted that it is unwise to delay making an external opening. In most cases, when this is done, the internal opening rapidly closes, and cure proceeds as if no such opening had ever existed. But occasionally, either because the opening is very large, or from some other cause which has not been explained, the communication with the lung remains patent, and often very free. This is shown by the peculiar whiffing sound when the patient expires, exactly like that which is heard when a pulmonary abscess has been opened, and by the rapidity with which any sapid material, such as iodoform, injected into the wound, is tasted in the mouth. These cases are very unpromising to deal with, and indeed sometimes appear to be absolutely hopeless. The best that can be done is to keep the external opening free for a very long period, and cautiously to shorten the drainage-tube. If the injection of such a cavity be attempted, a most painful fit of coughing may be set up.

It remains only to deal with those cases—happily, I believe, becoming more rare since the early treatment of empyema has been put upon a more satisfactory basis—where the lung has expanded to the utmost possible extent, the chest-walls have fallen in as far as they can, producing sometimes great deformity, and the diaphragm has risen to its highest limit, and yet there remains a cavity bounded by dense fibrous walls. Here it is only possible—and it is right—to perform the operation named after Estlander, its inventor,—namely, to remove large portions of ribs from that part of the chest-wall which bounds the cavity. The rule is to take away the longest portions opposite the middle of the cavity, and smaller ones as the upper and lower limits are reached. Thus portions of as many as seven or eight ribs have sometimes been removed, and occasionally a good result has been obtained. But it is obvious that if the cavity extends to the apex of the thorax it will be impossible to deal with it in this way; and even under apparently favorable circumstances it has not unfrequently happened that, although the cavity has been very much diminished, an incurable sinus has remained. In performing this operation a flap may be reflected corresponding in size to the part of the chest-wall which has to be opened. Or, if it be preferred, the ribs may be removed through incisions parallel to their long axes. It is quite easy to remove portions of three ribs through each of such incisions. The hemorrhage is often rather free whilst the ribs are being exposed, but after this removal no trouble is usually given by the intercostal arteries. The whole of the periosteum and thickened pleura corresponding to the ribs

that have been removed must be cut away, to prevent the reformation of the bone. In doing this all the intercostal arteries of the part are necessarily divided, but often they do not bleed at all, and they seldom give any trouble to speak of. It has often struck me (and I think the suggestion was made by the late Mr. Marshall) that it would be a good plan to supplement this procedure by dividing the ribs from which portions have been removed, both near the spine and near the sternum, so as to allow of their more complete collapse; but I have never yet put this plan into practice.

PLEURISY IN CHILDHOOD.

CLINICAL LECTURE DELIVERED AT THE EVELINA HOSPITAL.

BY GEORGE CARPENTER, M.D. Lond., M.R.C.P.,

Senior Physician to Out-Patients at the Evelina Hospital for Sick Children, London.

GENTLEMEN,—Recently we have seen several cases of pleurisy among the out-patients, and to-day I shall tell you about its peculiarities, causation, diagnosis, course, and treatment. In the first place, a very free formation of fibrin is usual, and, secondly, purulent effusions are frequent. Of 190 cases there were 114 males and 76 females. The fibrinous variety totalled 85,—48 right-sided, 36 left-sided, and 1 double. There were 105 empyemas,—50 right, 53 left, and 2 double. Of the former 7 died, and of the latter 31, and one was suffering from lardaceous disease. Two or three were doubtful. Of these cases 114 were five years of age or under, 75 of them being empyemas. Many commenced with a sudden sharp febrile attack; some few gave a history of a preceding exposure to wet and cold (10 in 101). Occasionally injury in the shape of a fall or other traumatism appears in the history as the starting-point. Many were caused by pneumonia, broncho-pneumonia, bronchitis, and pertussis followed by pneumonia, etc., also scarlatina and measles, rheumatism, tuberculosis of the lungs and pleuræ, typhoid fever, diphtheria, mumps, and varicella. In many cases a rheumatic or a tubercular family history can be obtained. Children with Bright's disease are very liable to pleurisy. In acute nephritis the effusion may be double, without lymph, and therefore more of the nature of dropsy. Some cases are associated with a general tubercular implication of the serous cavities, or septicæmia with a rapidly fatal issue: in the latter colonies of micro-organisms will be found in the affected parts and organs. Pulmonary apoplexies may be complicated by pleuritis. In pyæmia the local condition may mask the general. Pleuritis may exist with or without infarcts, and infarcts without pleurisy. It may occur as an extension from pericarditis, or the converse, or it may even extend to the great vessels.

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In a female of two years, with double empyema, a swollen left

hand and forearm, and œdematous chest-wall, there was a thrombus of the left innominate vein extending to the subclavian and internal jugular veins. She had general tuberculosis. As occasional causations may be enumerated mediastinal new growths in the lungs and chest-walls, extension from neighboring parts, such as axillary abscess, general peritonitis, either tubercular or due to other causes, abscesses of the liver, either between the liver and the diaphragm or between the spleen and the diaphragm, suppurating hydatids in these situations, perityphlitic and perinephritic abscess, cellulitis of the neck, for example, secondary to tracheotomy, suppurating cervical glands due to spinal disease or rib caries, and foreign bodies in the air-passages. Twice I have seen a fibrinous effusion associated with cutaneous syphilitic gummata. One boy of five with a fibrinous effusion developed a white leg. In a boy of three with empyema there was free air in the sac. The right lung was collapsed, the pleural cavity divided in two by dense adhesions with openings. A small abscess-cavity in the lower lobe, filled with caseous material, communicated with a bronchus but not with the pleura. In a boy of fourteen months with empyema the left lung was collapsed. In the lower lobe was an orifice connected with a superficial cavity and indirectly with the bronchi, also a small abscess. On the under surface were three small apertures communicating with a net-work of ragged cavities partially separated by bridges of shreddy tissue, and here and there portions of solid lung-tissue becoming gangrenous.

For some unexplained reason, after exposure to wet and cold, or often following one of the causes previously enumerated, the child is seized with the usual febrile symptoms, accompanied by a temperature of 101°, 102°, 103°, or 104° F., or more. There may be headache, vomiting, lassitude, shivering, drowsiness, or even convulsions. The high temperature is of variable duration,—a day or two, perhaps a week, or even a month. Rarely there is delirium, or rarer still a typhoid state. Pain may be absent or very slight; occasionally it is acute, and the affected side tender. Pain may be felt elsewhere, and only in one particular spot, as at the shoulder, or the sternal, epigastric, umbilical, or hypogastric regions of the opposite side,[1] and in the limbs. In one left sided pleurisy it was down the left arm. Sometimes there are profuse sweating and diarrhœa, but these symptoms belong rather to purulent pleurisy, although they are not confined to this disease. The pulse

[1] Laennec and Gerhardt agree with this. See Fagge's "Principles and Practice of Medicine," 2d ed., vol. ii. p. 171.

and respiration ratio is altered; the respirations are increased in frequency, with laryngeal grunting and sometimes dilatation of the alæ nasi. When effusion is abundant the pulse becomes small, frequent, and irregular. Cough is frequent, short, hacking, and dry. In young infants collapse is a noticeable feature, and wasting is rapid. There may be coldness and blueness of the extremities, as well as of the lips. Orthopnœa may occur, during coughing, and marked cyanosis. The effusion of fluid has then been very rapid and copious.

Often the child is brought to the physician on account of anorexia, wasting, and cough, and is carried into the room or drags his weakened limbs with difficulty. Physical examination alone in such a case can clear up the diagnosis. There may or may not be fever. In many pleurisy is detected only after physical examination. The pleurisy merges into the general disease. Thus, in pneumonia, broncho-pneumonia, and bronchitis a friction-sound may be heard. Disease commencing as pneumonia is rapidly merged into pleurisy with effusion, often of a purulent type. In many instances pleurisy, though present, escapes detection. A friction-sound may be heard in bronchitis revealing a concomitant unexpected pleuritis. Pyæmia may mask pleurisy. Pericarditis with pleural extension may overwhelm pleuritis, and overlying pleuritis in pulmonary apoplexy remain undetected. Attacks of dry pleurisy are occasionally submerged by the accompanying fever. Friction may be present or absent, the diagnosis depending upon the stethoscope. It may possess the usual characters, appear as a moist râle or a dry rhonchus, be heard to-day and gone to-morrow. Its dependence on the depth of respiration, the effect of the position of the child, whether it is reclining or erect, modifying or annulling the physical signs, and the importance of detecting any pericardial friction-sounds, must be remembered. Friction may be heard anywhere over the chest, but the favorite seats are the nipples, the axillary regions, and about the scapular angles. Friction fremitus may sometimes be detected.

Passing from the initial stage, we arrive at that of fluid effusion. This will be found under several types. In one, not uncommon, there are signs of consolidation of the upper or upper and middle lobes, with deficient, very rarely absent, vesicular murmur over the lower lobe. Rarely the breath-sounds may be annulled at the extreme base. Such might pass for apical consolidation, and so it is, usually fibrinous; but occasionally this compression of the lung by bands of lymph causes tuberculosis. Sometimes the breath-sounds are cavernous, but cavernous breathing is often heard over simple solid areas in children's

lungs. When the consolidation is that of croupous pneumonia, a trace of albumin may sometimes be detected in the early stages. The opposite side gives extra-puerile breath-sounds and frequently subtympanitic resonance. Percussion gives fluid dulness and resistance, both important; but, remember, such may be encountered over a solid lung. Because the dulness is not of fluid character, it does not follow that fluid cannot be present. The note may be merely deficient resonance, and occasionally stomach resonance is obtained as far as the angle of the left scapula. Percuss lightly, the chest-walls yielding, and it is easy to displace fluid and reach crepitant lung. In another, the chest may be dull from apex to base, with good but distant vesicular breath-sounds, or distant tubular breathing perhaps heard on a deep breath only, and then only expiratory, or distant vesicular breath-sounds with moist râles. As before, the breath-sounds on the healthy side will be extra-puerile, not bronchial, as sometimes called, and moist râles may be heard. With this extreme effusion there may be apical resonance or hyper-resonance, the breathing clear and distant, expiration prolonged and unduly audible compared with inspiration. Dulness then does not usually extend to the spine. A sector-shaped area of resonance may be obtained over the root of the lung and its immediate neighborhood. In another there is dulness over the lower lobe and deficient entry of air. Sometimes the breath-sounds are distantly tubular. In either case there may be loud tubular breathing at the upper limit of dulness, friction-sounds or not, sometimes friction-sounds alone, sometimes pneumonic crackles only. Skodaic resonance can not infrequently be obtained over the corresponding apex, above the clavicle, below it in both situations, and sometimes behind. Skodaic resonance is also obtained in basic consolidation, sometimes apical. Basic consolidation itself may give all the signs of fluid. Often the method of respiration deceives: now there is vesicular murmur, again with a certain laryngeal intonation the breathing is tubular, often the expiratory sound alone has this quality, and bronchophony is for the first time appreciated. Bronchophony contra-indicates fluid, but when in doubt do not hesitate to explore. Again, râles present yesterday have disappeared to-day; tubular breathing produced at a solid apex and conducted to the opposite root is present at one examination and gone at the next. Another factor in these productions is unequal action of the two sides of the chest. A case may first come under your care as an acute lobar pneumonia, but the customary crisis[1] is

[1] Sometimes extensive rises and falls occur after the crisis for a day or two or more before equilibrium is reached. Rarely the case terminates by lysis.

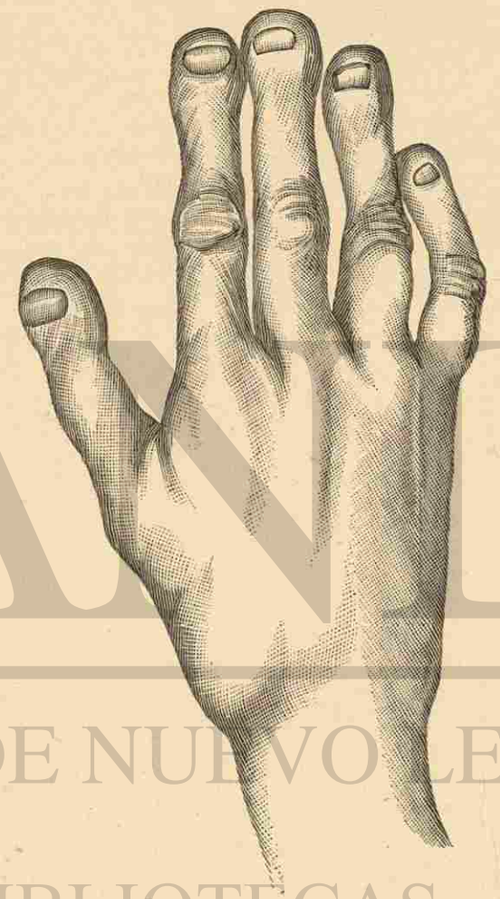
passed by, the temperature remains up, and the signs of consolidation give way—this is the important point—to those of fluid. Sometimes the temperature falls as usual and then again rises, remaining more or less elevated, but accompanied by a change in the physical signs. Broncho-pneumonia or a simple bronchitis may be responsible. With fluid effusion at one base friction may be heard over the other. Accumulations of pus may become localized and confined by adhesions, limit themselves to the apex, the base, the axillary region between adjoining lobes, the lung and the diaphragm or mediastinum or pericardium. When there are two or more cavities they often communicate by a sinus or sinuses, but they may be shut off. In a child aged ten months the space containing pus was like a honey-comb. Thus, it may be necessary to explore beneath the clavicle, or it may be quite beyond reach and the methods of physical diagnosis at our command. Fibrin alone may give the signs of fluid when no fluid is present.

The position of the cardiac impulse is a most important factor in forming a diagnosis. In right effusions the impulse may be in the nipple-line or an inch or more outside this. In left effusions cardiac impulse may be epigastric or in the fourth and fifth or the third and fourth right interspaces. A triangular area of dulness can be mapped out. This impulse is occasioned by the impact of the right ventricle in the interspaces, and of the right and left ventricles in the epigastric region. The heart by some is thought to rotate on its axis, by others to be pushed bodily over to the right. In one autopsy on a child of six the heart was placed vertically in the middle line of the body and the pericardium partially adherent to it. In another, a child of four, more of it lay to the left of the sternum, but chiefly behind it; in another, aged three, the mediastinal contents were shifted to the right without cardiac rotation; in another, right-sided, aged ten months, the heart was a little displaced to the left.

Cardiac bruits are sometimes caused by the kinking of vessels. The liver and spleen also share in the dulness; thus the spleen may be displaced and enlarged or not, the lower edge of the liver reach to the umbilicus with nutmeg alterations, or not, according to the chronicity of the case. The affected side does not usually show much alteration, but it may be more rounded, or even bulging; sometimes it is smaller. Obliteration or even bulging of the interspaces happens occasionally, best seen by looking from above downward. Rarely there is fluctuation. Sometimes there is œdema. Increase of cutaneous thickness may be determined by pinching the skin and comparing it with that of the opposite side.

In a female of four with purulent pleuritis in the upper part of the chest, this was uniformly bulged forward from the clavicle to just below the nipple, and the sterno-clavicular joint was loosened. The overlying skin was œdematous. In another purulent pleuritis in a male of three there was marked localized bulging of the lower ribs of the right side. Bulging of the chest-wall and cutaneous œdema signify purulent collections. Empyemas sometimes point, usually in one place, rarely in more, and mostly to the front of the chest, passing through an interspace with denuding of the periosteum. Abscesses may be pale, or inflamed and painful. In a boy of seven there was a fluctuating swelling over the front of the left fifth, sixth, and seventh ribs. In a girl of six, it formed over the left fourth, fifth, and sixth ribs. In a boy of three, perforation was at the seventh left interspace in the nipple-line, with commencing subcutaneous abscess; and a boy of nine had an elastic fluctuating prominence over the right hypochondriac region. Pus may also find its way into a bronchus, the abdominal cavity or some of its organs, the loins, or the lower extremities. Sometimes these chest tumors have a transmitted cardiac impulse and the heart sounds are plainly audible over them. Such are called pulsating empyemas. Pulsation may be detected in the interspaces near and above the nipple without an external tumor. Puffiness of the face and eyelids, like that of renal disease and pertussis, is seen in some cases. The affected side will be found more or less immobile. Sucking in of the lower interspaces is noticed in some. The quantity of fluid is variable. The most I have seen is forty-six ounces of clear fluid in a child of six, but Heyfelder aspiratcd six pints from a boy of the same age. The smallest quantity I have diagnosed and evacuated is one-half ounce of pus from each pleura in a child of five, with immediate disappearance of physical signs. The average, perhaps, is eight to ten ounces, caught at the time of operation, but matter will drain away for some hours afterwards, to the extent of many ounces. Vocal resonance and vocal fremitus are of very little service. Cry resonance and cry fremitus are useful. Œgophony is of little value. In the early stages the child usually lies on the healthy side, but with effusion in quantity he lies on his back or on the affected side, but there are frequent exceptions. In chronic cases the fingers and toes become clubbed, the chest falls in, the ribs crowd together, and the spine is curved laterally, the concavity pointing to the affected side. The child wastes. The right heart hypertrophies and dilates, and the various alterations in the organs accompanying this condition make their appearance. Owing to this wasting, dropsy is rarely observed. A female aged six had been ill two years;

the diagnosis was phthisis. She had signs of consolidation in the upper half of the left chest. Above the breath-sounds were cavernous, below they were distant vesicular or tubular. The dulness was absolute. Once or twice the other root appeared consolidated, but the



Clubbing of fingers in chronic empyema.

sounds were conducted, otherwise they were extra-puerile and the resonance was subtympanitic. Expectoration was muco-purulent, sometimes little, at other times profuse. She had a contracted chest with lateral curvature. The cardiac impulse was in the fifth and sixth

interspaces, one and one-half inches outside the nipple. Epigastric pulsation.[1] Liver edge two and a half fingers below the costal margin. Her lips were rosy; she was in fair condition, and could walk. Taking into consideration the extreme cardiac displacement, the position of the liver, and the physical signs, I thought there must be fluid. Several times I explored, and on each occasion entered fibrotic lung. Finally I obtained fluid from the axillary region. It was suggested at the time that a dilated bronchial tube containing pus had been tapped; but five ounces of pus were evacuated from her chest.

In the dry stages a friction sound is the only evidence, but pleuritis may be inferred from the symptoms. The several other possible causations of pain under the left nipple must be remembered. Given effusion, is it serous or purulent? The fact that it accompanies an exanthem with perhaps diarrhœa, sweating, a sallow complexion, marked wasting, and an obviously low state of health, are in favor of pus,—*in favor*, mind. Temperature, as a diagnostic sign between the two, is worthless: it may be high with serum, normal with pus. Exploration with a hypodermic syringe fitted with an exploring needle is alone reliable. Upon its passage into the pleural cavity, a fibroid, pneumonic, or crepitant lung will be readily felt. If it passes into the pleural cavity, fluid may not flow on account of fibrinous blocking of the needle. Pus may come from a dilated bronchial tube. In one case autopsy showed a pneumonic lung riddled with abscesses, where puncture almost anywhere would have withdrawn pus. If conducted with proper precautions no danger need be feared. Recently, however, in a case of mine, although the position of the heart was not in favor of fluid, the resident explored for pus and withdrew ten minims. The child coughed immediately, blood poured from its mouth and nostrils, and death occurred from asphyxia. A necropsy disclosed a cirrhosed lung with dilated pus-containing tubes. A large vessel had been punctured. Some few cases are tubercular. Is this tuberculosis? The observer must watch for signs of cerebral implication in the shape of ocular or facial pareses, weakness of a limb, a cerebral pulse, respiration of irregular rhythm or Cheyne-Stokes type, great frequency without sufficient pulmonary cause, paroxysms of cyanosis, optic neuritis, tubercles in the choroid,[2] and convulsions, vomiting not obviously peritonitic, a prolonged and irregular temperature without evidence of imperfect drainage or

[1] Right-sided cardiac hypertrophy also displaces the apex to the left.

[2] Tuberculosis of the Choroid, by George Carpenter. Illustrated Medical News, December 7 and 14, 1889. Plates. British Medical Journal. Plates.

sepsis, and œdema of the hands and feet. A tubercular history and the presence of tubercular lesions elsewhere must receive due but not too weighty consideration. A fluid effusion is sometimes difficult to diagnose. A child of six, a cured empyema, was thought to have typhoid. There was comparative dulness and somewhat deficient breathing, quite compatible with her history. Three times she was most carefully examined. After fifteen days a swelling appeared over the scar, and a quantity of pus escaped. A large pericardial effusion may be mistaken for pleurisy. A carefully mapped-out area of dulness will negative free fluid, but it might still be a localized empyema. A hydatid cyst or an abscess between the liver and diaphragm or in the liver would give the signs of fluid localized to the corresponding axillary region. Short of an operation and digital examination, its origin cannot be decided upon. The same remarks apply to perisplenic abscesses. By careful mapping, a lung invaded by mediastinal new growth will not cause trouble, and when fluid is present in the sac its cause can be determined. Dropsy of the pleura is both-sided, and is not common in heart-disease, congenital or acquired. Double hydrothorax in acute Bright's disease has been mentioned. Apart from these, double pleurisy suggests pneumonia, tuberculosis, pyæmia, or septicæmia. The duration of pleurisy may be brief, a few hours only. Serous effusions usually clear up rapidly. Aspiration sometimes, from admission of air or from instrumental contamination, renders serous effusions purulent. I think that empyema starts as such, but some hold to the contrary. The fluid does alter in character sometimes: thus, I have seen at the first aspiration sero-pus, at the second clear blood-stained fluid, at the third laudable pus. On absorption redux friction may occur. The physical signs from fibrinous deposition occasionally do not altogether disappear for some time. Pus may become absorbed. Thus, pus was withdrawn from the right base. Some days afterwards, when prepared for operation, the physical signs were rather in favor of solid lung. Several dry punctures were made into solid lung. The chest-signs finally cleared. The quantity of pus was probably small. From absorption and non-corresponding lung-expansion are found the chest deformities already described. Rarely the pus is stinking: this may be from communication between the pleural cavity and the bronchial tubes. With a large effusion, sudden death may occur from failure of the respiratory centre, œdema of the opposite lung, or twisting of the inferior vena cava. Persistent consolidation of the upper lobe may remain with even a moderate empyema. Thus, in a child of two, with left empyema,

consolidation of the upper lobe persisted for two months, and then rapidly cleared. In another under two, similar consolidation continued for eight months after the empyema was cured. Mercurial inunctions were tried. Two months later there were signs of resolution, resonance returned, breathing was vesicular, entry of air fair, and a few râles. General condition excellent. I do not doubt that he quite recovered.[1] These cases seem to be pneumonic. Signs of progressive breaking down of the lung, in the shape of gurgling râles, fever, wasting, and obvious going down hill, betoken phthisis. Some die of general tuberculosis, others of exhaustion. In the early stages the autopsy may show suppurative peritonitis, pericarditis, meningitis, and mediastinal implication. Others die of pneumonia of the affected or opposite lung; some of œdema of the lungs. A serous or purulent effusion may be discovered on the opposite side, having passed undiagnosed. A male of three and three-quarter years had a chocolate-colored effusion in the left chest, both lower lobes collapsed and tuberculous, and in addition general tuberculosis.

I have already mentioned localized and multiple empyemas. The collapsed lung lies in the apex of a groove between the spine and the mediastinum; its position is determined by the root. Old adhesions will alter this arrangement. When adherent to the pericardium, the heart may be pulled from the chest-wall. On section airless and of a violet color, losing its contained blood it becomes slate-gray, smooth, and dry. The pleura may be thickened one-sixteenth of an inch or more, the septa dense, the tubes empty or pus-containing, sometimes dilated. First there is simple compression, later inflammatory cell-growth, chiefly in the septa, one method of production of the cirrhotic lung.

Simple uncomplicated effusions tend to rapid cure. In double hydrothorax with acute Bright's disease the outlook is bad, as also in empyema secondary to renal mischief. Double hydrothorax in heart-disease betokens a failing heart. When tubercle starts the process a fatal termination may be expected. Pneumonia or broncho-pneumonia as the starting-point adds to the risk, and the onset of pneumonia is of grave omen. Of the seven deaths mentioned, there were one from mediastinal new growth, one from pyæmia (bone), one from pneumonia, three from tuberculosis, and one from exhaustion after several aspirations.

[1] Recently this child attended my out-patient dispensary. The chest was quite healthy, and, with the exception of scarring, no difference could be detected from the opposite side.

Uncomplicated empyema cases do well if taken early. In cirrhotic cases the prognosis is that of the accompanying heart-disease. Prolonged suppuration any time over four months is apt to produce lardaceous viscera. Of thirty-one deaths from empyema there were five cases of pneumonia on the same or opposite side, two had peritonitis in addition, and one tubercular meningitis, four succumbed to tuberculosis in one form or another, one to scarlatinal nephritis and exhaustion, one to interstitial nephritis, two to exhaustion, one with cheesy tracheal glands, two died suddenly, two died out of the hospital, one was undiagnosed (coma and convulsions), two had lardaceous disease, one had pus in the abdomen, one pyæmia, in four post-mortem was declined, one died of (?)peritonitis, one with fetid pus, one on operating-table, one while washing out the cavity (convulsions, high temperature), one from abscess and gangrene of the lung.

For the relief of pain and cough nothing is better than small, frequent doses of opium in the shape of pulv. ipecac. comp. In a robust child, three or four leeches may be applied over the seat of pain. Dry-cupping is also useful. Cold may be applied by an ice-bag, but this in young children not infrequently induces collapse, and therefore, if used, it should be carefully watched. Warmth may be employed by heating cotton-wool or Gamjee tissue, the painful spot being painted with glycerin and belladonna. Frequent changing being necessary for the application of these measures, they are not recommended, and the same objections hold for cold compresses. Hot fomentations, with or without opium, changed hourly, are beneficial, but they induce a pustular dermatitis. Poultices, being heavy, are not advocated: they should, if used, be changed every three hours. A cotton-wool jacket is useful. Greater immobility may be obtained by strapping the chest: this should pass on to the healthy side for a couple of inches back and front. An abdominal binder for the restraint of the diaphragm will sometimes prove beneficial. The child should be put to bed, and the room kept at a temperature of 65° F. The bowels must be opened and a saline mixture administered. The diet should be fluid. With effusion, three points require attention,—viz., the quantity and the quality of fluid effused, and the duration. If the pleural cavity is full, aspirate at once, lest sudden death anticipate you. By quickly opening the chest and performing artificial respiration, I once revived a child who had ceased breathing. Simple fluid, even if moderate, should not be left *in statu quo* longer than three weeks, lest the lung contract adhesions. If there is still fever, do not aspirate, as the fluid is sure to reaccumulate. Do not use a sharp hollow needle, or you will wound the lung. It is

not necessary to remove all the fluid: withdrawal of a moderate amount will suffice. Cease aspiration as soon as the child commences coughing. Sometimes two or three aspirations will be necessary. The bowels should be kept open, and a diuretic mixture containing digitalis administered, to which may be added iodide of potassium. The dull area may be painted with tincture of iodine daily. I find it a good plan to rub into the chest for ten minutes, night and morning, Scott's ointment. This may be mixed with unguentum iodi. This method is useful with damped breath-sounds from thickened fibrin. If pustular dermatitis appears, the application should be temporarily stopped. A useful plan is to limit the quantity of fluid to half a pint in the twenty-four hours; the solid diet which you will be now giving your patient must then be presented as dry as possible.

There is only one method of dealing with empyema, viz., immediate evacuation. Aspiration may be resorted to, but its usefulness is limited. It is sometimes successful when the empyema is small, is useful for young infants, or where the shock of operation would be dangerous, or preliminary to incision when the chest is very full. Another method is the passage of a medium-sized trocar and canula into the chest. Through the canula a divided rubber drain with glass insertion may be directed after the canula has been removed. The skin grasps the chest end, the other passes into a quart-glass measure, under the cot, containing some antiseptic solution. This is the siphon plan. The tube in the chest quickly loosens, the siphon action soon ceases, and the case is difficult to manage in other respects and to keep aseptic. Of all methods, simple incision through an intercostal space is the best. As regards site, it does not matter much: some advocate one, others another. The axillary interspaces are widest. With local collections, the incision must be guided by the site of the pus, and then at its most dependent position. If pointing, it may be opened there or elsewhere. Here empyemas are generally opened in the seventh or eighth interspace behind, and a large-sized rubber drainage-tube inserted. We use a rubber tube to which a rubber shield is fixed. I like it to pass into the pleural cavity and no more, just sufficient to keep the wound patent and allow free drainage. I also employ a short silver tube with a shield.[1] When there is much pus, it is better on the insertion of the tube to allow it to drain away gradually into a sufficiently thick antiseptic dressing. Serious consequences may follow

[1] A nest of three is made by Messrs. Down Brothers, of St. Thomas's Street, London Bridge, from my specifications.

sudden disturbance of the mechanism of the lung, such as œdema, frothy expectoration, and possibly suffocation. Fibrinous masses presenting at the wound must be removed. In a girl of three, I once removed two fluidounces by measure of membrane on the third day. Hemorrhage at the time of operation is rare. Dangerous collapse and cessation of breathing sometimes occur, and must be treated on the usual lines. Many of the cases were treated under carbolic spray. To say nothing of other drawbacks, I think its chilly blasts were responsible for two cases of pneumonia.

At this hospital, Sir Joseph Lister's methods of antiseptic dressings are adopted all through until the wound is superficial, when a simple dressing is employed. The tube is to be removed, cleansed from pus and fibrinous casts, and replaced. When it has entered the pleural cavity a rush of air passes through the tube. After a few days the discharge becomes serous. The tube may be driven out by coughing from squeezing of the ribs or impact of the re-expanding lung. When the discharge is trifling, a drachm or so, the tube may be discarded. There is no time-limit; occasionally a week is sufficient. In the course of a few weeks I have several times seen it surrounded by a tunnel of bone. In some cases several of the ribs are united by bony growths. With reaccumulation of pus there are two indications, a rise of temperature, sometimes not much above 99° F., and alteration of physical signs. Rise of temperature does not necessarily mean imperfect drainage or reaccumulation. In several cases it was due to scarlet fever or measles, in others to pneumonia, tonsillitis, general tuberculosis, or peritonitis, sometimes to no apparent cause, and again it appeared to be owing to a small superficial collection of pus about the wound. Conversely, a subnormal temperature need not cause alarm: this is very common in childhood, and not infrequently the temperature may be as low as 95° F. The lung can often be seen at the bottom of the wound, of a slaty color or lymph-covered. It may remain motionless or appear close to the chest-wall. With a large cavity a considerable rush of air will be heard. If close up to the chest, the lung-movements appear up and down, and not to and fro. Probing, unless there is a large and obvious cavity, is likely to prove misleading. Even if there is *apparently* a cavity of some extent, the discharge being trifling, an attempt should be made to banish the tube. After operation there is usually fair entry of air, with vesicular breath-sounds, but these may be conducted: it was so in a child with a completely-collapsed lung. When the upper lobe is consolidated, the physical signs persist for a variable period. Below the

wound there may be distant tubular breathing, vesicular murmur, or râles, according to the condition of the lung. Tubular breathing may be conducted to the opposite side, and in this case there will be resonance. With complete sealing of the cavity there may be deficient entry of air and impaired resonance. In aid of this there are several forces,—viz., full re-expansion, or the falling in of the chest with partial expansion, pushing up of the diaphragm, displacement of the mediastinum by the healthy lung. The greater the collapse the more the deformity and the greater the strain on the right heart. Cases of a few days' or weeks' duration are discharged showing merely a scar, sometimes flattening under the clavicle or about the nipple, or slight chest-shrinkage and some lateral curvature. Time and a healthy constitution will efface such traces in many cases. Expansion of the lung is induced by coughing, crying, and breathing with closed glottis. The atmospheric pressure is greater *via* the bronchi because the dressing acts as a valve, preventing the escape of air from the sac. Rarely is it necessary to excise ribs. When a child has for several months worn a drainage-tube with a profuse discharge, and when the lung shows no signs of re-expansion, or none beyond a certain point, it is advisable to facilitate the fall of the chest on the lung. Symptoms of lardaceous disease make the closure of the cavity imperative; but do not wait for these. In chronic cases, where the chance of re-expansion is remote, it is best to resort at once to subperiosteal resection. Sinuses remain in some few cases and necessitate resection. Such may be owing to a necrosed rib merely. Thus, in one child many inches of rib were exfoliated. Washing out the pleural cavity serves no good purpose, and is dangerous. Patients have died from coma, convulsions, and hyperpyrexia. If the pus is stinking, the washing out may be tried, but even then I have seen no good results. Carbolic acid and mercurials are readily absorbed. Lotio sodii chlor. one in fifteen has not this drawback. Do not keep your patient in bed longer than necessary. If he be old enough and can walk around the room without fatigue, he should be allowed to do so. Increased work is then thrown upon the lungs, and a fuller expansion invited. The day after operation, provided chloroform sickness has passed, he may have solid food. Cod-liver oil and wine of iron are to be administered. When the wound is superficial, or before that, if possible, he should be at the sea-side, and for this purpose I know of no better place than Newquay, in Cornwall, with its equable climate and invigorating Atlantic breezes. In a small section pus reaccumulates and requires reopening of the wound, sometimes resection. Thus, one case on each occasion healed, after

twice presenting herself for treatment, and finally underwent resection. The disease in this case extended over a period of five years. Now she is fast approaching womanhood, and the chest, with the exception of scarring, shows little if any trace of the empyema. From my experience gained as resident medical officer and registrar here, and subsequent acquaintance with many of Dr. Goodhart's and Dr. Taylor's old cases in my out-patients, I can tell you that the treatment of empyema by early incision is most encouraging and successful. Unfortunately, feebly-organized tissue invites tubercular deposits. Thus, in a cured empyema patient aged six it was so in the adhesions. She died of tubercular meningitis. There were no tubercles elsewhere. Finally, I have seen a serous effusion on a cured empyema side three years later; also pus on one side, serum on the other.

Surgery.

SARCOMA OF UPPER JAW.—EPITHELIOMA OF UPPER JAW.—CANCER OF BREAST.—CELLULITIS OF FINGER AND HAND FOLLOWING AMPUTATION OF THE FINGER; LYMPHOMA OF THE NECK.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL.

BY JOHN ASHHURST, JR., M.D.,

Barton Professor of Surgery and Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania Hospital, etc.

SARCOMA OF UPPER JAW.

I SHALL show you to-day two cases of tumor of the jaw, both of which call for operation. The first patient is an old man who applied to the eye dispensary with the statement that he was suffering from some impairment of vision. The surgeon in charge recognized that there was a tumor involving the upper jaw, and sent him to me. He has quite a large growth involving the upper maxillary bone, pressing the eye upward, but, as far as I can ascertain, the disease has not passed beyond the limits of the jaw. I believe it to be a sarcoma. These cases were formerly considered encephaloid, but modern microscopic methods show that the large majority of cases formerly regarded as encephaloid are really sarcomata. The prognosis in these cases of sarcoma of the upper jaw is always grave. In the first place, operation for the removal of the upper jaw is formidable, and not infrequently attended by death from shock or hemorrhage. In the second place, there is, of course, the possibility and the probability, as in all other cases of sarcoma, that the disease will recur either *in situ* or in some other part,—usually *in situ*, because sarcoma does not tend to become diffused as does carcinoma, though you may have sarcoma transferred by embolism, little portions of the growth being carried by the circulation and forming new starting-points for the disease. What may be considered as a favorable feature in this case is that the growth does not appear to have spread beyond the limits of

the bone. When the disease involves the soft tissues of the cheek and the skin, it is an unfavorable feature, for you cannot remove the entire growth without removing a portion of the skin, and then a fistulous opening remains. If the skin and mucous membrane are removed, there is nothing to underlie the skin so that the wound can be closed by a plastic operation. Then, again, if the tumor spreads backward, involving the palate, it is an unfavorable element, because you are not sure of removing the whole of the growth. The most favorable cases are those in which the growth is limited to the jaw itself.

The operation for excision of the upper jaw may be done in various ways. The plan which I prefer is that suggested by Sir William Fergusson, and is that which makes the smallest external wound, while it affords full exposure of the growth and enables you to deal with it in a satisfactory manner. An incision is first made through the median line of the upper lip, then around the nose to the internal angle of the orbit, and then transversely below the orbit as far as is necessary. This affords a free exposure, dividing but few vessels, and is a smaller incision than any other that gives the same exposure. An additional advantage is that the scar is to a large extent concealed in the natural folds of the parts. The deformity is less apparent than in any other operation, although this is, of course, a minor point. The great risk in this operation is hemorrhage. There is some risk of shock, but the main risk is hemorrhage from branches of the internal maxillary artery. I always like to be provided in these cases with the actual cautery. We have here both the Paquelin cautery and the ordinary hot iron. Usually the internal maxillary artery can be tied, but sometimes this cannot be done readily, and you have to control the bleeding by pressure or by prompt cauterization.

The operation is generally begun by removing the second incisor tooth, but as this patient is already edentulous there will be no occasion for that procedure. It is well to begin by deeply grooving the alveolus with a small saw. Three bone-sections are required: first, from the nostril to the mouth; second, from the nostril to the inner angle of the orbit; and, third, the outer attachment of the bone. In this way I remove the bone with the tumor, and any remaining portions of the growth I dissect out with scissors. We are able here to tie the main branch of the maxillary artery, and we ligate all bleeding points. As it is possible that some small portions of the growth may remain, I shall touch the surface lightly with the cautery, which will cause a superficial slough and is an additional precaution against hemorrhage. Having done this, I wash out the wound with

boracic acid solution and close it with silver-wire sutures, applying an antiseptic dressing. With the exception that the cheek is a little sunken, the deformity is not very great.

EPITHELIOMA OF THE UPPER JAW.

The next patient also has a tumor of the jaw, which appears to be an epithelioma originating in the gum, pretty far back, and spreading to the adjoining surface. Cases of this kind, where the disease originates in the mucous membrane and only secondarily involves the bone, do not require the removal of the entire bone. If you remove the ulcerated surface and the bone connected with it, you get a satisfactory result. The disease is on the right side, and I shall make an incision through the cheek, beginning at the angle of the mouth, in order to have a free exposure of the parts. I map out with the knife the portion of tissue which I shall remove, and with bone forceps cut away all the diseased bone. I then apply a saturated solution of chloride of zinc. This I think better than the actual cautery. The object is to destroy any portions of the growth that are left, or what is sometimes called the halo of malignant disease. There may be a few cells in the surrounding tissue which are not recognized at the operation. If there is a great deal of hemorrhage, it may in these cases be desirable to use the actual cautery instead of the potential cautery.

CANCER OF THE BREAST.

The last case that I will show you to-day is another example of mammary cancer, and I am glad to show it in connection with the other patients whom I have shown you, as it illustrates some different points of the disease. In this case the patient is a young colored woman, and I believe the tumor to be one of the rarer forms of carcinoma, encephaloid or medullary cancer. There is one portion of the breast which evidently contains a large cyst. It is bluish in appearance, and presents somewhat the appearance of what is known as Brodie's serocystic sarcoma, which is a proliferous cyst with intracystic growths, and which often simulates medullary cancer. It is impossible to say positively whether this is a cystic degeneration of an encephaloid carcinoma, or whether it is an example of a rare condition, viz., a non-malignant and a malignant growth existing together in the same breast. This cannot be determined until after the operation, and we may have to wait for a microscopic examination. I believe, however, that this is a carcinoma, on account of the dimpling of the skin and the presence of the other signs, to which I have called attention on

previous occasions, and also on account of the fact that the axillary glands are involved.

The treatment is clear. It is to excise the growth. It will also be necessary to open the axilla and remove the diseased glands. Looking at the tumor, you will notice the dimpled appearance. The cyst is quite evident, and the dimpled appearance of the skin is also present over the cyst, so that I am disposed to think that this is a later change in the malignant disease. I shall remove the breast in the ordinary way by two elliptical incisions, taking care to keep well into the healthy parts and away from the malignant growth. I then control the hemorrhage and remove the masses extending up into the axilla. Having removed all the diseased tissue, I paint the surface with a solution containing fifteen grains of chloride of zinc to the ounce of water, and then introduce a drainage-tube and close the wound with silver sutures, the cavity being thoroughly washed out by syringing through the tube, and an antiseptic dressing being applied.

CELLULITIS OF FINGER AND HAND FOLLOWING AMPUTATION OF THE FINGER.[1]

The case that I now bring before you is that of a man who was admitted to the hospital the day before yesterday. He had sustained an injury of the finger, and had had the first phalanx of the second finger of the left hand amputated at another hospital. After the operation he went to his own home, and, as often happens in these cases when the patient is not under careful supervision, complications have arisen which have rendered his condition much more serious than it was at first. The patient applied here with a temperature of 104.4° F., suffering intense pain, and altogether in a very uncomfortable state. The finger was much inflamed and swollen, and there was a tendency to the spread of inflammation up the forearm. There are several inflammatory affections which may occur as wound-complications, which must be distinguished from each other because their treatment somewhat differs, although two or more of them may coexist in the same case. In the first place, there may be diffuse inflammation of the cellular tissue. This is what we have here, the inflammation starting from the injured part and involving to a certain extent the hand, but not going as far as suppuration; this is ordinary cellulitis, which we often have following upon accident or injury where the patient is not properly cared for. Then we may have, in connection with this, inflammation

[1] Delivered at the Pennsylvania Hospital.

affecting not the cellular tissue directly, but the lymphatic vessels, constituting lymphangitis, which is always accompanied by inflammation of the lymphatic glands. In lymphangitis or angeioleucitis there are bright red lines passing from the affected part up the limb, the color being of an almost scarlet hue, and the lines being fine and sometimes constituting a net-work. With this there is also inflammation of the lymphatic glands. Those of you who are near enough can see that there is here a tendency to the formation of red lines on the forearm. Sometimes more than the first set of glands will be involved, and not only those at the elbow but also those in the axilla may be enlarged, the septic substances absorbed from the wound having passed through the first filter, as it were,—for the lymphatic glands serve the purpose of a filter,—and having reached the second.

Then there is another form of inflammation of vessels,—that is, inflammation of the veins, constituting phlebitis. Properly speaking, phlebitis is a condition secondary to thrombosis. When inflammation affects a vein, its first effect is to cause clotting of the blood. The older writers supposed that there was an inflammation of the lining membrane of the vein, which led to pyæmia. Their explanation was that suppurative inflammation affected the lining membrane of the vein, and that the pus formed was carried into the general circulation. As a matter of fact, that does not occur. The first effect of the inflammation is to cause thrombosis or clotting of the blood, which at once cuts off the seat of injury from the general circulation. You may have secondarily what Virchow calls mesophlebitis or periphlebitis. There may also occur embolism from a portion of the clot breaking off, and this may lead to the so-called metastatic or secondary abscess. On the other hand, you may have a large portion of the clot break off, blocking some branch of the pulmonary artery, and perhaps leading to death in this way. You may have ordinary embolism, or you may have capillary embolism causing these infarcts or metastatic deposits. Phlegmasia alba dolens originates in a thrombosis of the large veins connected with the uterine sinuses, and as a result there is plugging of the femoral vein. From this you may have a fragment carried into the general circulation, blocking the pulmonary artery and causing instant death. You may have the same condition of the veins of the upper limbs, what might be called a milk leg in the arm. You may have it in men as well as in women. It is a thrombosis of the large veins due either to traumatism or to the absorption of septic material.

We have still another form of inflammation the result of injury,—that is, neuritis. This is attended with pain and tenderness in the line

of the nerves, but not with redness, unless the inflamed nerve is quite superficial. With care, the diagnosis between inflammation of the lymph-vessels and inflammation of the veins can always be made. Inflammation of the lymphatic vessels presents bright red, almost scarlet, lines passing up the arm, sometimes in the form of a net-work. The redness in phlebitis is of a dusky hue, much deeper than in angeioleucitis. In the latter condition there will be certain points at which there are painful swellings from inflammation of the glands. You do not find this in phlebitis. An inflamed vein feels like a hard cord from the contained clot. There may be at certain points of the inflamed veins parts that are somewhat enlarged, in the position where the valves exist. There is also great tenderness over the inflamed vessels. You do not have much tenderness over the red lines of angeioleucitis. In simple diffuse inflammation of the areolar tissue there is, of course, the general redness of inflammation, without the redness being limited to the course of the vessels. There is a form of cellulitis which is not erysipelatous. Many writers believe that where you have a diffused inflammation of the cellular tissue it is always the result of erysipelas. This is not the case. Often erysipelas does cause this diffused inflammation of the areolar tissue, but you may have it from traumatic causes. I do not believe that in this case there is any erysipelas. It is a simple inflammation which has extended along the planes of cellular tissue, and which has now reached the hand, and may end in the formation of a palmar abscess. On the back of the finger there is a large prominence, which is yellowish on account of the presence of pus, and is pointing. This "pointing" is an interesting process. It is due to the gradual disappearance of the wall over the collection of pus, the result of pressure and a process analogous to interstitial absorption. A part of the wall is probably thrown off inwards, increasing the amount of pus, and the wall gradually becomes thinner until a small opening takes place, or sometimes a small vesicle first forms, which breaks. When you find this "pointing," it is well to make an incision, as you will thus save more skin than if you allow the abscess to break of itself. I find no evidence of pus in the hand. The question as to the opening of felons and palmar abscesses often comes up. As soon as you are sure that pus is present an incision should be made, and by it you may prevent destruction of the sheath of the tendon and sometimes of the bone. On the other hand, it is not desirable to make an incision simply when the finger or hand is inflamed. For even where there is a threatened abscess, the inflammation will sometimes subside under careful treatment without the formation of pus. Such

a case should be watched, and as soon as you are satisfied that pus is present an incision should be made. We shall to-day evacuate the pus in the finger and watch the condition of the hand. For opening felons and palmar abscesses the best instrument is a bistoury. The best way is to introduce the knife directly into the part, and enlarge the opening as you withdraw the instrument. In this way you give the minimum amount of pain, and if the patient draws his hand away it tends rather to facilitate the operation than to defeat it. You should not open a felon directly in the middle line of the finger, as you might thus injure the tendon. Neither should you open it quite on either side, for then you might wound one of the digital arteries. The incision should be made midway between the median line and the lateral aspect of the finger. After opening it is not desirable to squeeze the part: allow the pus to evacuate itself by the natural contraction of the tissues, assisting the removal of the discharge by simple washing. In these cases it is better not to use corrosive sublimate, which causes much pain if it comes in contact with a fresh wound. The poultice will be reapplied. If you think it necessary to make pressure at all, it should be done through wet cotton or moist sponges. In that way little pain is caused. We are now using a poultice made with laudanum, which forms a very soothing dressing.

The patient is taking internally a fever mixture, containing sweet spirit of nitre, morphia, and acetate of ammonium. In simple cellulitis it is not necessary to do anything more. In cases of cellulitis from erysipelas I think it desirable to put the patient on the use of the tincture of chloride of iron, which is very valuable in phlegmonous erysipelas and may be of advantage also in the cutaneous variety. The same remedy is adapted to the treatment of angeioleucitis, which often accompanies cellulitis. In phlebitis, as general treatment, we usually administer quinine. In neuritis, anodynes are probably the most serviceable remedies, both generally and locally, and benefit may also be derived from the use of counter-irritants.

LYMPHOMA OF THE NECK; OPERATION.

The next patient is a young man who presents himself with a large tumor on the right side of the neck, in the posterior triangle. We recognize the tumor as a lymphoma. This term lymphoma is used for all tumors of which the structure resembles that of the lymphatic glands, and which are found either in those glands or in their proximity, or even in places where there is no lymphatic structure. Another name is lymphadenoma, which is frequently employed for the diffuse

form of the affection known as Hodgkin's disease. In this affection the lymphatic enlargements are found in many different parts of the body, and run a malignant course. Where the tumors originate in lymphatic glands there is more hope from interference. Sometimes these enlargements depend upon the presence of tubercle. One argument in favor of removing enlarged glands is that by so doing you may prevent the development of general tuberculosis. It is believed that the glands are centres of infection, and that from them the disease may extend to other parts of the body. The older writers believed that the presence of lymphatic enlargements of the neck was a safeguard against tuberculosis; and I remember the late Dr. George B. Wood lecturing in this hospital and congratulating a patient upon the presence of these enlarged glands in the neck as probably having saved his life. At that time it was not thought proper to operate upon enlarged glands; but, as I have said, the modern view is rather the other way, that they are centres of infection, and should be removed as a prophylactic measure. Very radical operations have been urged. My own feeling is that here, as in dealing with every malady, the surgeon must vary his treatment on consideration of the circumstances of the individual case. If in a young person we find several enlarged glands in the neck, and if we can treat the case satisfactorily without an operation, it is, I think, proper to do so. In many cases where suppuration has occurred, and where the glands do not present a very distinct mass, you will find it almost impossible to remove them satisfactorily. You may remove a certain number, but others will remain. In these cases it may be better to deal with each gland as it enlarges. If suppuration occurs, evacuate the pus and scrape out the gland with the sharp spoon or curette, and repeat this operation as other glands become involved. By doing this you will have a more satisfactory result than by attempting excision, and without the risk of extensive scarring in the neck. I do not, therefore, think that an operation which does not seem to be called for on account of the size or connections of the tumor is justifiable simply on the possibility that twenty or thirty years afterwards the patient may become the subject of tuberculosis. If, however, the growth can be readily removed, and is of such long duration that it probably cannot be satisfactorily treated in any other way, it is proper to operate. In this case you can see a large mass in the posterior cervical triangle and extending under the sterno-cleido-mastoid muscle. I may find it necessary to divide this muscle and afterwards unite it by catgut sutures. I shall begin with an S-shaped incision, and cautiously work down towards the deep attachments of the mass. The external jugular vein comes

in view, and I tie it in two places and divide it between the ligatures. By working under the sterno-mastoid muscle I gradually separate the tumor, tying all vessels that bleed. As you see, the carotid artery and the internal jugular vein are exposed. Having removed the growth, a drainage-tube is introduced, and the wound is closed and dressed in the usual manner.

A CASE OF SUPRA-PUBIC LITHOTOMY.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY COLLEGE HOSPITAL, LONDON.

BY MR. CHRISTOPHER HEATH,

Holme Professor of Clinical Surgery in University College, London, and Surgeon to the Hospital.

GENTLEMEN,—You saw me last Wednesday open a patient's bladder above the pubes and extract a stone, which is here in a box. You will remember I said at the time that the reason I preferred to open the bladder was because I knew that it was a phosphatic stone in an elderly patient with a diseased condition of that viscus, and I thought therefore it would be better to open the bladder and to extract the calculus above the pubes than to attempt to crush the stone and wash the fragments out, which I no doubt could easily have done. When I had opened the bladder I found what I had not expected,—that there were several fragments of an old calculus; and it appears that this patient had been in another hospital a year ago. He was put under ether there, but it is not clear what was done to him. I think there can be no doubt, however, that a lithotrite was passed and a small stone broken up; but it is quite evident that those fragments were never removed, and that they have been in the patient's bladder for the last year or more, and have set up a considerable amount of irritation there, with chronic cystitis, and it was for that reason that he applied to the hospital. He came here with no knowledge that he had a stone in the bladder, but with the ordinary history of old people with chronic cystitis,—viz., that he had very offensive alkaline urine, which he had to pass frequently; and he came in order that he might be submitted to the treatment which had done him good before,—viz., having his bladder washed out. I passed an instrument for him when he first came, and drew off a considerable quantity of highly ammoniacal, offensive urine. That urine was very turbid, contained a large quantity of pus, and those who were with me at the time will remember how exceedingly offensive it was; and so I proceeded to treat the patient in the way in which we

in view, and I tie it in two places and divide it between the ligatures. By working under the sterno-mastoid muscle I gradually separate the tumor, tying all vessels that bleed. As you see, the carotid artery and the internal jugular vein are exposed. Having removed the growth, a drainage-tube is introduced, and the wound is closed and dressed in the usual manner.

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ordinarily treat cases of chronic cystitis; that is, I directed that his urine should be drawn off periodically, that his bladder should be washed out once a day, and, finally, that he should be taught to use his own catheter with the view of carrying on the treatment when he went out. But when I saw him a second time I asked him a few more questions, and he spoke rather more of pain than he had done before, and I said, "Well, pain is a symptom rather of stone in the bladder than of cystitis, and I think it would be wise to pass a sound, and see whether there is a stone or not;" because, as you will remember, chronic cystitis is one of the results of stone in the bladder, and patients who have anything like senile enlargement of the prostate are exceedingly likely to have both chronic cystitis and stone, the stone being the result, usually, of the cystitis, but in some cases the cystitis being the result of the stone. I passed a sound and without the least difficulty felt a stone, and I had the opportunity of demonstrating its presence to those who were with me on that occasion, because by holding the handle of the sound while I moved it they were able to feel the instrument impinge upon the stone. There could be no question whatever about it.

Under these circumstances, it was quite clear that we should not be doing justice to the patient by merely treating him for chronic cystitis so long as the cause of the trouble or the presumed cause of it remained; and it became a question what would be the best plan to adopt for the removal of the stone. Now, I was quite sure, from the feel of it, that it was not a very large stone, and I felt also tolerably sure that it was mainly a phosphatic stone. There would therefore have been no mechanical difficulty in introducing a lithotrite of the ordinary form and crushing the stone, or in washing away the fragments afterwards by what is now known as lithotrity at one sitting, or Bigelow's operation, by which we generally succeed in clearing the bladder of all the fragments of the stone at one operation. It is an exceedingly good operation, and I have done it many times now, and I do not think there is a better operation for certain calculi; but then you must bear in mind the particular circumstances of this patient. He is a man of seventy; he has had chronic cystitis for some considerable time, and, as is so common with males who reach the age of seventy, he cannot completely empty his bladder. There is, when he has made water, a certain residuum of urine still in the bladder, which is apt to decompose, to set up irritation, and to become alkaline. This decomposition tends necessarily to the rapid reproduction of a phosphatic stone. Now, the experience of all surgeons in these cases is that, although you may clear the bladder of the actually existing stone, there is such a

tendency to reproduction of phosphatic material that you do not really cure your patient. The tendency to form a calculus continues, and from time to time, at intervals of a few months, probably, the lithotrite has again to be used and the bladder again cleared. Under such circumstances I think it is better, and I followed out the treatment in this case, to clear the bladder absolutely of the stone, to make quite sure that no little bit of any kind is left behind, and also to insure the bladder rest and drainage by doing some cutting operation.

Of course, if you cut open the bladder by any method, the bladder enjoys rest for the time being, and that, after all, is a very important factor in the cure. You know how we endeavor to give rest in all cases where we wish healing to take place. The best example is a fracture. You secure a fracture by splints, and healing takes place in the ordinary natural method. You have a bladder to treat which has been constantly irritated for some months, or even years, by the presence of decomposing urine and even calculous matter; you give that bladder absolute rest for some weeks, and you find that it becomes healthy, and the patient, of course, experiences great relief. Nothing is more trying for a patient than that constant irritability of the bladder and constant straining to make water which you find in these chronic cases, and therefore I think there can be no doubt that it is an immense relief to the patient to have his bladder absolutely at rest, and the water coming away mechanically without any effort on his part, for some few weeks. Well, then, having made up my mind to do some form of lithotomy, the question was, what would be the best method to follow. A few years ago there would have been no question about it, because surgeons were all in the habit of doing some form of perineal lithotomy, either the lateral or the median. The lateral operation perhaps gives more room, but it has dangers of its own, and therefore, where the object is simply to take out a small stone and afterwards to give thorough drainage, median lithotomy, which is an easier operation, perhaps, may be preferred; but within the last ten years or so the operation above the pubes, which you saw me do on Wednesday, has come very much into fashion, and I was very glad to avail myself of it, and, as you saw, I succeeded, without any particular difficulty, in reaching the bladder by this method.

It is curious to look back on the history of this operation. It appears to have been done first by a French surgeon, Peter Franco, in 1541, and, as far as the record of his cases goes, it appears that he first cut a child. He happened to have a boy with a large calculus which made a prominence above the pubes. You will remember that in chil-

dren the bladder is considerably higher than it is in the adult, and therefore in a child's bladder with a large stone there would naturally be a prominence above the pubes. Well, finding that to be the case, and finding, also, that he could not push the stone downward in the bladder so as to make it prominent in the perineum at all, he appears to have cut upon the stone in the median line and to have extracted it without any particular difficulty; and other surgeons followed his example. The well-known Cheselden was a great lithotomist in London, and his portrait may be seen at St. Thomas's Hospital, where he held the special appointment of lithotomist—he was not surgeon—to the hospital, and where he performed several supra-pubic lithotomies in children and adults with very good results. Then, for some reason, he seems to have abandoned this operation, and it almost fell into disuse, while the lateral operation or the median was employed by surgeons generally, although every now and then, in the case of a very large stone, surgeons did venture to do the supra-pubic operation. The objection to doing it on the adult was the danger of wounding the peritoneum. I dare say you know that up to about thirty years ago surgeons had very exaggerated ideas of the danger of opening the peritoneum. To open the peritoneum at all in the case of hernia, for instance, was thought to add considerably to the danger of the operation, and anything like a free incision into the peritoneum, such as we are in the habit of making nowadays for ordinary ovariotomy, or operations of that kind, was never thought of. Well, we know now that with strict cleanliness, and possibly with antiseptic precautions (though I believe cleanliness is really the great point), you can open the peritoneum without any particular risk to the patient. I do not mean that it is a thing to be done rashly or unadvisedly; but still the peritoneum may be opened and the patient recover without any bad symptoms. But there was a danger of opening the peritoneum when the bladder was involved, an extra danger, in this way,—that fluid from the bladder might get into the peritoneum; and you can easily understand that any urine getting into the peritoneum might cause serious trouble for the patient, and particularly urine from a diseased, chronically-inflamed bladder. Alkaline urine, probably swarming with bacteria, would be very detrimental to the peritoneum, and therefore the danger was not an imaginary one, although, of course, we know that the bladder and the peritoneum may be opened at the same time and the patient recover. I will just remind you of a case that occurred here last year; I think it was almost the last operation that the late Mr. Berkeley Hill did in this room, and perhaps you may

remember that he was going to open the bladder to extract a growth and by some mischance the peritoneum was opened too. I remember the circumstance extremely well, because I was present at the time. Well, he recognized the accident, and the peritoneal cavity was well washed out and the tear was carefully stitched up, and that patient made an absolutely good recovery: so that I quote it as an example where a patient has recovered, even with the untoward accident of opening the peritoneum and the bladder at the same time.

What brought the operation of supra-pubic lithotomy into fashion again was the discovery that by putting a bag into the rectum and distending that, as well as the bladder, the bladder could be brought up to the front of the abdominal wall and could almost to a certainty be opened without involving the peritoneum. Now, the credit of that discovery really belongs to Dr. Garson, and I have here Dr. Garson's paper with the illustrations in it, and I would like to point out to you the anatomy of the parts. This paper was first read before the Association of German Surgeons at Berlin, and afterwards published in the *Edinburgh Medical Journal* of 1878, and here is the paper. Now, if you will notice, there is a frozen section of the male pelvis, and you see the urethra leading, of course, into the contracted bladder; behind is the symphysis pubis. On the opposite side you will see the drawing made from the same preparation when the bladder had been moderately distended with fluid and when the rectum had been also considerably distended. And now you will observe what the result is, that whereas in the contracted condition the apex of the bladder is level with the symphysis pubis, here, on the contrary, when the bladder and the rectum are considerably distended, we have the front of the bladder coming into apposition with the abdominal wall, and the reflection of the peritoneum is about a couple of inches farther up, so that the risk of wounding the peritoneum there is exceedingly small, because the distention of the bladder and also the distention of the rectum push the peritoneum well out of its position. That paper at first did not seem to produce any results, but Professor Petersen, of Kiel, in 1880, brought before the same society—the Society of German Surgeons—some cases showing the practical outcome of this suggestion. He showed that by putting an egg-shaped india-rubber ball into the rectum and distending this ball with from ten to fifteen ounces of fluid you could push the bladder forward, and by distending that again with some eight or ten ounces of fluid the peritoneum could be got completely out of the way and the front of the bladder brought underneath the abdominal wall. Petersen's success was so great that

it immediately attracted attention. Sir Henry Thompson had several successful operations, and he devoted a lecture to the subject in this lecture-room some few years ago, and since that date it has become a recognized operation for large stones. Large stones, of course, can thus be got out very much more easily than they could through the perineum, and every one knows that large stones extracted through the perineum are very apt to inflict considerable damage upon the soft parts, and to kill the patient simply by laceration or stretching of the tissues. To return to our patient: I will just say, in passing, that on Wednesday I had a little difficulty, more than I ought to have had, because I had not sufficiently distended the bag in the rectum; I had miscalculated the amount, for, although I put eight ounces of fluid into the bladder, I had not put sufficient into the rectum, and you may remember when I cut through the abdominal wall that the bladder did not present quite as much as it ought to have done, therefore I had to push it up with a catheter before I could make quite sure that I had got it, and then there was no further difficulty.

In cutting through the abdominal wall your incision should go well down on to the pubes and about two inches above it, making altogether a three- or four-inch cut, according to the size of the stone. Then, working strictly in the median line, the surgeon gets at once upon the bladder; and at this part of the operation I avail myself of a suggestion by Sir Henry Thompson, who recommends the use of a blunt instrument. He had a particular instrument made, but I am quite content with this little one, which is an ordinary nail-cleaner, and the sharp point of which I find exceedingly useful for tearing through the tissues down to the coats of the bladder, so as to prevent any of the veins from bleeding. In that way you can make quite sure you are actually on the bladder and push the peritoneum up, and then it is wise either to put a tenaculum through the coats of the bladder, or, as I prefer to do, to put in a couple of stitches, and thus secure the bladder before you open it. If you open it simply by pushing a knife in, the moment the fluid is out of the bladder it sinks a little back into the pelvis and eludes your grasp; and therefore it is safer to put in a couple of stitches and to hold the bladder in that way, as you saw me do, so as to be quite sure to puncture it in the median line and have no difficulty from its slipping back. Having opened the bladder, you pass your finger in; and the finger is, after all, the best judge of what the size of the stone may be. I found, as I expected, that I had only a small stone to deal with, but the moment I touched it it broke up into these fragments, which are really fragments of another stone connected by phosphatic material on the surface. It seems to have been broken up by a previous operation, and then all these fragments became coated with phosphates, and there was a good deal of phosphatic material over the coats of the bladder.

Now, of course there could be no difficulty whatever in extracting so small a stone as this one has proved to be. I first took out the main portion with a small pair of forceps, and then I removed the rest with the ordinary lithotomy scoop. But if it had been a large stone, of course the opening would have had to be made commensurate.

I have brought here this stone also, which I took out of a gentleman's bladder three years ago, in order to show you to what a considerable size these collections may grow. This specimen is perhaps one of the largest that has ever been extracted above the pubes. It weighs between nine and ten ounces, and the extraordinary thing about it is that it projected above the pubes, could be felt there, and must have been there for a long time, and yet the patient, who was under forty years of age, had been suffering from bladder-symptoms all his life. The medical man whom I met, it so happened, had brought this man into the world, as he had attended his mother in her confinement, and he had known the patient all his life. The patient told me that from his earliest days he had had bladder-trouble; and the extraordinary thing is that, up to the time that I saw the patient, nobody seemed to have thought of passing a sound into his bladder. Of course one doesn't like to speak harshly of a brother practitioner, but that patient ought to have been sounded many years before, when the stone was of small size, and I am rather inclined to agree with Sir Henry Thompson when he says in one of his lectures, "A large stone means neglect on *somebody's* part," not necessarily the doctor's, because the doctor may not be consulted; but that a patient should go on with symptoms pointing to stone in his bladder without getting advice means that he lets that stone go on growing until, from a small and comparatively simple matter, it becomes an extremely dangerous thing.

With a small stone like this, as I say, there was no difficulty in extracting it. And then comes the question, What should be done after that? Should I close the bladder, or should I leave it open? Now, you will easily understand, from what I said before, that my object was to get proper drainage for the urine, and I had no intention whatever of closing the bladder; on the contrary, we put in a large tube fitted with a flange, to insure that the urine shall run out for some time, so as to give the patient perfect rest. But I was not content with that, for you will remember that I proceeded to treat the lining mem-

brane of the bladder with a strong solution of nitrate of silver; that I passed this little vulcanite speculum through the wound and introduced through it a wire which has a spiral at the end, so as to enable it to hold a piece of cotton-wool. That cotton-wool was dipped in a solution of nitrate of silver, twenty grains to the ounce; and you will remember that I mopped freely all round the bladder. Now, I ventured to do that because I have had considerable experience in the treatment of chronic cystitis in the female bladder. In former years, when I was connected with the Hospital for Women, I had a good many cases there of chronic cystitis, without stone, following after confinement, or from various other causes. In these cases I found that by dilating the urethra, which is in the female extremely short, so as to admit the small speculum, I was able to mop out the interior of the bladder very easily, and was able thus to change alkaline and offensive into acid and comparatively healthy urine almost immediately. Therefore I adopted the same plan here. I should not have ventured to do it if I had not had a free drain from the bladder, because the complicated male urethra would be apt to be irritated by any treatment of that kind, and it is quite conceivable that if you mopped out the patient's bladder without securing a thorough drain you might have retention from spasm or irritation of the urethra. But, knowing that I had a thorough drain, I was not afraid to pass the speculum and mop the bladder thoroughly, and with this result: the next day when I went round I told the house-surgeon to draw out with a small tube and a syringe a small quantity of the urine from the bladder, and I tested that immediately, and found that it was distinctly acid. Now, I do not think that patient had had acid urine for a considerable number of months, and yet here the very day after the operation the urine was distinctly acid, and it was fairly clear also. I may say that the next day, when we went round, if you remember, on Friday, I had the same experiment repeated before you, and then we found that the acidity had passed off and that the urine was neutral, or slightly alkaline; and it has remained so ever since. You will see when I dip this reddened litmus-paper into the test-tube it becomes slightly blue, for the urine is not acid; it is neutral, or very slightly alkaline. So, at all events, we have gained very considerably there; and now we shall be able to wash the patient's bladder out. I shall employ a weaker solution to wash the bladder out with, and I have no doubt the result will be that we shall get the bladder into a very much more healthy condition before the wound closes. Now, just contrast in your mind's eye the urine now passed with the urine as it was before the operation. It was stinking, ammoniacal urine, containing a large quantity of pus, having the characteristic appearance of "ropy mucus," and passing with a flop from one vessel to the other when poured out. Now it is limpid urine; there is a little sediment, and if you put the test-paper in you will see that it makes it just a little darker, so that it is as nearly neutral as it can be, and therefore satisfactory, considering that it is now four days since I mopped the bladder out, and that nothing further has as yet been done.

This patient is going on as well as he possibly can for so old a man, and I have no doubt he will make a perfectly good recovery. His future will be that of most old men who have any bladder-trouble at all,—and almost all old men do have bladder-trouble,—viz., he will probably not completely empty his bladder, but he will be able to make water. At the time of the operation I passed my finger well down to the prostate, to see if there were any projecting third lobe, as we call it, at the neck of the bladder. If there had been, I was quite prepared to remove it with a pair of scissors, to do what is now called *prostatectomy*,—*i.e.*, to cut out a portion of the prostate, after the manner of the Leeds surgeons. But when I put my finger down to the neck of the bladder I found that there was no projecting mass of the kind, and there was nothing to warrant any interference. No doubt there is an enlargement of the prostate, but it is not very marked. Under those circumstances the patient will be able to make water satisfactorily up to a certain point, but he probably will never quite empty his bladder, and that is the fact which is so important to bear in mind, that there is a certain amount of urine left after each micturition, which gradually accumulates to three or four ounces. If you allow the patient to go to bed with that three or four ounces,—of course, further secretion of the urine going on,—the bladder soon becomes more or less full, and he then has to get out of bed to empty it. This may recur two or three times in the night, and the patient's rest is very much broken, and you will find that his health suffers accordingly. If, on the contrary, you pass a catheter for him the last thing at night, or teach him to pass one for himself, then the patient empties his bladder absolutely when he goes to bed, and he goes for five or six hours before the accumulation of urine is sufficient to make him wish to empty his bladder, and you secure a very considerable amount of rest for him. That is the thing which we shall teach this patient. He has been already taught once, so it will not be difficult. We shall provide him with a catheter; he will then be able to draw off his urine every night when he goes to bed, and if he is at all a clever person he will be able

to wash his bladder out also, and in that way, starting him with a perfectly healthy bladder, by the rest we shall give it, I hope we will prevent the re-formation of the stone. The question of further treatment here I hope will not arise. If it should, it will be very easy to pass a sound and see if there be any new stone developed, and, if necessary, to deal with it with a lithotrite.

This patient did very well for nearly three weeks after the operation, and then, I am sorry to say, he sank into a condition of collapse and finally died. The fact is, he was worn out by the irritation of this large stone, and I have no doubt that if I could have procured a post-mortem we should have found his kidneys in an exceedingly diseased condition, the result of the prolonged irritation.

TUMOR OF THE HARD PALATE; ACUTE APPENDICITIS; PERINEPHRITIC ABSCESS.

CLINICAL LECTURE DELIVERED AT THE JEFFERSON MEDICAL COLLEGE HOSPITAL.

BY W. W. KEEN, M.D., LL.D.,

Professor of the Principles of Surgery in Jefferson Medical College, Philadelphia.

GENTLEMEN,—I have three cases to bring before you to-day, one of appendicitis, another a perinephritic abscess, and the third a tumor which arises from the hard palate. I shall operate on the mouth case first, since it is never wise to operate on infectious cases and afterwards on those which are not, lest the later ones become infected and suppurate.

TUMOR OF THE HARD PALATE; REMOVAL; RECOVERY.

CASE I.—This man is twenty-six years of age; his father died of phthisis; his mother is healthy, and he himself has always been healthy until ten years ago, when he noticed a small tumor on the back part of the roof of his mouth. This has gradually increased since, and now he has difficulty in swallowing and talking. When I examine the mouth I find that there is a tumor attached to the roof of the mouth at the junction of the hard and soft palates. It is not connected with the base of the skull, as I have determined by the finger and the laryngoscopic mirror. It is about the size of an egg. There is some bleeding and some discharge.

The trouble in these cases is chiefly the hemorrhage. What I propose to do here will be, first, to split the cheek backward and slightly downward so as not to injure Steno's duct, and, as far as possible, to avoid the branches of the seventh nerve. Then I can lay his head on one side with a pillow under his shoulder, and the blood can escape sideways and tracheotomy will not be necessary. I will then sever the attachments to the soft palate on each side and below, next chisel it loose from the hard palate, seize the mass with a pair of forceps and quickly wrench it loose. I will perform the operation as quickly as pos-

to wash his bladder out also, and in that way, starting him with a perfectly healthy bladder, by the rest we shall give it, I hope we will prevent the re-formation of the stone. The question of further treatment here I hope will not arise. If it should, it will be very easy to pass a sound and see if there be any new stone developed, and, if necessary, to deal with it with a lithotrite.

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sible, on account of hemorrhage. Speed but not haste will be my object. Two or three sponges pressed firmly upon the bleeding surface will arrest most of the hemorrhage, and if pressure does not stop the bleeding I will tie any large vessel, or, if necessary, use the Paquelin cautery. In some of these cases it is difficult to get a good light, and I propose to have a mirror with a small electric light attached to my forehead, so that I can throw the light in the back of his throat, if necessary.

In these cases you must always operate as quickly as possible, not wasting time over each bleeding vessel (except those in the cheek incision), until the tumor is removed. You must operate quickly, too, for another reason,—namely, that you cannot give ether while operating, and you wish to avoid pain by the speedy termination of the operation.

I have now split the cheek and controlled the hemorrhage. Next I get him well etherized again, and now you see the tumor. It is, as I said, about as large as an egg. I think it is a sarcoma of the spindle-cell variety, but it is rather hard, and may be a fibroma, which will have to be determined by an examination with the microscope. The tumor having been quickly removed as above described, I find the hard palate has been left intact except at one small point, and that only the anterior layer of the soft palate has been removed. The bleeding is not very serious. Pressure has stopped most of it very readily, and the Paquelin cautery does the rest. The wound in the cheek is then sutured and dressed. [The microscopic examination showed the tumor to be a small spindle-celled sarcoma. The patient easily recovered, and went home in ten days, with a healing ulcer in the roof of the mouth.]

APPENDICITIS; OPERATION; RECOVERY.

Case II.—This case is an admirable lesson upon the need of operation in appendicitis in spite of seeming but delusive betterment. I wish you to understand it thoroughly, as you will often have to deal with its like, and the treatment of late years has improved amazingly, owing chiefly to the labors of American surgeons. Several series of post-mortem examinations have shown us that about one-third of all adults, taken indiscriminately, at some time or other during their lives have suffered from attacks of appendicitis from which they have recovered. It is, therefore, a very common disorder, though it is by no means always recognized, for the attacks, unless severe, are often believed to be simply colic or other mere intestinal disorder. Willard Parker, who, in 1867, was the first to impress upon the profession the need for operation, called it, in the vocabulary of his time, "peri-typhlitis," under the impression that the trouble began in the head of the colon; but we now know, from more accurate observation, that in ninety-nine cases out of one hundred the disorder begins in the appendix; not from the historical grape-seed or the hypothetical apple-pip, but from a concretion of fæcal matter, from ulceration beginning in the mucous membrane following occlusion of the calibre of the appendix with retention of its secretion, or from gangrene. Speaking clinically rather than pathologically, these cases may be divided into five classes. First, those with slight but well-recognized symptoms, which after reaching a certain point pass on to resolution. This is probably the course of the large majority of cases, and especially of the unrecognized ones. Secondly, those in which rupture of the appendix takes place without previous agglutination of the intestines. The contents of the appendix escaping into the peritoneal cavity will produce a widespread and often fulminating peritonitis, so that your patient may die even within twelve hours. In the first class no operation is necessary; in the second class no recovery is possible without the speediest operation that can be done. The third class lies between these two, and of this class our present case is an admirable instance.

This patient, a man of forty-five years, began to be ill on February 3, eight days ago, with pain all over the abdomen; but by the third day the pain had gradually become focussed in the right iliac fossa and was quite severe. He entered the hospital night before last, and when I saw him I came ready to do an operation if necessary. He had had considerable fever, but it had abated so that when he entered the hospital his temperature was only 100°. He was not suffering much pain, and was, in fact, what might be called almost comfortable, although sick. In the right iliac fossa I found distinct tumefaction and resistance to the touch over an area about two inches in breadth, parallel with Poupart's ligament. The right leg was flexed at the hip, as this was the most comfortable posture. There was no œdema, nor was the tenderness very great, nor the pain severe. The most tender point was two inches from the anterior superior spine on a line drawn to the umbilicus. To find this point of greatest tenderness (which Stimson has proposed to call "McBurney's point," as McBurney was the first distinctly to formulate its existence and to point out the manner of finding it), examine with the tip of one finger, and not with the whole hand; or, better still, ask the patient to indicate with one finger the point of greatest tenderness. The point corresponds about with the attachment of the appendix to the colon; and if you find tenderness here with the other rational

signs of appendicitis, you may be almost sure that that is the disorder you have to deal with. But this so-called McBurney's point may be delusive. The point of greatest tenderness is really the point of greatest inflammation. I have seen it just under the liver when an abscess was caused by gangrene of the tip of an appendix ascending behind the colon, or at the brim of the pelvis when the appendix lay transversely.

As the case was not urgent, the time evening, and the light necessarily insufficient, I decided to watch the case for twenty-four hours or more. Yesterday morning his temperature was only 99°, and I thought it barely possible that he might escape without an operation. But this morning I found that his temperature had gone up to 101.2° last night, and I decided instantly to operate, and shall do so before you. This is the eighth day of his disorder. Willard Parker laid down the rule that an operation ought to be done between the eighth and twelfth days, but, as the statistics of Dr. Fitz have shown, sixty-eight per cent. of our patients would die by that time, and so, instead of waiting till the eighth or twelfth day, surgeons gradually learned to operate more promptly, until Fitz, in a discussion last year before the Association of American Physicians, declared that the second or third day was not too early, and in my opinion he is quite right.

All this time you will notice that I have been illustrating the third clinical class, in which a localized abscess forms, by the case in hand. You must remember that this third class is suddenly transferred sometimes into the second class by the rupture of the abscess and the consequent lighting up of general peritonitis.

Hence when you operate in such a case as the present, with undoubtedly a local circumscribed abscess, you must be extremely careful to use the greatest gentleness after you have opened it, lest you should rupture the sac and pour out its contents into the peritoneal cavity. I have known a rough assistant to rupture such a sac during the operation, undoubtedly producing the death of the patient; and not rarely it will rupture spontaneously, if the operation is delayed, with a similar fatal result. Hence the great danger of delayed operations.

The fourth class of cases are those which run a long chronic course for months or a year, but these are very rare. The fifth and last class are those of recurrent appendicitis, in which there may be even a dozen attacks, as in Bernardy's case, in this city, in which twelve attacks occurred within eleven months. For a very excellent discussion of the whole question of appendicitis in its various bearings I beg to refer you to the Transactions of the New York State Medical Society for 1891.

To return to our case. Having now looked over its various symptoms, you will see that the man does not seem seriously sick, and that his temperature is not high, and yet, as I think I shall convince you, he must absolutely be operated on, for he undoubtedly has an abscess which, if not evacuated, places his life in the greatest peril. Remember especially that, when two, three, or four days have passed and no betterment occurs and the local and physical signs remain the same, with slight tumefaction, tenderness, and moderate fever, your duty is to operate. If, as in the vast majority of cases, you find pus, you will almost certainly have saved life. If you find none, you will probably find the appendix diseased; and if you find nothing wrong,—which will not be more than once in a hundred times,—the operation will scarcely have added anything to the risk of the patient. If the appendix be found distended, inflamed, or otherwise diseased, tie it as close to the cæcum as possible and cut off the diseased end, and then you may either invaginate the stump and cover it by a few stitches through the outer coats of the colon, or simply disinfect it and let it alone, as you prefer.

I now make an incision parallel with Poupart's ligament and two finger-breadths above it, over the site of the tumefaction. After cutting a little distance into the abdominal wall I notice that the tissues are matted together, which clearly indicates to me that there is inflammatory trouble and probably pus underneath, and in a moment you observe the pus escaping. The amount is nearly half a pint, as far as I can judge. What now shall I do as to the appendix? I shall search for it with great care. Inserting my finger, I feel a round, finger-like body, but considerably thicker than the finger, adherent to what I believe to be the head of the colon. It is bound to the colon by recent lymph, and it is separated with as much ease as the two layers of the pleura in a recent pleurisy. I find this to be the appendix, though it is so surrounded by thickened tissue and new formation that it is almost impossible to assert this positively until I have removed the mass. Having loosened it down to the base, I throw a silk ligature around it and cut it off. You will notice that the portion removed is almost all inflammatory tissue surrounding the undistended tube of the appendix. The free end of the appendix is gangrenous, and has completely sloughed away, thus opening the calibre of the appendix and permitting the escape of the contents. The abscess which we have opened is intraperitoneal, and yet shut off from the general peritoneum by the agglutinated neighboring intestines.

I shall wash this out with hot water, insert a drainage-tube, and

partly close the opening. We shall have necessarily considerable suppuration for some days to come, with gradual shrinking of the cavity and its final obliteration. I was not careful, you notice, to disinfect the cut end of the appendix, for the simple reason that the whole abscess is already infected, and it would be needless trouble and waste of time to do so.

One other point in reference to such a case. It emphasizes the need for the physician to call the surgeon in consultation at the very outset of the case, not necessarily for his knife, but for his best judgment. The physician should have the benefit of the surgeon's experience from the very beginning, and the surgeon should know the case "at first hand," instead of through the statements of the physician when he is called at a later period. The surgeon should be familiar with the case, with a view to early and, if need be, instant interference. The longer the delay before the surgeon is called the greater the probability of danger to the patient.

[In five weeks the patient went home well, after a prolonged but uncomplicated convalescence.]

PERINEPHRITIC ABSCESS; OPERATION; RECOVERY.

Case III.—This third case is an example of a comparatively rare form of abscess, and yet, curiously enough, about three years ago I went straight from one house to another and operated on two such abscesses, both patients, I am glad to say, recovering without a drawback. The case is one of perinephritic abscess in a Russian, aged thirty-three. He comes of a tubercular family on his mother's side, but this is the only taint we can discover. He was never sick until the 1st of January of this year, when he was suddenly attacked with great pain in the right lumbar region without apparent cause. This was attended at first with some jaundice, and his temperature was very high. He had all the ordinary phenomena of high fever, but no especial symptom was noted. He was admitted to the hospital on the 7th of February, suffering with great pain in the right loin, marked tenderness, slight fulness, and rather extensive dulness, far more so than would accompany a normal kidney. There was no solid tumor; palpation by bimanual examination simply gave a sense of increased resistance without fluctuation. The skin was not discolored. He was not able to lie on the right side. His morning temperature was 102.3°, but ran up at night to 105°. Physical examination of the viscera, both of the chest and of the abdomen, showed nothing abnormal. The specific gravity of the urine was 1022; no sugar, no

albumin, no pus, no peptones. The diagnosis, you see, is one made by exclusion. There is an acute inflammatory process, as shown by the swelling, pain, tenderness, and induration. There is probably accumulation of fluid, as shown by the dulness and the absence of a solid tumor, and by the increased resistance. It is certainly not pyelitis nor any other affection inside the kidney, for the urine is normal, and there is no increased frequency of urination. His high temperature shows the septic character of the process. I propose, therefore, to dissect down to the abscess, evacuate, scrape, flush, and drain.

The incision I make is an oblique one in the loin, precisely such as you have seen me make to reach a floating kidney. When I reach the muscular wall I find considerable matted tissue,—another evidence, just as in the last case, of the inflammatory process going on at a deeper level. There is no œdema on the surface, but it does not surprise me to find a deeper œdema in the layers of connective tissue between the muscles of the belly-wall. I have now reached, you observe, the lumbar fascia, which looks yellowish, is elastic, and evidently tense; and on my making a slight cut in it you see the pus welling out in large quantities. After making an opening the whole length of my incision, I pass my fingers into the cavity, and I can pass down behind the kidney, in front of the muscles of the abdominal wall, well into the right iliac fossa, and again far up towards the liver. The cavity has held undoubtedly from one to two pints of pus, and, as you can easily understand, for such a cavity to contract and heal will take considerable time.

With a sharp spoon I scrape away the flaky, granular tissue which lines the cavity. There is not much bleeding, and not a vessel has required a ligature. The slight oozing is quickly checked by thorough flushing with hot water, which answers a double purpose as a hæmostatic and a cleansing agent. I then insert two large rubber drainage-tubes, one passing up and the other down, at the ends of the incision, and hold them in place by stitches through the skin. A large sublimate dressing completes the case.

[At the end of a week the patient's temperature, which had been kept up by the septic process, suddenly dropped to 96°, without apparent cause and without apparent ill effect, but after two days it rose to normal and fluctuated a little above it for some time afterwards. In three weeks he went home, with a simple linear wound, not quite healed, but with the cavity entirely obliterated.]

FRACTURE OF CERVICAL VERTEBRÆ.

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY ROSWELL PARK, M.D.,

Professor of Surgery in the Medical Department, University of Buffalo.

THE first patient this morning is a boy of eleven, whom I first saw a moment ago. On the 9th of August he got under a horse attached to a wagon, and was hurt, nobody knows exactly how, about the upper part of the trunk and the neck. He was unconscious for a number of hours, and his parents think that he just escaped with his life. When he recovered consciousness, the peculiar condition of his arms to which your attention will be called was noticed. He stands now with his abdomen far forward; his mother says that he used to stand straight like other boys. The lower angles of his scapulæ project a good deal, as if he were sprouting wings, and there is atrophy of the muscles above the spines of the scapulæ. Over the cervical spine there is a swelling. This was not noticed for some days after the injury, for he had a hemorrhage from the left lung after the accident, and his head could not be lifted from the pillow. As soon as he was able to be raised, this peculiar alteration of the contour of the neck was noticed. It is now very much smaller than it was. Without attracting his attention by asking if it hurt, I have handled the swelling without eliciting any expression of pain, and on pressing downward on his head and having him jar himself by rising on the toes and coming down suddenly on the heels, I find that there is no sensitiveness of the vertebræ, as there would be if there were some acute inflammation. On bending his head backward, pain is produced. He cannot raise his arms except to an angle of forty-five degrees from the body. He cannot extend the wrists, and there is almost complete loss of power in the hands. There is some contracture of the stronger flexor muscles of the arm. On inquiry, I learn that his arms have not been straight since he was hurt, and that the dropping of his hands was noticed as soon as he recovered consciousness, a few hours after the injury. This time is too short to

allow us to account for the condition on the theory of inflammation in the spinal cord or nerves. Here is certainly an alteration in the contour of the cervical spine, and the case lacks the symptoms of acute inflammation, while there is no present suffering nor evidence of general paralysis. The boy, as you see, stoops over to pick up an object from the floor, and could seize it if it were not that he has lost prehensile power. How can we account for the condition here present?

My diagnosis is that something happened either at or immediately following the moment of injury, from the effects of which the boy is still suffering, and that this something, so far as the spinal cord and nerves are concerned, was a hemorrhage. If it were in the substance of the cord there would be greater paralysis. He has, you observe, motion in the shoulders and arms but not in the forearms and hands. The hemorrhage was probably between the dura mater and the bone, or possibly between the dura and the pia mater. The lesion, as far as the bones of the neck are concerned, is probably a partial dislocation and fracture of one or more vertebræ, since a dislocation without fracture, I should think, would cause more paralysis than we have here. The tumor which is now felt in the neck, I presume, is due to the throwing out of callus. The boy has lost the flexibility of the cervical spine on account of this mass of callus, and he raises the head by bending the dorsal and lumbar spine. This accounts for the marked protrusion of the abdomen, to which I called your attention when he first entered the room.

I suppose that what the family care for more than for my diagnosis is to know what prospect there is for the improvement or entire relief of this condition, and what treatment is to be instituted. This condition has now existed for seven weeks, and any nerve which is not permitted to accomplish its function for such a length of time, even without losing its integrity, would lose its sensitiveness, and its power to transmit motor impulses would be impaired. I presume there is still some blood-clot pressing on the nerve-trunk, and the absorption of that clot is desirable. About the only means of accomplishing absorption is to resort to alteratives, especially potassium iodide and mercurials. We may further use cauterization or other forms of counter-irritation over the spine. We must also avail ourselves of massage and passive motion of the joints and the stimulus of electricity in order to bring back so much of function as may be. I should think the boy would be able to recover at least a useful degree of power in the upper extremities, but I should not like to promise perfect recovery. For the parents, much more specific information will be necessary, so

that they may carry out the manœuvres of massage. The manipulation must begin at the finger-tips, and each digit must be worked at separately; the hand should then be manipulated. At least fifteen minutes should be devoted to each extremity twice a day. The constant current should also be used for five minutes, on account of its action on the nerves. Faradic electricity must also be employed, for its action on the muscles.

By a curious coincidence, I have another and much worse injury of the cervical spine to show you. This is an Italian who was hurt September 23, while working in a sewer, something falling on his head and back. The man is completely paralyzed below the chest, and he has very nearly complete paralysis of the arms, for, though he can partly raise them, he has little control over them. One element in the prognosis of such a case is to note if any change has occurred in the area of anæsthesia and in the power of motion. The former has remained unchanged, and he moves his arms somewhat more than at first, but he still has no power over his lower extremities.

The question arises, Is this a case of dislocation, fracture, hemorrhage, or what? The patient was unconscious immediately following the injury, and when he recovered consciousness he was unable to move. This fact would point to a more sudden pressure on the nerves than that produced by blood-clot due to hemorrhage. We know about where to look for the lesion here, for he has paralysis up to and partially involving the arms, showing that the brachial plexus is partly but not entirely involved. The injury must, therefore, be between the uppermost and lowermost nerves which enter into that plexus. Without a careful electrical study we can hardly determine accurately just what nerves are affected. The injury must be looked for in the lower part of the cervical spine or at about the level of the first or second dorsal vertebra. The injury is too low to permit us to detect any displacement by an examination of the pharynx, although the examination has been made in order to be thorough. I am told that when the patient came in, the orderly and one or two members of the house-staff thought that they discovered crepitus and some displacement. That was four days ago. I find now one very tender spot behind the fifth or sixth cervical vertebra, and this is the point at which the crepitus was found. I do not get crepitus now, and I should hardly expect it so late with such effort as I should consider it proper and humane to make. When the patient entered, his bowels had not moved, and his bladder was distended almost to bursting. These conditions were, of course, relieved.

You will notice that we have on the patient's head an apparatus such as is used for vertical extension in connection with the application of plaster jackets. In this case the traction is horizontal by means of a weight and pulley. Theoretically, he ought to bear a weight of many pounds; practically, we find that he winces if more than three pounds be used. Theoretically, such cases as this ought to be operated upon; practically, the results of operation are disappointing and the operation itself is hazardous. It may be that in this particular case so little damage has been done to the cord that it is capable of spontaneous repair. Under the circumstances, therefore, I do not feel justified in operating immediately, but I shall carefully watch the further progress of the patient, and decide for or against operation as his symptoms shall dictate.

Clinic, three days later.—I call your attention again to this case of supposed fracture of the cervical spine, and I shall now do the operation before you whose propriety I canvassed at the preceding clinic, and about which I then came to no conclusion. Since you last saw the patient, his condition has in no wise improved. In fact, his temperature has risen, his paralysis seems more complete, and it is very evident that he cannot live long unless some relief is afforded him by surgical measures. In other words, I feel that the case will surely prove fatal if left alone, and will probably prove so if operated upon; yet I feel it my duty to give the patient the slight chance which operative measures offer. For this purpose he has been fortified by the usual "fortification mixture" which I like to use before operations, and which consists of the following combination:

℞ Acid. nitro-hydrochlor., 2;
Liq. arsenici chlor., 8;
Tinct. digitalis,
Tinct. ferri chlor., āā 30;
Tinct. nucis vom. ad 100.

Of this the dose is from thirty to fifty drops after each meal. Besides this, the cervical and occipital regions have been shaved, and there has been applied to them for twenty-four hours a green-soap poultice.

Now, the patient being anæsthetized, he is turned upon his face, care being exercised that in this position he suffers no obstruction to respiration from the pillow upon which he lies. A straight incision is made from the second to the seventh cervical spinous process. The next step of the operation consists in dissecting off from the posterior aspect of the arches of the vertebræ all the soft parts attached thereto.

I now find that the spinous process of the fifth cervical vertebra is broken loose; and undoubtedly it was this that imparted the sensation of crepitus which was detected when the patient first entered the hospital. Having now exposed the arches, as intended, I find in them no solution of continuity nor any sign either of fracture or of displacement. This makes it very doubtful whether, upon cutting them away, I shall discover any abnormality at all, so far as the bony parts are concerned. With the sharp-bladed forceps I now cut through the arches of the fourth and fifth vertebræ on each side, and sever the intraspinous ligament and the ligamentum nuchæ, above and below, so that the posterior wall of the spinal canal can thus be removed *in toto*. Beneath now lies exposed the dura mater, which appears perfectly normal, and in or through which I discover no sign of displacement or injury. I next pass a probe for an inch upward and downward inside the spinal canal, in order to detect, if possible, any prominence or obstruction which might imply dislocation, but I find none. Next I cut away the posterior arch of the sixth vertebra, and repeat the examination with the probe, with the same negative result. Consequently I am forced to conclude that there is no narrowing nor distortion of the spinal canal. But the symptoms of this case point unerringly to disturbance in or about the spinal cord, and from this fact, and our present opportunities for observation, one must judge that the trouble is purely intraspinous. The only question remaining to be decided here is whether to open the dura or not. So far as previous experience enables me to judge, there is about the dura in this particular instance no appearance indicating undue tension or the presence of blood or pus. No pulsation of the cord was seen when I first exposed the dura mater, nor has any appeared since. It would seem that nothing is to be gained by opening the dura. Moreover, the patient's general condition makes me desirous to close the wound as soon as possible. Consequently, the soft parts are restored to their place as accurately as possible, and held there with buried and superficial sutures of silkworm-gut and catgut, and an aseptic dressing is applied.

Clinic, three days later.—You will be interested to learn the fate of the case of spinal injury operated upon at the last clinic. The patient rallied well from the operation, and that evening was no worse than previously,—though, I must also say, no better. The following morning, however, the paralysis seemed to have extended, and soon after he became comatose, and during the following night died. Upon autopsy, it was discovered that no fracture nor dislocation existed in the spinal column, and that at no point was there an effusion of blood between

the cord itself and its bony canal, but that opposite the sixth cervical vertebra there was a point of softening, central and acute, with accompanying and surrounding degeneration, and to this were due both his symptoms and his death. This softening must be accounted for by some minute internal hemorrhage caused by laceration of the substance of the cord, corresponding to the condition known as contusion of the brain. It is well known that acute degeneration often follows such injuries to the cord.

Reviewing for a moment the operative features of his case: of course, if one had known exactly the internal condition, operation would not have been performed. We had in this instance, however, to do with an Italian who was not brought to the hospital until two or three days after the injury, who spoke no English, and from whom, even by means of an interpreter, we could gather very little information, and whose friends furnished absolutely no information of any value. Could we have learned accurately with regard to his condition immediately after the injury, a clearer diagnosis might perhaps have been made. Under the circumstances, it would appear that the patient was given every chance which conservative or radical surgery could afford, with the information at hand.

PIGMENTED HAIRY MOLE OF FACE; COLLES'S FRACTURE; SPINA BIFIDA; TUMOR OF THE SPLEEN.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL.

BY ROBERT ABBE, M.D.

GENTLEMEN,—Our first patient is a boy with a large, hairy, pigmented mole of the face. (See Fig. 1.) The skin is hypertrophied; it is wart-like in parts, and the growth covers more than half of the nose, reaching nearly to the inner canthus, and extending two and a half inches out upon the cheek. It may be removed by various methods,—by escharotics, by the actual cautery, or by incision. You may shave it, but this is likely to be followed by a reproduction of the trouble, as well as by a cicatrix. The actual cautery causes probably more scarring than any other method. The use of caustics is followed by a variable amount of scarring, depending upon the particular caustic employed. You may use Vienna paste, arsenical paste, terchloride of antimony, nitric acid, or a paste of sulphuric acid and charcoal; but they all give rise to the formation of more of a cicatrix than is desirable. A recently-introduced caustic known as sodium ethylate is found to be efficient in the removal of the growth and at the same time to leave a soft cicatrix. This new caustic is simply a solution of metallic sodium in absolute alcohol, and is prepared by successive saturation of the alcohol with metallic sodium at different temperatures until a paste is obtained. Metallic sodium, as is well known, has a decided affinity for water, and its alcoholic solution, or the sodium ethylate, possesses the same property, although to a somewhat less degree. When applied to the tissues of the body, it quickly dehydrates them, and forms a pellicle on the surface. At the same time, the epidermis is destroyed and the hair-follicles are penetrated. After two or three applications at intervals of about ten days, there is usually ulceration of the pigmented area, and finally a soft and non-contractile

eschar is left. Before applying treatment to this case, the surface has been well shaved and cleaned, and then dried very thoroughly. The application is best made with a camel's-hair brush, beginning at the margin of the mole. Some apply it with a glass rod, but with this one cannot control its application so thoroughly. This application, so far, has not caused this boy any pain. The method is not applicable for the destruction of hair upon the healthy skin, as it does cause some scarring.

Small moles upon the body or face the size of the finger-nail can often be removed by an elliptical incision, which is preferable to all other methods if the growth be not too large. The edges are to be united by many very fine sutures, and in the course of two or three years all trace of it will have gone.

A CASE OF COLLES'S FRACTURE.

A glance at the deformity which this woman presents tells you that she has a fracture of the lower end of the radius, known as Colles's fracture. Good apposition of the fragments has already been secured, but there is still a little fulness low down upon the wrist, indicative of slight overlapping. Usually no crepitus is elicited in these cases, and at the time of the occurrence of the fracture there is a slight rotary displacement of the fragments: the distal portion is displaced outward, backward, and rotated. In replacing it, the forearm above the fracture is grasped by the operator's fingers and palm, the end of his thumb pressing the lower fragment into position during extreme extension. The operator's other hand grasps the patient's hand, and makes traction, extension, and rotation. Thus there are three motions executed in setting a fracture of this description, although they are performed so quickly as to appear almost as one. These are—(1) depress the distal fragment towards the ulnar side, (2) depress it towards its palmar position, and (3) rotate it slightly.

The case is being treated by the usual straight anterior and posterior splints held in position by strips of rubber adhesive plaster.

SPINA BIFIDA.

You have already seen this little four-months-old infant subjected to the operation of aspiration and drainage for a spina bifida. Since the last aspiration it has not done well; there has been an elevation of the temperature, which at present is 103°. There have been no head-symptoms, but the little one has taken but little food. The tumor evidently includes some of the nerves supplying the lower extremities,

as there is scarcely any retraction when they are pricked or pinched. Such a tumor as this is a constant menace to life; for sooner or later it will become tense and ulcerate through, resulting in loss of cerebro-spinal fluid, and death in a few days. Smaller tumors of this nature will often remain stationary for life. We find to-day that there is inflammation of the sac, and that it is considerably more swollen than it was day before yesterday. The cases which have been operated upon while the sac was inflamed, and before suppuration had occurred, have been successful, and hence we are warranted to-day in interfering, notwithstanding that the child is very sick. There is no cause for the elevated temperature other than the inflammation of the sac.

In dealing with cases of spina bifida there are only two methods to be considered,—viz., (1) injection of the sac with the iodine and glycerin solution known as "Morton's fluid," and (2) the removal of the sac. If the sac were opened with the child in the erect position, the cerebro-spinal fluid would drain out, but if this be done while the subject is recumbent, and the tumor placed at a higher level than the head, there will not be a sufficient quantity of fluid lost to injure the child. It is often difficult to obtain sufficient skin to cover the parts, on account of the extensive thinning of the integument. The opening into the spinal canal is usually not larger than the little finger; but, as a rule, the larger the sac the larger this opening. When the sac is cut off at the pedicle the opening in the bone requires to be covered, and then the skin brought together over that. Briefly, the operation for the removal of the sac consists in making an elliptical incision, dissecting under the skin in all directions until the small pedicle is reached at the small opening into the sac of the dura mater. Having opened the sac and examined its contents, the sac can then be tied off.

On attempting the operation in this case, it was found that the whole cauda equina was spread over the sac, so that it was considered inadvisable to proceed further.

TUMOR OF THE SPLEEN.

This next patient is a woman having a huge tumor of the spleen, which I have watched for some months. (See Figs. 2 and 3.)

You see the line of dulness and the edge of the spleen. She gives a history of having had this for nine months. She has had two or three children, and a miscarriage, after which she bled considerably. Since this time a swelling began in the side and has steadily grown to the present size. I first saw her when it was a little smaller than now.

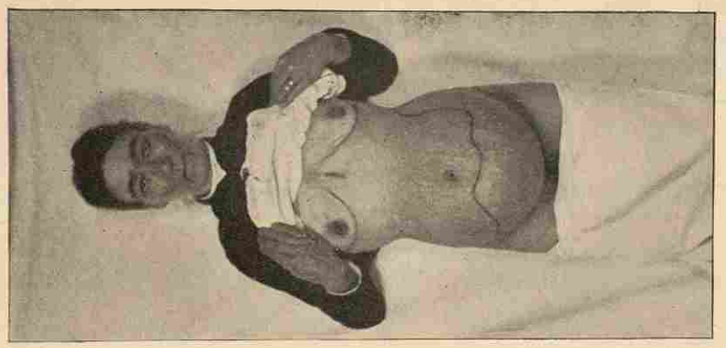

FIG. 3.—Anterior view of the woman with a tumor of the spleen, showing lines of dulness.

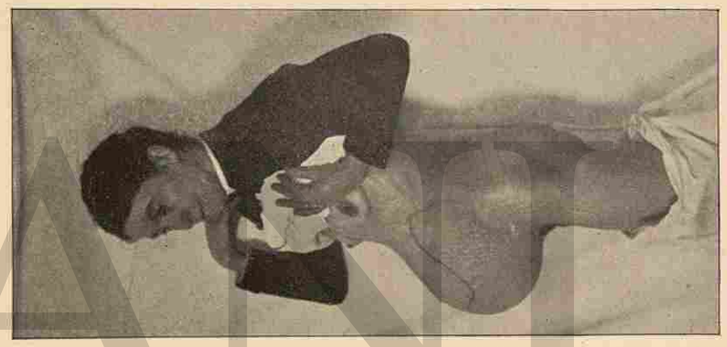

FIG. 2.—Lateral view of site and lines of dulness of a tumor of the spleen.

FIG. 1.—Large pigmented, hairy mole of the face.

Her condition at that time was very bad; she was pallid, feeble, and depressed, and suffered some pain. The tumor had the same kidney shape and relatively the same position in the abdomen. Her pallor suggested leucocythæmia, and a microscopical examination of the blood showed one white corpuscle to thirty red. The position and appearance of the line of dulness indicated that the tumor was not ovarian or malignant, but that it was simply a very much hypertrophied and displaced spleen. It is hard and freely movable in the abdomen. The edge is rounded on one side, and on the other is sharp. No tumor in the abdominal cavity would give this edge except the liver. The tumor is absolutely dull; it is round, smooth on its surface, and has notches on its edge. These are distinctive signs of a displaced and hypertrophied spleen. But you say, May it not be a cystic degeneration of the liver, spleen, kidney, ovary, or uterus? May it not be a tumor of the omentum, or an ovarian tumor, or an ectopic gestation? If a uterine myoma, the tumor would be rotund; if ovarian, it would also be rotund, and would not have this shape, unless it were one composed almost entirely of a cyst which had been tapped. But this tumor has not been tapped. A hydatid tumor is spherical and fluctuating. An hypertrophied kidney might become displaced in this way; but the history would be different. If it were a suppurating kidney, there would be hectic and other constitutional disturbance. Such a tumor as this might be a malignant growth in the abdominal wall, in the omentum, or in the subperitoneal layer, as, for instance, a retro-peritoneal sarcoma. If it were retro-peritoneal, it would be spherical and not movable, and it would not be resonant in the region of the colon, for it would raise the colon. The same would be true if due to a large kidney. This is a most important diagnostic sign. If it were malignant disease of the omentum, it would be spread out broadly over the abdomen, and would feel hard and knotty.

Nothing has this perfectly smooth surface except the spleen or a cyst connected with some of the viscera. But here there have been no disturbances of the intestines, stomach, or other viscera, and therefore by exclusion we have arrived at a diagnosis of enlarged spleen. The spleen is a part of the lymphatic system, and yet in this case there is no lymphatic enlargement elsewhere. When smaller than this one, these splenic enlargements are due to malaria; but this is too large, and the history excludes this origin. A wandering spleen will often be somewhat enlarged, but not so much as this. A hypertrophic change in the spleen occurs in two diseases,—viz., leukæmia and malaria. Examining the blood, we find this leukæmic condition.

Cases have been known to run a course of six weeks and terminate fatally by exhaustion; others last for months or years. When first seen by me, she had been ill only about three months, and she was then leucocythæmic. She has improved a great deal since then, and I think she will recover, but she is troubled with prolapse of the uterus and rectum, due to pressure of the tumor.

In the treatment of this condition, the most satisfactory results have been obtained from the administration of arsenic, quinine, and iron. Arsenic is a favorite remedy for all troubles of the lymphatic system. A free inhalation of oxygen is reported to have cured three such cases. In this instance we used first oxygen, and subsequently "electro-ozone," which is water saturated with ozone through the agency of electricity. It acts very much like peroxide of hydrogen, and is an active oxidizing agent. It was given in tablespoonful doses, three times a day, and has produced a striking effect on this patient in increasing the processes of oxidation. During the summer she has been taking it constantly, and has improved in color, tone, and strength. She is taking now some arsenic. As yet, surgical interference is attended by too great fatality to be thought of. It has been proposed to tie the splenic artery with a view of preventing its growth, and the method seems worthy of consideration. I shall perhaps at some future time propose to make an exploratory incision to determine the exact condition of the surface of the tumor, and also the existence and extent of adhesions. Of course the spleen has been removed many times,—about eighteen times for leucocythæmia, about fifteen times for traumatisms, hydatids, abscesses, or for painful enlargement of the spleen due to malaria. Out of sixty-six cases, twenty-two have recovered. This spleen would probably weigh from twelve to fifteen pounds. There is no reason to think it is malignant, except in the sense that leucocythæmia borders on malignancy.

TWO CASES OF TUMOR OF THE PAROTID REGION.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL, ANN ARBOR, MICHIGAN.

BY CHARLES B. NANCREDE, M.D.,

Professor of Surgery and of Clinical Surgery in the University of Michigan, Surgeon to University College Hospital, etc.

Ladies and Gentlemen,—My first case to-day is a woman, aged forty-nine, with a tumor in the right parotid region, which has been growing for four years. I think that the facial nerve may be involved in the tumor, and if I can dissect it out, so as to prevent hopeless paralysis of that side of the face, I will do so; but I have warned the patient that the facial nerve may be unavoidably cut.

I think it is probably a fibroma or enchondroma much like one we had last year, but I am afraid, from the age of the patient and its rapid increase in size within the past few months, that it may contain sarcomatous elements. I shall not make a bold cut, but shall dissect cautiously, as I might divide the facial nerve with my first incision.

These tumors are deceptive as to their apparent mobility, because the more superficial portions may move upon the deeper, while these are really firmly attached to the surrounding tissues.

Here you see this portion of the tumor, about the size of a hen's egg, passes deeply behind the lower angle of the jaw. I think it is an outlying lobule of the parotid gland; if the whole gland was involved we should have more fixity and the growth would involve the deepest portion of the gland, which is not the case here.

I make my incision through the skin, carefully dissecting down to the capsule of the gland, watching for any nerve-filaments that may cross my line of incision; then, starting from above, I shall try to enucleate the gland from above downward towards its pedicle, which contains the blood-vessels, effecting this by dry dissection as much as possible. Always tie the pedicle of these tumors, as there may be a small arterial branch, which if cut off close to the main trunk will cause such bleed-

Cases have been known to run a course of six weeks and terminate fatally by exhaustion; others last for months or years. When first seen by me, she had been ill only about three months, and she was then leucocythæmic. She has improved a great deal since then, and I think she will recover, but she is troubled with prolapse of the uterus and rectum, due to pressure of the tumor.

In the treatment of this condition, the most satisfactory results have been obtained from the administration of arsenic, quinine, and iron. Arsenic is a favorite remedy for all troubles of the lymphatic system. A free inhalation of oxygen is reported to have cured three such cases. In this instance we used first oxygen, and subsequently "electro-ozone," which is water saturated with ozone through the agency of electricity. It acts very much like peroxide of hydrogen, and is an active oxidizing agent. It was given in tablespoonful doses, three times a day, and has produced a striking effect on this patient in increasing the processes of oxidation. During the summer she has been taking it constantly, and has improved in color, tone, and strength. She is taking now some arsenic. As yet, surgical interference is attended by too great fatality to be thought of. It has been proposed to tie the splenic artery with a view of preventing its growth, and the method seems worthy of consideration. I shall perhaps at some future time propose to make an exploratory incision to determine the exact condition of the surface of the tumor, and also the existence and extent of adhesions. Of course the spleen has been removed many times,—about eighteen times for leucocythæmia, about fifteen times for traumatisms, hydatids, abscesses, or for painful enlargement of the spleen due to malaria. Out of sixty-six cases, twenty-two have recovered. This spleen would probably weigh from twelve to fifteen pounds. There is no reason to think it is malignant, except in the sense that leucocythæmia borders on malignancy.

TWO CASES OF TUMOR OF THE PAROTID REGION.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL, ANN ARBOR, MICHIGAN.

BY CHARLES B. NANCREDE, M.D.,

Professor of Surgery and of Clinical Surgery in the University of Michigan, Surgeon to University College Hospital, etc.

Ladies and Gentlemen,—My first case to-day is a woman, aged forty-nine, with a tumor in the right parotid region, which has been growing for four years. I think that the facial nerve may be involved in the tumor, and if I can dissect it out, so as to prevent hopeless paralysis of that side of the face, I will do so; but I have warned the patient that the facial nerve may be unavoidably cut.

I think it is probably a fibroma or enchondroma much like one we had last year, but I am afraid, from the age of the patient and its rapid increase in size within the past few months, that it may contain sarcomatous elements. I shall not make a bold cut, but shall dissect cautiously, as I might divide the facial nerve with my first incision.

These tumors are deceptive as to their apparent mobility, because the more superficial portions may move upon the deeper, while these are really firmly attached to the surrounding tissues.

Here you see this portion of the tumor, about the size of a hen's egg, passes deeply behind the lower angle of the jaw. I think it is an outlying lobule of the parotid gland; if the whole gland was involved we should have more fixity and the growth would involve the deepest portion of the gland, which is not the case here.

I make my incision through the skin, carefully dissecting down to the capsule of the gland, watching for any nerve-filaments that may cross my line of incision; then, starting from above, I shall try to enucleate the gland from above downward towards its pedicle, which contains the blood-vessels, effecting this by dry dissection as much as possible. Always tie the pedicle of these tumors, as there may be a small arterial branch, which if cut off close to the main trunk will cause such bleed-

ing as to require tying of the main artery; I tied it in this case, but there is a sharp arterial hemorrhage, which comes from directly over the course of the temporal artery, which I shall arrest temporarily by pressure.

The wound is now ready to close, the last vessel having ceased to bleed, yet it has required only eleven minutes; the time is not greater than would have been required with the more free use of the knife, with prolonged search for and tying of bleeding vessels. The question is, Did it originate from the gland-tissue? I have torn the tumor somewhat in taking it away; it was not cut into; I feel some hard substance in it, which may be calcareous matter or even true bone; I do not think from its history of four years that it can be sarcomatous.

Now, as I cut the tumor open, we find something which looks badly for the patient. It is glistening gray in appearance, and cuts like cartilage, while the surrounding tissue looks like a sarcoma, though the history is against this. My assistant, who has been suturing the wound, says she moves the right angle of the mouth: so we have not cut the seventh nerve.

I once spent a long time in dissecting out what I thought to be the facial nerve, and then found it to be shreds of fascia; it is better to be too careful than not enough so. I cannot see the nerve, so I do not think that I have gone through the deep fascia; but as there was a large artery cut, and not secured by an efficient ligature, which may have been the temporal, I shall instruct the attendant to watch the patient closely, and, if there is secondary hemorrhage, place a narrow roller bandage over the course of the bleeding vessel and apply the knotted bandage of the head until the wound can be reopened and the vessel tied.

Dr. Darling has closed the wound by using interrupted sutures, placing a very small rubber drainage-tube in the lower angle, and the outer dressings as usual.

[*One week later.*—The tumor which I removed at the last lecture had its origin in an accessory parotid gland, was sarcomatous as I suspected, and did not include the facial nerve or temporal artery. There was no recurrent hemorrhage from the vessel which was cut. There is now a scar about four inches in length, and no pus formed during the healing process.]

Second Case.—Here is a tumor on the left side of the face in a woman of forty-seven years which has been removed once before. It is much like the one we had last week; it may be that the parotid gland is involved, and therefore the facial nerve and temporal artery.

If I should cut the nerve, I shall at once bring the two ends together by using fine catgut sutures,—that is, if I see the nerve; for you are apt to cut it without noticing it in this dense scar-tissue. A tumor in this situation was removed from this patient seven years ago. From experience we know that tumors of this region are likely to be malignant, and the report of Dr. Gibbes was that the mixed growth that we removed last week was sarcomatous. The same-sized growth as this, if on the arm, would only take a few strokes of the knife to remove, but in this region I shall do as much dry dissecting as possible with the handle of the knife and my fingers. I would again emphasize the importance of avoiding the use of the knife as much as possible in removing tumors of the neck, tying their attachments before cutting, as Professor Van Buren was once compelled to tie the carotid, because another surgeon had cut off a small branch close to the main vessel, instead of twisting, tearing, or tying off the pedicle.

As a tumor has been removed from this region once before, I find this one adherent to the surrounding parts, and I shall have to use the knife more than in the former case. As soon as I begin to enucleate the growth it breaks down, and the various nodules will have to be separately removed. It seems to be cartilaginous, and there is but little bleeding. This case closely resembles one that I operated upon in the clinic the latter part of last year, which I was successful in removing without injury to the nerve, although the tumor passed deeply behind the angle of the jaw and rested on the carotid artery. As the wound is much smaller than the one last week, we will employ a few strands of catgut for drain instead of the rubber tubing, and close and dress as in the former instance.

RESULT OF EXCISION OF THE KNEE-JOINT; INTERCONDYLOID FRACTURE OF THE HUMERUS; HARE-LIP; TONGUE-TIE; FRACTURE OF THE SHAFT OF THE FEMUR.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL, PHILADELPHIA.

BY HENRY R. WHARTON, A.M., M.D.,

Demonstrator of Surgery, University of Pennsylvania; Assistant Surgeon to the Hospital of the University of Pennsylvania; Surgeon to the Children's, Presbyterian, and Methodist Hospitals.

GENTLEMEN,—The first case which I present to you to-day is one of rather unusual interest, on account of the duration of the disease. This man, who is twenty-three years of age, was operated on at the Presbyterian Hospital last summer, having his left knee-joint excised.

His history is as follows. Twelve years ago he was a patient in the Children's Hospital for disease of the knee-joint, and at that time the operation of excision was suggested and refused. The patient left the hospital and had no special treatment afterwards. He was able to attend to his work, but his knee became very much flexed, and he was able to walk only by bearing his weight on the tip of his toes. His knee became swollen and painful, as abscesses were constantly forming and discharging during this time, and he was often laid up from his work. On examination, I detected dead bone, and found that the articular surface of the tibia was carious, and I recommended that he have the joint excised. This he agreed to, and I excised the knee-joint about four months ago, and, as you now see, the wound is solidly healed except a little sinus at the outer portion; he has firm bony union, and walks with very little limp.

I show him to you as one of those cases in which there is a satisfactory result after excision of one of the larger joints. Of course the patient has some shortening after the removal of the articular surfaces of the tibia and femur; but before the operation the patient

had marked contraction of the knee, which caused greater shortening, and he walked on his toes, while now, you notice, he can place his heel on the floor. We do not expect to obtain good joint motion in excisions of the knee-joint, but aim to get bony union, and have a rigid knee, as in this patient's case.

He came to see me to-day to find out what could be done about this little point where pus is discharging. It is very common where you have great infiltration of the tissues, as was the case here at the time of operation, to have a sinus persist for some time, and it is a matter of little consequence, as long as the limb is useful. There is little to do at present except apply soothing dressings to the opening, such as boracic acid ointment or zinc ointment, and allow the patient to put his weight on the limb. If this sinus persists after several months, it might be advisable to curette it carefully, or lay it open and pack it to secure satisfactory repair.

As he walks across the floor, you notice how well he walks; there is some shortening, of course, from the removal of the bone, but this can in a measure be corrected by having the heel of the shoe on the affected limb raised about an inch.

INTERCONDYLOID FRACTURE OF THE HUMERUS.

This little patient met with an accident a week or ten days ago, by falling and injuring the right arm. The patient was brought to the hospital and examined by Dr. Smith, who detected an injury at the right elbow-joint. I am glad to be able to show you this case, as it is a frequent form of injury in children, who seem predisposed to either fracture or dislocation involving this joint.

The patient came in with pain in this region and loss of function. On examination Dr. Smith found no mobility of the inner condyle of the humerus, but widening of the joint as compared with the elbow-joint of the other side; there was no well-marked crepitus. Widening is significant of fracture of the lower end of the humerus. This is probably one of those splitting fractures where the fissure extends between the internal and the external condyle. These fractures are likely to recover with less deformity and more usefulness of the joint than if there was a complete separation of one or the other condyle. In these cases it is important that an examination be made carefully and systematically. There is probably no class of cases that has caused surgeons as much trouble as these injuries at the elbow-joint, for these are the cases which are constantly brought into the courts for damages from the attending surgeon for maltreatment. It is very important on

taking charge of any case with this injury that the surgeon should be extremely careful as to the examination and the prognosis.

We know that in the elbow-joint the condyles of the humerus and the olecranon process of the ulna bear a definite relation to each other in their normal position: so, in examining these cases, see whether the olecranon and the condyles occupy their relative position,—that is, the condyles should be on a line with the point of the olecranon. This is sometimes difficult to make out on the first examination, particularly where there is much swelling; usually there is no difficulty; but if there is any obscurity as to the nature of the injury, I consider it advisable to give an anæsthetic. Finding these three points in a line,—the internal and the external condyle and the olecranon process,—we know that there is no dislocation.

The injury most often confounded with fracture of the condyles of the humerus is posterior dislocation of the bones of the forearm. In this dislocation we have this relation disturbed, have the olecranon process extending behind the line of the condyles. You should first make yourselves certain of the position of these three points. Then examine systematically the head of the radius, and see if it will rotate upon pronating, rotating, and supinating the forearm. You can do this by putting the finger over the head of the radius and making these motions of the forearm. The region of the joint should also be examined by pressure, feeling for the prominent ridge of the ulna, which would be out of line if broken in this region. You should also gently flex and extend the arm, to see that the joint motion can be accomplished, and then compare the elbows with regard to the matter of broadening, which may be an important diagnostic point in these splitting fractures between the condyles. On comparing the breadth of the two elbows, you can make up your mind as to whether it is broader on the injured arm than on the other. If there is no change in the relation of the bones of the forearm with the arm, you can make up your mind as to the presence of fracture, and set aside the question of dislocation. Then, again, you may be able to elicit crepitus in certain cases. Before speaking of the treatment of these cases I will say a word as to the prognosis. In many cases of fracture of the lower extremity of the humerus, in spite of the most careful treatment, you will find that a certain impairment of the joint function occurs. This is often very marked immediately after the splint is taken off, and for several months after the fracture, but as the patient uses the arm the joint motions are apt to improve. It is always important to give the parents or the friends of a child a very guarded prognosis as to

the joint function after an injury of this nature. One cause of the impairment of a joint function, even when the bones are brought into their relative positions properly, is the deposit or the throwing out of a large amount of callus, which may be placed upon the anterior surface of the humerus, so that the coronoid process of the ulna is caught upon the mass of callus, thus preventing complete flexion; or it may be thrown out on the posterior surface of the humerus, and the olecranon is caught on the callus, and the arm cannot be completely extended. This disposition of the callus is outside of the control of the surgeon, and it is the reason why in many cases the results are not so good as we expect. In other varieties of fracture, where one or other of the condyles is separated, it may be impossible to retain the fragment in its normal position, and we have union occur with the fragment in this position, disturbing the mechanical arrangement of the joint.

As to the treatment here, I think that the best method, if there is much swelling and tenderness, is to apply lint saturated with lead-water and laudanum for a few days. At the same time you apply this dressing you can employ one of two varieties of splints. The splint generally used in these cases is what is known as the anterior angular splint. The hand is held in a position with the palm pointing towards the anterior surface of the arm, which is flexed to about a right angle, and the splint is applied and held in position by the turns of a roller bandage. The use of the primary roller in these fractures is a question which should be considered. In adults we generally use it to control the muscular spasm and to limit the swelling; in children, however, unless the swelling is great, I do not think that the use of the primary roller is essential. And I think it is wiser in many cases simply to use lead-water and laudanum for a day or two, and the anterior angular splint; the dressing should be renewed in twenty-four or forty-eight hours after the first dressing, and then at less frequent intervals, the splint being discarded at the end of three or four weeks. Joint motion may be made at the end of three weeks. I do not think that passive motion should be made earlier in these cases, then gentle motion should be made, holding the fragments with one hand while it is being practised.

Another form of splint which is used in these fractures is the internal angular splint, which has been used in this case. This we will reapply and hold in position by a bandage.

In applying these splints you must be careful to see that they are well padded on the side which comes in contact with the arm. At this

stage of the fracture very little support is needed, and we obtain this by securing the splint to the arm by the turns of a roller bandage so as to give fixation to the joint. This splint will be kept on about three weeks, at the end of which time union, I have no doubt, will be quite firm, and we will then make passive motion. I think the result in this case will be quite satisfactory.

HARE-LIP.

This infant, eight months of age, is brought in from the country, some distance from the city, and I have just seen it for the first time. You can see at a glance that it is suffering from hare-lip. This child, you will notice, has what is known as single hare-lip, which is complicated from the fact that there is present a rather prominent intermaxillary bone: so it is not the ordinary form of single hare-lip. It is also complicated with a cleft palate, which is quite a common complication in cases of hare-lip.

There are many varieties of hare-lip,—the single, which this case represents; the double hare-lip, in which there is a double fissure, one occupying either side of the median line; and in these cases, in addition to the fissures, there is liable to be a projection of the intermaxillary bone, and this latter may be so prominent as to necessitate its removal before coaptation of the fissured lips can be made. Then we have various forms of complicated hare-lip, complicated by fissures of the palate, and by the position of the fissure, or by the absence of certain bones.

In this case we have a partial development of the intermaxillary bone, which is quite prominent, and there is a marked cleft in the palate.

The operation for the repair of hare-lip consists in first freshening the edges, then controlling the hemorrhage by the use of ligatures, and then bringing the edges together by hare-lip pins and twisted sutures. In very young children, for this operation, I do not think it advisable to give an anæsthetic; but this child is eight months of age, and is strong enough to struggle considerably and interfere with the manipulations, so that I consider it wise to give ether in this case. In children under six months of age I do not usually give the child an anæsthetic, but prefer to control the patient by pinning a towel around the chest, having him firmly held during the operation. Ether is irritating to the mucous membranes, and I have seen trouble from its use at times in very young children.

The first step in the operation is to separate the lip from the gum

in the region of the fissure so that we can slide the lip freely from one side to the other; this is a very important step in the operation; this separation you notice I make freely. I next introduce a narrow-bladed knife at the upper part of the fissure on each side, and cut directly down and freshen both edges of the fissure. Now having freshened both sides, I have two raw surfaces, which I bring together with two hare-lip pins, one introduced near the lower edge of the fissure, the other just below the nose; a figure-of-eight suture of strong silk is thrown over each of these pins and tied, and you see the edges of the fissure are brought into good apposition. I also introduced a fine silk stitch at the prolabium which holds the mucous membrane in contact, and another between the two pin sutures. The only dressing that we will apply consists of a strip of plaster, simply to support the parts and hold the lips in contact. The pins should be prevented from pressing on the skin, by placing little pieces of plaster under them. In freshening the edges of the fissure the mistake is often made of not removing enough tissue to get two raw surfaces of sufficient size: it is better, to secure union, to remove the tissue freely rather than sparingly. These hare-lip pins will be removed in three days, at which time the union is generally quite firm; the two silk sutures may be allowed to remain for a few days longer, and the wound will generally be solidly healed at the end of a week.

TONGUE-TIE.

This little boy is suffering from an affection known as tongue-tie, which consists in a congenital shortening of the frænum linguæ, causing certain of the tongue-movements to be restricted. This deformity can be relieved by a trifling operation, which is done by placing the broad extremity of a grooved director directly under the tongue, and then taking a pair of scissors and cutting the contracted frænum, and then tearing the cut frænum backward for a little distance with the finger; by holding the tongue upward with the grooved director the frænum is made tense, and it can then be divided without danger of cutting the vessels. There is no special after-treatment required in these cases. If there is any soreness or pain, it is well to use some astringent or antiseptic mouth-wash, as the chlorate of potassium and myrrh or a weak solution of boracic and carbolic acids. Tongue-tie is usually a congenital affection, but I have seen very aggravated cases of this deformity occur in cases of gangrenous stomatitis from adhesion of the under surfaces of the tongue and floor of the mouth consequent upon sloughing of the tissues of the floor of the mouth.

FRACTURE OF THE SHAFT OF THE FEMUR.

The next patient I show you is a young man who met with an injury of the thigh a week or ten days ago. He fell from a ladder and received a fracture of the femur in the middle third. At the time of admission he had only about one-half inch of shortening; which is a very moderate amount of shortening for a fracture of the shaft of the femur. Now, as regards the diagnosis of these injuries, when we are called to cases of this nature it is important to examine them most carefully. We may have fracture occurring at the neck or in the shaft or at the lower extremity of the femur, or the injury may be a dislocation of the head of the femur. It is important to diagnose the injury accurately, as the treatment is very different.

The prominent symptoms of a fracture of the shaft of the femur are shortening, preternatural mobility, and pain, and if we move the limb we get crepitus; we also have loss of function,—that is, the patient cannot lift his leg or bear his weight on the injured leg. Where we have these symptoms we can be almost certain that we have a fracture of the shaft of the femur. We judge somewhat as to the seat of fracture by the age of the patient; in patients after middle life, especially in women over fifty years of age, who have had a fall, we would examine the case not so much to ascertain whether there was a fracture of the shaft of the bone as of the neck; remember that fractures involving the neck of the femur, whether the fracture be inside or outside of the capsule, are frequent in women of fifty years and over. In intracapsular fracture there is less shortening than in fractures of the shaft of the bone. In the former the shortening is usually one-half inch, and at the most one inch; whereas in those of the shaft we may have shortening of three or four inches. Dislocation of the head of the femur is not apt to be confounded with fracture of the shaft of the bone, but the diagnosis of fractures of the neck of the femur is often more difficult; but you can generally separate these two injuries by observing the position of the foot, the amount of shortening, the mobility or rigidity of the joint, and the position which the great trochanter occupies in relation to fixed points of the pelvis. Having made up your mind as to the nature of the injury, that it is a fracture of the shaft of the femur, the next point is to decide upon the treatment. Many of these cases are injured at a distance from their homes or the hospitals where they are going to be treated; and we should make some provision for their safe and comfortable transportation, so that as little damage as possible may be done to the soft parts in connection with the seat of fracture. In the middle third of the thigh we have a large nerve and artery and vein which may be injured by the rough edges of the fractured bone. A patient with a fracture of the shaft of the femur, if the limb is allowed to roll about, may have serious injury done to these blood-vessels, such as laceration followed by hemorrhage, or sometimes the long-continued pressure from the displaced bone has caused a thrombus of the femoral vein, which has been followed by gangrene. Therefore, in a case of fracture of this portion of the femur, see that the patient's limb is put in such a condition that he can be transported without danger to the soft parts or to the bone itself. To accomplish this, you may not have any regular splints that can be applied, but you can fix the fragments by temporary dressings. You can take narrow strips of wood or laths, wrapping them in cotton, straw, or pieces of cloth, and applying them to the anterior, inferior, and outer and inner surfaces of the leg and thigh from the pelvis to the sole of the foot, securing them firmly in position by a bandage or by strips of old muslin. If you have not laths or strips of wood you can use pasteboard boxes, breaking them up and doubling them until you have them stiff enough to support the parts in position, and applying them on the anterior, posterior, and inner and outer surfaces of the thigh, securing them in position by a bandage or strips of cloth. Whatever dressing you apply, the main point, and that which you should remember, is to apply a dressing which will prevent mobility of the bones at the seat of fracture.

I will reapply the dressings in this case, that you may see the dressing we use in this hospital for fractures of the shaft of the femur. The principle of the treatment of fracture of the femur is to put the parts in proper position,—that is, to reduce the deformity and take measures to prevent shortening, and to keep the foot and leg in their proper relation to the thigh until union has taken place. This is accomplished by the use of extension by means of an adhesive plaster extension apparatus, and lateral support by means of bran-bags and splints, a long external splint and a short internal splint. We also require counter-extension, which is furnished by the weight of the patient's body by elevating the foot of the bed, so that he cannot slide down in the bed when extension is applied.

We expect to have in these cases a satisfactory result, with a little shortening. It is exceptional that we have a recovery without any shortening. Union cannot occur in the bone without leaving the bone a little shorter.

The extension apparatus which I apply in this case is made by

taking a strip of adhesive plaster two and a half inches in width, and long enough to extend from the outer to the inner side of the knee, making a loop below the sole of the foot. In the centre of this loop is placed a block, which is perforated for the insertion of a cord to which the weight is attached. The leg should be shaved to remove the hair, if present, as by so doing the plaster gets a better hold on the skin, and can be removed with less pain to the patient.

In the manipulations necessary to dress these fractures the patient is often caused a great deal of pain, and we should have all the dressings on hand before we begin; and we should have an assistant make extension on the foot, which prevents spasm and sudden jarring of the bones at the seat of the fracture. We now warm the plaster extension strips and press them against the outer and inner side of the leg, carrying them up to the knee. I do not see any advantage in carrying them above the knee. The most perfect extension is, I think, obtained by not carrying them too high. It has been urged, in objection, that extension applied below the knee would interfere with the ligaments of the knee-joint, producing their subsequent relaxation. This is, I think, a purely theoretical objection, as I have never seen any injury result from this practice. Three strips of plaster are carried around the leg, one just above the malleoli, one at the middle of the leg, and one just below the knee, and a roller bandage is applied to the foot and leg over the plaster extension apparatus, covering in the malleoli.

In this case you notice that the knee-joint is swollen in the region of the patella. Dr. Smith says it was much worse when it first came into the hospital: this is a quite common complication in fractures of the femur. It need cause no anxiety, as it usually disappears in a few weeks. The patella floats quite freely and can readily be depressed, and when the pressure is removed it comes up again. The effusion is less marked than it was, and will probably entirely disappear in a few days.

Having applied this apparatus for extension, we next make lateral support by the bran-bags, which are held in position by a long external splint and a short internal splint. The splints are wrapped in a splint cloth, which is placed under the limb, and the gutter between the splints is filled by the ordinary bran-bags, and the splints and bran-bags are held in contact with the limb by strips of bandage, which are passed under the limb and secured at three points on the leg and thigh and two points around the patient's body. The foot of the bed is elevated slightly, and a weight of from five to ten pounds is attached to the extending cord, which passes over a pulley fastened to the foot of the bed,

and the dressing is completed. This, I think, is the most satisfactory method of treatment of fracture of the shaft of the femur; and the results are usually very satisfactory. Some surgeons prefer, for lateral support, instead of splints, to use sand-bags. I prefer the bran-bags and splints, from the fact that I think we get by their use less angular deformity at the seat of the fracture. The principle of treatment is exactly the same. In fractures of the neck of the femur we have no tendency to this deformity, and we simply use extension as here applied and sand-bags for lateral support. The principle of treatment in fracture of the femur is extension and counter-extension with lateral support. The extension is usually made by weights of eight to ten pounds, according to the case: some require more, others less. This is kept on for about a month, and the bones are then usually quite firm; we then take off the extension apparatus and splints, and apply a binder's-board or a plaster splint, and at the expiration of six or eight weeks the patient is allowed to get out of bed and go about on crutches.

LYMPHANGITIS; LIPOMA; THE TREATMENT OF PALMAR ABSCESS; THE DIAGNOSIS AND TREATMENT OF ABSCESS OF THE LIVER.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY ARPAD G. GERSTER, M.D.,

Professor of Surgery, New York Polyclinic; Visiting Surgeon to the German and Mount Sinai Hospitals, etc., New York City, New York.

LYMPHANGITIS.

GENTLEMEN,—This child has a fistula in the region of the lachrymal sac, which has been incised. There has also been a purulent discharge from the right ear, and in front of the ear you notice a well-marked but diffuse swelling, which on pressure does not elicit fluctuation. As you know, there is a group of lymphatic glands in front of the ear, which occasionally suppurate, and this seems to be the condition present here. What is to be done? In all glandular morbid conditions we must ascertain the cause of the trouble. We know that the glands are very rarely the primary seat of the malady, and hence whenever you find such a swelling you should always look for something in the vicinity as the primary cause. Thus, if there be a swelling of the glands in the submaxillary region, you should carefully examine the condition of the nose, eyes, ears, throat, and larynx, and in nine-tenths of all such cases you will find the primary cause in one of these situations, and not in the glands. Occasionally serious mistakes will arise from a neglect of this precaution. A middle-aged man, having a conspicuous swelling of the lymphatic glands, was sent to me by his family physician with a diagnosis of cancer, and with a request that it be removed by operation. It was indeed a cancer of the lymphatic glands, but when I heard the man speak I at once made up my mind that this was not the primary affection. I ascertained that he had had aphonia for over a year, and a laryngoscopic examination showed cancer of the larynx. Although his family physician had had him under observation for almost a year,

he had not even suspected the presence of this growth in the larynx. We all occasionally make mistakes, but the most important point in this case was not the mortification of the physician, but the fact that if the malady had been recognized early enough one-half of the larynx might have been removed, or the tumor excised, and the patient's life prolonged, and he would have been spared the disappointment to which he was subjected. Please remember, then, as a working rule, that in every case of glandular enlargement the primary cause must be sought for elsewhere. In the child before us, I not only look at the swelling but I look into the condition of the neighboring organs,—the eyes, the ears, and the throat,—to find the causal indication. A causal indication is that indication for treatment which is directed against the cause producing the malady. If there be otitis media causing this glandular infection, the rational treatment will be to treat the otitis media, and not the glands alone, and when the glandular affection is not of long duration this treatment will be sufficient. Suppose there is a small suppurating wound of the hand, and you find red streaks up the arm to the axilla, indicating extensive lymphangitis, and in the axilla several enlarged and tender lymphatic glands. These glands are threatened with suppuration from the absorption of poison from the small wound. You must not treat the axillary glands, but you must treat the small poisoned wound in the hand which has caused all this trouble, and the moment you evacuate the poisonous material from this wound the lymphangitis and the glandular swelling will begin to subside. This is an every-day illustration of the importance of finding the cause of a glandular affection. To be sure, there are cases where the causal indication might be fulfilled and yet the secondary trouble will not disappear, as, for instance, where actual suppuration or a caseous degeneration has been set up in the gland. Even in these cases the rational surgeon will, if possible, dispose of the cause also. There is nothing more precious in the practice of surgery than a clear knowledge of one's position, and a clear statement to those concerned with us in it, regarding the exact condition presented: the happy relation between the surgeon and his patient is more important even than scientific or financial success. It is no disgrace to say, "I do not know," for it takes some knowledge and understanding to be able to say it. But to come back to our patient. There is an otitis media here, and a threatening suppuration of the lymphatic glands about the ear, which has lasted only a few days. I believe that by improving the condition of the ear, and establishing proper drainage, this glandular swelling will subside; but

usually this condition will continue, acute suppuration will occur, and an incision will be required. The case should be referred to the ear department for treatment. Chronic suppuration, especially if of tubercular origin, will not get well after simple incision, but will leave a sinus for a long time.

LIPOMA.

This man has a movable, lobulated swelling in the right loin, which is elastic but does not give the sense of fluctuation, and it has existed in all probability for a long time, but has not attracted the patient's attention until recently. Although in a rather unusual locality, it is in all probability a lipoma. I do not believe there is any solid connection between the tumor and the base upon which it rests. This is very important; for if the tumor be not freely movable upon the base, be careful about the diagnosis until you have made an exploratory puncture, for such a swelling may be a cold abscess. These abscesses increase in size very slowly, give rise to scarcely any symptoms, and are lobulated, instead of having the rounded appearance of an acute abscess. If we find that this tumor is only a lipoma, we shall propose to him to have it removed, for if left alone it will probably eventually grow to such a size as to cause inconvenience. If we find it is a cold abscess, we must at once look for the cause of this abscess: the most probable cause would be ostitis of one of the ribs. If any bone-disease be present, it will not be sufficient to evacuate the abscess, but we must remove all diseased bone, and leave behind a wound the walls of which are made up of healthy tissues. Under such circumstances the wound will heal promptly, and not only this, but it will remain healed. Of course this does not prevent other portions of the body from being independently infected with tubercular material. I find by puncture that this swelling contains no pus: so the diagnosis of lipoma is sustained, and I shall proceed to remove the tumor.

Lipomata generally occupy those places where we normally find an accumulation of fat, hence principally along the entire dorsum from the nape of the neck to the thighs, on the front of the thighs, on the belly, and on the breasts. The majority of them are well-defined encapsulated growths, the lobules of fat being enclosed in a capsule of dense connective tissue. There is, however, a form of lipoma called diffuse lipoma, which is an accumulation of normal fat under the skin in those localities of the body where there is normally the most fat, and it is difficult to say where the normal fat ends and the lipoma begins. The distinction between circumscribed lipoma and diffuse

lipoma is important only on account of the technical difficulties connected with the removal of the latter class. The probability is that as soon as you have exposed the capsule of such a growth you will find there are a great many septa to be divided, and that they are very much more vascular than in the circumscribed variety; and, more than this, you must draw an arbitrary line between the normal and the abnormal fat. Fortunately, diffuse lipomata are very rare; I have seen only three of them in twenty years of practice. Occasionally lipomata are found in strange places; I have seen one which worked its way between the layers of the neck in the lower cervical triangle down to the œsophagus. You find lipomata quite frequently in the vicinity of hernia; they are called subserous lipomata; they often extend between the abdominal walls, and are very difficult to excise. All these, however, are exceptional.

We have now introduced some cocaine solution subcutaneously over the site of the tumor, and, as the parts have already become anæsthetic, I make a free incision and dissect down to the tumor. There is no difficulty in dissecting out the tumor, and, having secured the bleeding vessels with catgut, the wound is closed with several interrupted catgut sutures, the lower angle being left patulous for drainage. Powdered iodoform, "protective," iodoform gauze, a compress of sublimate gauze, and a bandage complete the dressing, which will be changed after about two days. When these tumors are small, local anæsthesia will be sufficient; but when they are large, general anæsthesia must be employed, and, in fact, it is much more comfortable for all concerned to employ general anæsthesia, as the dissecting out of the deep adhesions is usually painful even though cocaine has been injected.

Regarding the prognosis of operations for lipomata, I would say that they pursue a uniformly favorable course at the present day; but if you will look back in the older text-books you will find it stated that operations for lipomata are peculiarly liable to be followed by suppuration and erysipelas. This was explained on the ground that fat was poorly nourished with vessels; but we know now that this is not the true explanation, and that these accidents were due to uncleanliness in operating.

PALMAR ABSCESS.

The next case is one which very frequently comes under the notice of the general practitioner: it belongs to a class of affections which probably make up the bulk of all surgery. The major part of surgery

does not consist in laparotomies, extirpations of goitres, gastro-enterostomies, and similar brilliant operations, but of much more modest work, which, however, is none the less important. A correct recognition of the condition will enable you to attack it and adopt a strictly conservative course. The word "conserve" means "to maintain in a normal condition,"—this is true conservation; but with some men the meaning of this term has been twisted out of its true significance, and they have told us that to be conservative means to be a coward, a temporizer, a man who is undecided and does not know what to do, and hence does not do anything. I protest against this construction of the term. A surgeon who is dealing with a destructive suppuration of the palm of the hand, and who, recognizing the true condition, takes a knife and evacuates even a few drops of pus, is a truly conservative surgeon, for he sacrifices nothing; whereas the false conservative waits until it is necessary perhaps to amputate a limb. The proper employment of the knife at the right time and in the right place is a properly conservative step.

The dorsum of this man's hand is œdematous, while a superficial examination of the palm shows but little. Glossiness and pallor with infiltration constitute an early indication of suppuration in this region. The pallor is due to the tension on the tissues, which keeps the capillaries empty. Thus, in an acute osteomyelitis of the thigh, in an early stage, an examination of the affected limb shows it to be swollen, glossy, and very pale, and it pits on pressure, but later on, when the pus has perforated the periosteum and has found its way underneath the integument, the skin flushes up: hence a careful observer should not be deceived by the presence of pallor instead of redness. The glossiness and pallor of the hand indicate a deep-seated inflammation, and the dorsum will be found also very much swollen, and sometimes red. It is on this account that young surgeons are sometimes deceived into making incisions into the dorsum instead of into the palm of the hand. Inflammations involving the palmar aspect of the hand are of peculiar significance, because they put in jeopardy the usefulness of one of the most wonderful and most useful parts of the human body, and they occur most frequently in those who most need the hands,—namely, among laboring men. It is from the little punctures and abrasions which they suffer in their work that these inflammations arise. If there be a deep wound and a profuse hemorrhage, unpleasant consequences are not common, and popular judgment has expressed this by saying that "a wound which bleeds well heals well." The reason is that such a wound terrifies the patient, and he will leave it alone, and

also that such a wound is thoroughly cleansed by this very outflow of blood, so that if it be left alone it will usually heal readily. But how different is it with these slight wounds and punctures! A little serum exudes from the broken surface, and any dirt which may have been deposited in the bottom of the wound is covered over by a crust of dried serum, and, as there is no great discomfort from the wound, the patient continues at his work, and the movements of the muscles help to drive the infection farther into the lymphatics, until finally there is a deep-seated and extensive inflammation. Now, what is to be done for injuries of this kind? In the first place, you must always thoroughly cleanse such a puncture with soap and water, or with vinegar, and then cover up the part to insure its rest. Later on, when inflammation has developed, and there is pus formed underneath an unyielding fascia where the chances of spontaneous perforation and escape outward are very slight, a certain tension is produced, and as the fascia opposes its action it burrows and breaks through into the sheaths of the flexor tendons, and if still unrelieved the suppuration may extend to a bone, causing necrosis, or into a joint, causing suppuration there. Now, what is the rational treatment? Is it plunging the hand into warm water, or putting on a poultice, or painting the surface with iodine, or using a piece of plaster? No: there is a septic collection, and it is bound to make its escape, and the most rational treatment is to let it out, as this will relieve the enormous tension of the parts whose vitality is not yet destroyed. Such an incision, if made early enough, will conserve the life of the tendons, of the phalanges, and of all the organs which are in imminent danger of being destroyed. But you may say to me, "This is all very well for a surgeon and a specialist, but it is a different matter with the general practitioner." While admitting that the general practitioner has greater obstacles in this direction to contend with, it does not follow that an effort should not be made to overcome these obstacles. There is a rule in surgery, laid down by the oldest authorities, "wherever there is pus, your duty is to evacuate it," and there is no region in the human body where there is an exception to this rule. I am not speaking now of cold abscesses, but of hot, acute abscesses. The fact that it is especially difficult to evacuate the pus in certain regions does not relieve us from this duty. By all means do not apply a poultice and endeavor to get out of the difficulty by this "*pons asinorum*." Let me show you by a rough diagram on the blackboard how and where you may safely make an incision deep into the palm. Looking at your own palms, you will see the lines corresponding in a general way to a capital M, the upper left-

hand stroke of the M being slightly curved and directed towards the thumb. Now, it is principally the superficial palmar arch which concerns us in connection with such deep incisions into the hand. You can make a free incision from the top of the thumb into the ball of the thumb as far as the up-stroke of the M; you can make a free incision along the entire index finger down to the M, and the same on the middle and fourth fingers, and along the little finger from its tip down to the wrist-joint. You can also make incisions from the right-hand stroke of the M down to the wrist-joint. The only portion you must not injure is a little quadrangular space, the middle of which space lies between the first and second down-strokes of the M. Here you must not cut freely, but this does not mean that you cannot evacuate pus. You can do this by making an incision below and another above, and then working your way cautiously with dressing-forceps. The diagram shows you, then, that there is hardly any spot in the hand that you cannot open freely, and even if you must run the risk of cutting the palmar arch you would better do this, if necessary, provided you open the abscess as well. If you do cut the palmar arch, throw an elastic ligature around the limb to check the bleeding temporarily, retract the edges of the wound, and hunt for the bleeding points and ligate them.

It is stated popularly that a felon on the little finger or the thumb is a great deal worse than one on the other fingers, and this is true; it is a curious fact, which I can confirm. Why is this so? Because the inflammation, if unchecked, is apt to run up the forearm and arm and endanger the whole limb. Farmers, cattle-men, sailors, and ship-captains all know this, for they have to cut open their own felons. If such men recognize this matter and act promptly, certainly we physicians ought to recognize it also, and should ascertain the reason. As you know, the deep and superficial flexor tendons run in sheaths, and it is the peculiar configuration of these sheaths which explains this well-known fact. Let me show you in another diagram the general arrangement of the sheaths of the tendons of the fingers. The sheaths of the tendons from the first, second, and third fingers are closed sacs, extending from the first phalanx down into the ball of each of these fingers, where the sac ends blindly. The tendon is attached to the sides of the sheath, and for about one-fourth to one-half an inch nearer the palm the tendons pass through loose connective tissue before they enter their respective sheaths again. Now, in the thumb there is no such blind sac, the tendon-sheath extending into the palm. This is also the common arrangement found in the little finger. Now you

can see the reason for inflammations of the thumb and little finger extending upward. Corresponding to the endings of the blind sheaths of the three middle fingers there is a weakening of the palmar fascia at the ball of each finger, and through this weak spot spontaneous perforation outward will often occur. In the case of the thumb and little finger the inflammation can travel unchecked to the palm, and thence up into the arm. Hence a free incision to liberate the pent-up poison and to relieve the enormous tension which threatens the death of all the tissues should be employed earlier here than elsewhere.

Now, having made this free incision, how shall you treat the wound? In these days of antiseptic surgery, you are often told to put on "an antiseptic dressing;" but the principles governing our selection of dressings have a rational basis. I divide all dressings into dry dressings and moist dressings, and it makes no difference what chemical is in the dressing. If you have to deal with an aseptic wound, there being nothing poisonous in the wound, the discharges are clean and consist simply of clean blood and serum, which, even if retained, will be absorbed. This escaping blood will form a crust and seal up the wound. But in the case of a suppurating wound, one in which there is apt to be poison for several days after you have made the incision, because there are sloughs which will not become detached at once, you must avoid inspissation and the formation of a crust, and hence you must employ a *moist* dressing. A moist dressing is one which is applied moist, and which is covered with oil-silk to prevent evaporation, and, as a result, the discharges will be diffused through the entire dressing, the mouth of the incision will not be clogged up by crusts, the moisture in the dressing will assume the temperature of the body, the circulation of the parts will be stimulated, and it will act very much as a poultice, but in a much more cleanly fashion, and if you use some antiseptic fluid to moisten the dressing the discharges will be kept sweet and clean. Hence make your incision adequate, pack the wound with a strip of moist gauze, over this place a pad of moist gauze large enough to catch the discharges for twenty-four hours, and over this a piece of rubber tissue to retain the moisture, and a bandage to confine the dressing. Such a dressing is also under your control, and you will not be asked to be responsible for the meddlesomeness of the patient. It is a very pleasant dressing, and a cleanly one, and, as you know, it should be changed only once or twice in the twenty-four hours. The moment the discharge becomes serous and scanty and granulations spring up, the time has come to change from a wet to a dry dressing. If you still persist in using the wet dressing, you will have exuberant

granulations, or "proud flesh." Under a dry dressing the wound heals kindly and quickly.

Now let me say a word about the cases which are seen very early,—those cases in which the best laurels may be won. How is the small collection of pus to be found? There is no redness, no fluctuation, and the pain is diffused. Imagine the focus to be in that little spot under which is the palmar arch. Assuring your patient that you will not hurt him, and asking him not to look at what you are doing, you proceed to touch various points delicately with the end of a probe, beginning at the periphery of this area, and moving in a spiral course until you touch the point where the patient insists he feels the most pain. Having determined this spot, plunge a small tenotomy knife directly downward until the pus wells up alongside of the knife, and then, as you withdraw the knife, widen your incision. Now introduce a small bit of gauze and apply a moist dressing, and the patient will be able to resume work within two or three days.

DIAGNOSIS AND TREATMENT OF ABSCESS OF THE LIVER.

The next case is that of a young man who has been suffering from violent pain in the region of the liver, accompanied by a marked increase in the area of hepatic dulness. The liver is freely movable during respiration, and hence does not appear to be adherent to the abdominal wall. He has continuous fever, which is aggravated towards evening. As his general condition is growing steadily worse, I deem it proper to interfere. Nature, as a rule, is a bad surgeon in the treatment of acute suppurative processes. You know it is a popular belief, and even some medical men still hold the opinion, that external applications will attract pus towards the surface of the body; but it is an entirely erroneous idea, for the pus extends along the lines of least resistance, and this is often along the lymphatics in planes parallel to the surface, instead of towards the surface. Hence the old surgical rule, to evacuate the pus as soon as detected, is the one to follow, and the importance of this rule increases closely in proportion to the depth of the suppurative process. Assuming that in this case the suppurative process is in the parenchyma of the liver, can any one say that the pus will find its way to the abdominal surface of the liver and through the abdominal wall? It is just as likely to break through the diaphragm into the pleural cavity,—a very dangerous condition. Again, the perforation might take place into the general peritoneal cavity, with even more serious consequences. We know that a large number of these abscesses of the liver are confined within the

capsule of the liver, and assume very large proportions before perforation takes place, and that adhesions are usually formed before evacuation of the pus occurs.

In considering this subject systematically, I shall speak first of methods of diagnosis, and secondly of methods of treatment. In making your diagnosis of this condition you must make a careful physical examination, and, more than this, you must have direct evidence of the presence of pus before you undertake to make an incision. This information is obtained by exploratory puncture. If your puncture is negative, then you will consider that the fever and other constitutional symptoms which have been present might be due to malignant disease instead of to an abscess, and you will not make an incision. Again, there may be chills and elevation of temperature from retention of bile, and if you can find even a few drops of pus your hands are greatly strengthened. It is important that you should make your incision, if it be an abscess, at an early stage, otherwise your patient is likely to die of exhaustion, even after the incision. Let me impress upon you, then, the importance of making an early diagnosis of abscess of the liver. Now, what are the means of making the diagnosis? If we have to deal with an intumescence of the liver, and signs indicating firm adhesion between the liver and the abdominal walls, you need have no fear of making an exploratory puncture. The physical examination will show that on deep inspiration and expiration the tumor of the liver does not change its position with reference to the abdominal wall, and I beg you to bear this fact very carefully in mind. Do not plunge in a knife, as you might do in an ordinary abscess elsewhere; this desire to be brilliant is a bad thing, and the modern surgeon foregoes this brilliancy and avoids this plunging of a knife into anything. He works carefully from layer to layer, being guided at every step by the eye. In accordance with this practice, then, you must incise the skin, the fascia, and the abdominal muscles, until you reach the peritoneum, and if there be adhesions you will then observe that all the tissues of the abdominal walls are œdematous and infiltrated, and that serum will ooze from them. This reassures you, and, proceeding cautiously, you will soon be rewarded by finding pus. The wound should be shaped like a funnel, wide at the surface and narrow at its deepest portion. When the pus oozes up through the apex of this wound, you enlarge the opening with a dressing-forceps and establish proper drainage by the insertion of a tube.

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Sometimes the adherent part is under the diaphragm, and only a small portion is accessible. Would the method just described be ad-

visable here? No indeed, because some of the pus might escape into the general peritoneal cavity and cause a fatal peritonitis. Where you find that this condensation of the tissues does not extend over the entire wound, and instead the free surface of the liver is exposed, do not carry your incision any farther; even an exploratory puncture with a fine needle is dangerous. Within the last few years the technique of operating for these abscesses of the liver has been much improved. Open the abdominal cavity sufficiently to enable you to ascertain if there be any adhesions of the liver to the abdominal wall, and their location. If you find these adhesions posteriorly, it will be proper to close up your first incision and evacuate the abscess posteriorly: An adhesion always means that perforation is imminent, and if you should find that the abscess threatened to perforate through the diaphragm, you must adopt some other plan of treatment. Formerly it was customary to irritate the abdominal wall by the actual cautery, tincture of iodine, or vesicants, with the idea of exciting adhesions. These methods have been found both barbarous and inefficient. Chemical caustics, such as caustic potash, were applied daily, particularly by French surgeons, until the adhesions were formed, after which the abscess was evacuated by means of a trocar. A German surgeon, Professor Volkmann, suggested the exposure of the liver by laparotomy, protecting the peritoneum by sponges while the liver is punctured. After the withdrawal of the needle, the puncture is watched for a few moments to make sure that no pus escapes, and if pus is found the wound is packed with gauze for a few days, after which the adhesions are usually sufficiently firm to admit of free incision into the liver abscess. This is a good method, and I have practised it myself. Some surgeons have found, however, that in some cases the irritation of the gauze is not sufficient to excite the necessary amount of adhesive inflammation, even after the packing has remained in the wound for a week. Of course, if the gauze do not excite sufficient inflammation, irritant substances may then be applied to the gauze and sufficient inflammation produced, but by adopting this plan much valuable time is lost. Lawson Tait, an English surgeon, has shown that the liver can be treated just like the intestine in colotomy, or like the stomach in gastrostomy, and that it can be attached to the abdominal wall by sutures. Surgeons were afraid in former days to do this on account of the free bleeding from the liver, for such small but continuous hemorrhages may occur, when there is engorgement of the liver from interference with the portal circulation, as to cause death. While, then, this fear of interfering with the liver is well founded, it

has been found that in these cases of abscess of the liver it is comparatively safe to introduce a circle of stitches around the site of your puncture. If the case be a very urgent one, insert your sutures very closely and tightly, and make your incision at once. There is some risk, of course, in doing this, but the surgeon must determine in each individual case whether it is justifiable to take this additional risk. Ordinarily, however, it will be better to pack the wound with gauze for twenty-four hours, and then make your incision, for by this time firm adhesions will have formed. The incision into the liver is generally done with the thermo-cautery knife, because we do not like to use ordinary cutting instruments on the liver. When the incision is made with the cautery-knife there is not apt to be much bleeding. Again, the moment you have incised the liver and evacuated a large quantity of pus, the whole organ with its engorged vessels collapses, just as occurs in tracheotomy, and the tendency to bleed is proportionately diminished. I propose, then, at the hospital to insert the sutures as described, and make an exploratory puncture before making the incision.

There is a class of hepatic abscesses in which the prognosis is bad, even though you interfere surgically. These abscesses usually accompany chronic intestinal affections, especially ulcerative diseases, such as acute or chronic dysentery, where emboli are carried into the liver, and generally cause a number of separate abscesses. Although many of these abscesses become confluent after a time, others will still remain separate, and will not be evacuated by the one incision. These abscesses are only an indication of a grave constitutional condition,—pyæmia; and though you incise the abscesses in various parts of the body, one after the other, the patient will ultimately die. This fact shows the importance of ascertaining very carefully the previous history of the patient, for upon this must depend your prognosis. If the abscess be due to suppuration around an obstruction of the biliary ducts, as from a calculus, or due to an acute suppurative hepatitis from other causes, the prognosis is favorable. In the patient upon whom I propose to operate there is a history of a number of attacks of hepatic colic, and this leads me to believe that there is only a local trouble, and that the patient will probably be cured. This question of liver-abscess is of more importance in tropical countries, because in these countries they are more common, and unfortunately are more often of the metastatic kind, or those associated with pyæmia. I may say that a bold but cautious treatment of abscesses of the liver, other than the metastatic variety, yields very excellent results. There are, to be sure,

very rare cases of spontaneous cure of hepatic abscesses, just as there are occasional instances of spontaneous cure of empyema, but we know now how many cases of empyema formerly died from this let-alone treatment. So it is with liver-abscesses. I have seen a case of acute liver-abscess due to local cause which perforated the diaphragm and discharged its contents into the pleural cavity, yet the patient recovered perfectly without operation, and remained in excellent health for thirty years afterwards.

INCIPIENT COXITIS; SUBLUXATION OF CLAVICLE IN RICKETS, AND COXALGIA; THE PREPARATION AND APPLICATION OF PLASTER-OF-PARIS JACKETS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY V. P. GIBNEY, M.D.,

Professor of Orthopædic Surgery, New York Polyclinic; Orthopædic Surgeon to the Nursery and Child's Hospital; Surgeon-in-Chief to the Hospital for Ruptured and Crippled.

CASE I.—This child, whose case I present this morning, has had pain at night for two weeks or more. Suppose we look over this case carefully and pronounce on the sickness. In the first place, there is no defect in the gait; the back is perfectly straight. There is apparently no defect in the back or limbs. There are no preputial adhesions: so the child has no genital irritation. The ankle-joint seems perfect; you can flex and extend the hips; you can flex and extend the back and rotate it with perfect ease. Now let us take the left side. Here also we get good movement, and can abduct and rotate. I get my fingers well down into the iliac fossa, but find no abscess there. I can hyperextend each thigh, and the spinal column is quite flexible. There is no fever. The child has pain in the abdomen and in the left lower extremity. The measurements are—right side, fifteen and one-half inches; left side, fifteen and one-half inches: so the limbs are of equal length. The right thigh, four inches from the anterior spine, is ten inches; left thigh, at same point, ten inches: so there is no atrophy of the limb. The mother says the child has had pain for the last two weeks. There is a little more resistance offered on the left side than on the right side; it requires a little more time to get the thigh flexed. Reflex pain at night causes the child to scream during sleep. The characteristic of the night terrors of hip-disease is this,—that you will hear the child shriek, and will go to his bed expecting to find him awake, but he will be sound asleep. When the child first drops asleep after going to bed, the spasms start at once and will wake

very rare cases of spontaneous cure of hepatic abscesses, just as there are occasional instances of spontaneous cure of empyema, but we know now how many cases of empyema formerly died from this let-alone treatment. So it is with liver-abscesses. I have seen a case of acute liver-abscess due to local cause which perforated the diaphragm and discharged its contents into the pleural cavity, yet the patient recovered perfectly without operation, and remained in excellent health for thirty years afterwards.

INCIPIENT COXITIS; SUBLUXATION OF CLAVICLE IN RICKETS, AND COXALGIA; THE PREPARATION AND APPLICATION OF PLASTER-OF-PARIS JACKETS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

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him, and when he wakes he can control the spasm. In Pott's disease of the spine the child does not wake, but will moan in his sleep and toss in the bed. These signs are important in making the diagnosis where you have as few signs as are present in this case. It is next to impossible to have as perfect flexion as this in tubercular osteitis of the hip, or what is known as "hip-disease." He is two years and four months of age. Yet it is possible for a small spot of osteitis to be present in the head of the femur or in the left acetabulum of this child. If he had only this small amount of osteitis, it would be compatible with the symptoms. You can flex his thigh perfectly. Hyperextension on the sound side is good, and is not bad on the left. The ilio-femoral creases are symmetrical. In making the diagnosis we must differentiate between malarial neurosis and incipient osteitis of the hip; there are not enough symptoms for a clean-cut diagnosis. There are some things against hip-disease,—namely, the freedom of flexion and extension. On the other hand, there is a limited rotation, that function not being as good on the left as on the right side; there is no atrophy that would not be present in the beginning of hip-disease: so it is early hip-disease, or neurosis of some kind depending upon some toxic influence,—malaria, perhaps. Children who are malarial have pain about the epigastrium. It is quite common in the masked form of malaria. A child with hip-disease will always halt; from the very first there is a little impediment in the gait: a child that walks across the floor as this one does would show it at once. Then, again, there is no change in the femoral crease. Then there is a resistance to rotation on the left as compared with the right side. In hip-disease the child always cries at night and goes to sleep again easily. The thing to do with this case, I think, would be to put on a snug spica bandage of muslin, and instruct the mother not to let the child walk too much in the next few days, then have the case report to us again on Wednesday. It is a good plan to do something of this kind, for the parents think you are making an effort, and they will be better satisfied.

Case II.—This boy is twelve years old. He tells us that in the middle of January, 1890, he became lame, but previous to that he began to have pain in the hip and knee, which was first noticed in June, 1889. He would complain of pain for a day or so and would go a little lame, but in another day or so he would be quite well again. So his history shows that the disease began with attacks of slight pain and lameness, which would last a few days and disappear, but in January, 1890, he became permanently lame. He can walk pretty well.

Notice how he stoops to pick up anything; he bends the left knee, but not the right; it is not a spinal stoop; the spine is normally flexible, but he keeps the hip stiff. You all can see how he favors that right hip. He flexes the left hip and bends the spine, but keeps the right hip stiff. The mother tells us that he has had pains at night. As I lift him by the waist, you see how freely the spine is flexed. He bends over towards both sides very well, and there does not seem to be any limitation of movement. You see the external end of the clavicle projects a little more than that on the other side; that is a subluxation of the external end of the clavicle. Sometimes these project to a considerable degree.

That reminds me of a case that occurred in this city several years ago. The father of the patient got the Elevated Railroad to pay out twenty-five hundred dollars, convincing the company that the accident occurred in a collision on Forty-Second Street and Third Avenue. The girl got judgment against the company for this sum, and the money was put with a trust company. The girl was nineteen or twenty years of age. She was seen by some of the best surgeons of this city, among them one now dead who has a world-wide reputation. After about a year, the company's lawyers learned that this girl had been to the Hospital for Ruptured and Crippled several hours before the accident had happened. They found that she was there on a certain day about a month before the accident for this very trouble, and that a splint was made for her to hold the external end of the clavicle in place. It was also shown that she had been there on the morning of the accident to have the splint fitted; that after she had left the hospital the accident occurred and she had got into the crowd. She went home somewhat frightened by the commotion, and the parents thought it would be a good way to get some money out of the company. The father was sent to the State prison afterwards, and the girl was let off with a light sentence.

It was formerly thought that subluxations of the clavicle were rare, but rachitic children often present this deformity. I find a number of cases of lateral curvature in rachitic subjects who have this luxation, and I find it aggravated by certain exercises used for the correction of the curvature. Hence I have had to dispense with the exercise altogether. Lately we have a way of curing these that is very good. My attention was first called to it by Dr. Stimson, who had injected alcohol around the articulation, binding the parts with a roller bandage. Two or three injections serve to set up an inflammation around the joint. Several successful cases have been reported.

We have got thus far in the examination of this boy: the spinal column is normal; one hip is affected; while the history shows that he has been lame for over a year, but has been ailing for a longer time than that. We will now measure his limbs and test their functions.

R. A. (which means from the anterior superior spinous process of the ilium to the lower border of the internal malleolus) is 30½ inches; L. A. is 30½ inches.

R. U. (which is the measurement from the umbilicus to the lower border of the internal malleolus) is 33¼ inches; L. U., 33¾ inches.

So we have nearly one-half inch of practical shortening, but no real shortening. Now, if I measure the thigh at a given distance from the anterior superior spinous process, the circumference of the right is thirteen and one-half inches; the left, at the same point, is fourteen and one-half inches: so there is a difference of one inch. The right knee measures eleven and one-quarter and the left eleven and one-quarter inches. The right calf measures ten and one-half and the left calf ten and three-quarters inches, showing a difference of one-fourth inch. Now, suppose I take the left limb, which is the sound one, and see what he can do with it; then we can compare the right limb and the functions of the joints very accurately. If we flex this sound limb, we can bring it up to at least forty-five degrees. If we extend it, it comes down perfectly straight to one hundred and eighty degrees. If the limb be abducted, the outer side of the thigh can be brought over so that it lies on the table. If you rotate it, it rolls quite easily externally and internally. So the functions of this joint are perfect. Now on the diseased side. When we begin flexion the whole pelvis moves; we cannot flex beyond one hundred and thirty-five degrees. Now suppose we try to extend; we cannot extend quite down to one hundred and eighty; the popliteal space does not quite touch the table, while on the healthy side it comes down quite easily. We cannot adduct much to speak of, and we cannot abduct at all. There is no rotation, or but very little. Now, having the patient lie on his face, we will try hyperextension, and you see that I can hyperextend the sound limb, but not the diseased limb. I find some cicatrices here on the hip; they are from the injection of carbolic acid for an abscess, which he tells us was treated in that way. The abscess, as the cicatrices show, was just behind the trochanter major. Now we will test for joint-tenderness. I will make a lever of the femur by placing my hand as a fulcrum at the junction of the upper and middle thirds on the outer side; then, grasping with the other hand the limb at the knee, I abduct, and, as I do so, move the lever, which will at once show if there is any

tenderness in the joint. There is no tenderness as I make the test, and when I crowd my fingers into the iliac fossa there is no tenderness there. There is no sign of any abscess. From this history, and from the examination just made, we are warranted in making the diagnosis of tubercular osteitis of the hip. Although his age, twelve years, is a little beyond the age for the development of the disease, still there are cases that develop in adult life. Cases that develop at this time are usually exceedingly chronic; the onset may, on the other hand, be very acute and we have osteomyelitis.

I proceed to measure him for a hip-splint by taking the circumference of the pelvis just below the anterior superior spinous process. It measures twenty-four inches, five-sixths of which give us the length of the pelvic band,—twenty inches. For the stem, take the sound limb and measure from the anterior superior spinous process to the border of the heel; the length is thirty-three inches here, but make the stem two inches longer, thus allowing for a two-inch-high shoe. The order for a hip-splint, right side, without a rack and pinion, will be a twenty-inch pelvic band and a thirty-five-inch stem. Order at the same time a two-inch-high shoe for the left foot. The shoemaker can make this by the time the splint is completed.

The limb can be pulled down with adhesive straps, and buckled on to the foot-piece of the splint. With such a splint these children can walk about and play, can climb fences, and even, after a fashion, skate. They are, as a rule, free from pain. The splint is worn *night and day*. If an abscess form, it can be opened if it get in the way of the splint, or it can be aspirated and strapped. The main thing is to keep up the traction; keep the joint protected, and keep the patient out of doors.

Case III.—This boy was operated on last Wednesday, a week ago to-morrow, for rectangular deformity of the hip. I divided the femur subcutaneously. His highest temperature, which was low, was 100° the second day. It has been below that point since that day. He had some discomfort the second night, but has had none since. His limb is now down very nearly parallel with its fellow. He will need a small lift on his shoe, probably half an inch or an inch, and he will be able to ride horseback later on.

Case IV.—I had a case this morning of a girl who was well apparently except for this slight deformity. I divided the femur below the trochanter minor. I knew very well that when she got out of the hospital the adduction would increase, and by the time she was fifteen or sixteen (she is ten now) she would have a degree of deformity something

like this [illustrating]. So I thought it best to perform the operation. It is an operation that has not been attended with any bad results, so far as I know. I do not know of a case where the union has failed to take place perfectly after the division of the femur; out of a large number of cases I have never had any trouble. I put on a good compress of sublimate gauze over the little wound, have some one hold the limbs perfectly parallel, lay the child on a hip-rest and a shoulder-rest, put on a plaster-of-Paris bandage from the axilla down to the heel, taking in the foot, reinforcing the plaster in front with a piece of bar-steel, not removing the dressing until four weeks have elapsed.

CASE V.—I took down yesterday a hip in a girl sixteen years of age, who had a deformity of one hundred and twenty degrees in flexion by measurement. I operated on her just four weeks ago to-day and left her in this plaster bandage. When I took it down yesterday I found the union firm. I got movement in the trochanter and shaft perfectly. I used the Polyclinic splint with adhesive strips along the sides, peroneal straps, a pelvic band, and it came just to the bottom of the foot. In this case all I want is to protect this union a little longer, until it gets firm and strong enough to support itself. I use this splint, say, for two months after I take the plaster off.

THE PREPARATION AND APPLICATION OF PLASTER-OF-PARIS JACKETS.

We use plaster-of-Paris jackets in all forms of Pott's disease, and in lateral curvature of the spine where the deformity is very great. In all forms of Pott's disease the plaster-of-Paris jacket is a luxury. I use it here on account of the completeness of the splint, and because you do not have to depend upon instrument-makers for apparatus: all the responsibility of the case will fall upon your own shoulders. The solid plaster-of-Paris jacket should be used in all stages of Pott's disease. The corset should not be used except after a solid jacket has been applied for one or two years. If you recognize the fact that Pott's disease is really a broken back, that you have a serious and important breaking of the segments of the back, just as surely as when you have a fracture of a limb, I think you will have no trouble in properly treating the case. In fracture of the limb you require that the fragments shall be kept in place for five or six weeks, and in Pott's disease (broken back) you require one or two years. Nobody can arrest the progress of tuberculosis of the spine. Koch's lymph has practically failed, so far as this is concerned, so we are left just where we were before its discovery. Surgery at the present day

does not dare to go into the vertebræ, generally speaking. There are some who do it, but, as a rule, it is not practised. Then you have to put on something that will keep the spinal column in the normal position, or the best position you can get it in, and prevent further deformity, or, if deformity has not appeared, prevent it. You ought always to be able to diagnosticate Pott's disease before deformity arises. If I fail to enable you to do that, we shall not have gained what I hoped, so I shall take the next lecture for the consideration of the early diagnosis of Pott's disease, because you cannot over-estimate its importance. You ought to be able when you see a child walk peculiarly to tell whether it has or has not Pott's disease of the spine. If there is no deformity and you can diagnosticate it, so much has been gained. You ought to be able to cure such a case without deformity, and you can do it with a plaster-of-Paris jacket. You ought to make yourself expert in putting on such a bandage. It is one thing to make a jacket and another thing to put it on. You use a swing which can be ordered from an instrument-maker for six dollars and which will be all that you need for putting on plaster of Paris. Fasten this head-gear, made for the purpose, around the neck so that the head rests upon it comfortably (we seldom use the arm-piece now), then draw up the rope and pulley until the toes and heels just rest lightly on the floor, the child meanwhile holding its arms well up over the head. You require a seamless undershirt. For padding, Canton flannel answers very well, cut in strips. If you cannot secure that, common surgeon's felt will answer. Cotton is bad, because it lumps. The next thing you want is the best plaster; and it will pay any man, although he should have only one jacket to apply in the course of his life, to send to the S. S. White Dental Manufacturing Company for the best. They have offices in Chicago, Philadelphia, and New York, and will send you a six-quart can of plaster for seventy-five cents, or twelve quarts for one dollar and a quarter.

Let me illustrate the method by this child who has just been brought in and suspended in the swing. I will apply a plaster-of-Paris bandage from below upward, protecting the bony prominences by carefully padding them under the shirt. The bandages have been first soaked in warm water. The patient should be made perfectly straight. It is a good plan to make the lower and the upper part of the jacket a little extra thick. Keep the bandages perfectly smooth, and overlap the lower turn about half the width of the bandage. Four bandages have been sufficient. We will now have the child carried to this table and laid on its side; the bandage

will be trimmed, and after it has hardened the patient will be ready to go home. Leave the bandages on as long as they will stay; they usually last about two months; but never allow a child with Pott's disease to stand or sit up without good support.

The case of this little boy who comes in next, with the disease high up, will also require a jacket, with a head-spring in addition. The jacket has been put on just as in the first case, except that about three of the outside turns of the bandage were carried over the lower portion of the head-spring to fasten it in position.

The crinoline used for bandages must be sized with starch. If it is not sized with starch it is better to wash it. If sized with glue it will not stay on, no matter how good the plaster of Paris is. You can test it by trying it on somebody's arm, or by tasting it, or by the starch test. Cut it into six-yard pieces, two, two and a half, or three inches wide, according to the size of the patient. Roll or rub the plaster in its meshes the same day you are going to use it, if possible. When you take the plaster out of the can, seal the can carefully, and it will keep indefinitely in good condition. Rub it well into the meshes of the cloth, and then you are ready to use it. Roll in a loose roll the same as any other bandage, and you then have every requisite for a first-class jacket. If you prepare your bandages in this way the jacket will be set by the time you get through putting it on. Trim it under the arms and around the thighs, so that the patient can sit down with comfort.

SYNOVITIS OF THE KNEE-JOINT; PROCTITIS; PRIMARY SYPHILIS—BUBO; VARICOCELE; CYSTIC GOITRE; LIPOMA.

CLINICAL LECTURE DELIVERED AT THE RUSH MEDICAL COLLEGE.

BY JOHN B. HAMILTON, M.D., LL.D.,

Professor of the Principles of Surgery and Clinical Surgery in Rush Medical College, Chicago.

SYNOVITIS OF THE KNEE-JOINT.

GENTLEMEN,—The first patient I show you this afternoon is a lady seventy-four years of age. She has some trouble with the knee-joint, which is considerably swollen. We have an antecedent history of "rheumatism," and she says that there has been a good deal of pain in the joint. The probability is, therefore, that this was originally a case of synovitis, and that we have at present an effusion in the joint of synovial fluid. There is a chance that this condition is due to the presence of pus in the joint; and, in fact, at a hurried examination a day or two since, I presumed we had pus, but on examining the joint more carefully to-day I am sure that it is a synovial effusion. I infer it is synovial because the swelling follows the direct outline of the synovial sac. You remember that this sac passes above the upper border of the patella a distance of an inch and a half to two inches in some cases, and occasionally even farther. In this case the synovial sac is fully distended. There is not pain enough in the limb for us to conclude that we have to deal with pus. If it was a cold abscess, as it is termed, we should have had a pretty violent inflammation in the joint, and there would have been the constitutional signs of pus. As it is now, there is only a history of some pain such as a woman of the age of seventy-four, subject to rheumatic pains, might have in any of the joints.

We will now make an exploratory puncture by inserting a rather coarse hypodermic needle—after passing the point through a flame to disinfect it—directly into the swelling at the side of the patella, and by withdrawing the piston of the syringe, if the fluid is not too thick

will be trimmed, and after it has hardened the patient will be ready to go home. Leave the bandages on as long as they will stay; they usually last about two months; but never allow a child with Pott's disease to stand or sit up without good support.

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to flow through the tube, we will ascertain its character. As I suspected, it is synovial fluid, and not pus. These accumulations in the joint are due to either chronic inflammation or to acute inflammation of the synovial membrane. Sometimes even when the inflammation has been very acute the fluid becomes absorbed, and there is no swelling; at other times the fluid goes on accumulating until the joint-surfaces become stretched; they become infected, as in disseminated tuberculosis, for example, and pus is formed. The rheumatic enlargements of the joints are very apt to have an accumulation of synovial fluid. I think it is always better in such cases to evacuate it and inject either iodine or iodoform emulsion. In this case now under treatment there will be nothing more required than ordinary tapping, injection with iodine water, and allowing the injection to pass out.

As to the method of performing this operation, a trocar is introduced into the most prominent portion of the swelling, about the upper border of the patella, carrying the point of the trocar downward and inward until we have reached the sac; then the fluid is pressed out, and the iodine injected. Frequently these collections of fluid are too thick to flow through the trocar, in which case we are obliged to substitute a larger instrument for the small one commonly employed. There is another point in the puncturing of these synovial collections, and that is the practice of making the skin tight, pulling it up or down or transversely, so that when the fluid is withdrawn the skin falls back again over the slight puncture that has been made, like a valve, and in that way the air is kept out. I have now introduced the instrument into the joint underneath the patella, and shall withdraw the trocar, leaving the canula in place. Instead of the trocar and canula the aspirator is sometimes used, and is especially valuable in cases before the effusion becomes inspissated and too thick to flow through the tube. In this case the sac seems to be multilocular, and the fluid is dark and a little redder than that which is ordinarily seen in synovitis. Excessive synovial fluid is generally of a pea-green color, highly albuminous, in appearance somewhat resembling dropsical fluid. In this case it is a little brownish, as you see, and flows through the tube with a great deal of difficulty. We will now inject the joint and the synovial sac with the iodine water, washing it out thoroughly in all directions. As much of the iodine water as will not flow out of the canula with ease will be forced out. What may remain in the sac will be harmless. We will now apply a little iodoform collodion directly over the small puncture that has been made, and the limb will be enveloped with gauze and a plaster dressing.

PROCTITIS.

This man has a history of some injury to the rectum. We will try to find out what his present disease is. He is in pretty good health; his general system has evidently not suffered by the accident, whatever it was. On examination, I see a small nipple-like projection about two inches below the anus, from which pus is flowing. I will introduce a grooved director into this opening and endeavor to follow it to the source of the pus. We will place the patient in the lithotomy position and make a careful examination of the rectum. A Sims speculum may with advantage be employed in the rectal examination of this case. The introduction of the speculum into the anus can be done with ease by using the index finger as a guide, introducing the index finger first and then the speculum. In order to prevent the soft fecal matters from flowing out of the anus during the examination, we will pack the rectum carefully with cotton; we can then inspect the margin and the rectal wall. The parts seem to be in a high grade of congestion. There are several ulcerated patches, and on the right side corresponding to this opening I can see anteriorly a little pus pointing, which is doubtless from the internal opening of a fistula. Sometimes we are not able to follow the fistula carefully and easily, but with the finger in the rectum you can usually discover a hard round substance underneath the membrane, feeling as though it were a tube or a pipe buried under the mucous membrane, and this feeling is caused by the hardened and thickened tissues which surround the fistulous tract. The tissues become indurated from long-continued inflammatory infiltration. At the posterior portion of the rectum there is a denuded surface, the seat of an ulcer of considerable extent, and it might be said that we have here the lower rectum in a state of general inflammation or a condition of proctitis.

We will now remove the speculum and endeavor to trace the fistulous tract from the exterior, and I find by passing a smaller probe within a grooved director, for the director will not follow, that the tract reaches, as was supposed, directly inward and a little backward. We must make sure in this case that we reach the internal opening of the fistula. Sometimes the openings are quite high up; at other times the internal opening is at a point quite distant from the direction in which the fistula opens externally. Sometimes the fistulous tract passes all the way around the bowel, thus partially dissecting the rectum from the tissues, and forms what is called a horseshoe fistula. It is in these cases of fistula in ano that failures are frequently found and recorded,

because the entire fistulous tract is not incised. I have now passed the probe into the internal opening, and the patient is suffering no pain, for the reason that we have injected cocaine through the tract. I will endeavor to pass the grooved director alongside of the probe. The channel, as you observe, is quite narrow, and it is a matter of some difficulty to pass it. I will therefore be compelled to enlarge the external opening. Ordinarily, with the finger in the bowel, a grooved director can be passed directly through the fistula, and with a single sweep of the knife the fistulous tract can be cut through. In this case, however, it is so tortuous, and the tract is so narrow, that the usual operation cannot be performed. I have, by following down the probe, cut through the canal, and I here show you the bottom of the fistulous tract, looking almost like a blind sac with a new mucous membrane of its own and indurated thickened tissue at the bottom. I will now curette the tract carefully, so as to remove all pyogenic or infected tissue, and then bring together with sutures the walls of the incision, expecting that primary union will close up this tract, and we will gain two or three days in the healing process over the old operation, which consisted simply in laying open the fistula, packing it, and allowing it to heal by granulation. The point of entrance of this fistula into the bowel will be packed with iodoform gauze and allowed to heal by granulation, and the outer portion of the fistula, having been thoroughly curetted or scraped out, will now be brought together by sutures and closed up. Where these fistulæ have more than one opening, it is absolutely necessary that each of the openings should be traced and the tract carefully curetted, cut out, or laid open, so that it will heal up from the bottom or by approximation of the tract walls. One of these courses is absolutely necessary to be pursued if we would heal these fistulæ. Many a man is treated by simply laying open one fistulous tract. If others are allowed to remain, they go on forming pus, as the pus-bacilli are present; the disease reappears, the man becomes discouraged, and falls into the hands of some charlatan, when by a little care he might easily have been cured by laying open each of the fistulous tracts. The after-treatment in these cases consists in packing the wound with iodoform gauze, a little vaseline having been put upon it, and renewing this dressing morning and evening until the healing shall have been completed.

PRIMARY SYPHILIS—BUBO.

This patient has a history of chancre on the frænum, and he has now a hard swelling in the groin, which, on examination, is seen to be an enlarged lymphatic gland. This, of course, is a typical bubo following a

chancre. The soft chancre, or the chancroid, as it is sometimes termed, is usually followed by suppurating bubo, but not always, and the hard chancre by an indurated bubo. Some believe that the chancroid is but little less than a specific ulcer of the penis, due to an entirely different bacillus from that of the bacillus of syphilis proper. But the method of infection is precisely the same,—that is, by means of the lymphatics. When we find the lymphatic glands are swollen just above Poupart's ligament, following an initial sore on the penis, as in this case, we know perfectly well that the infection has come through the usual channel. It is believed that syphilis becomes constitutionally infectious through the lymphatic system exclusively, and it is on that account that the early excision of chancre was of itself praiseworthy in endeavoring not only to limit the disease and cause early healing of the site of the chancre, but also to prevent constitutional infection by destroying it at its beginning. In these cases we find that the lymphatic glands are acting as sentinels, as it were, to guard the system against constitutional infection. They are violently inflamed, they will soon suppurate, and it is a fact that the pus of chancroid seems to be the more virulent of the two, as there is a more active grade of inflammation, because in syphilis following the true Hunterian chancre there is an indurated bubo which does not suppurate, therefore less local inflammation is produced, and it is a less active infection. With the chancroid there is a suppurating bubo due to the active character of the bacillus of the chancroid. A few inunctions of mercurial ointment, the ordinary blue ointment, will be necessary in this case, and will probably dissipate the inflammation. In case the glands should long continue swollen, or should become permanently hypertrophied, it is well to enucleate them. It is always well, where there is not much surrounding tissue involved in the inflammation, to enucleate syphilitic glands. I believe it modifies the subsequent constitutional attack, from which this patient is bound to suffer to a very considerable degree. We know that active suppuration limits constitutional infection, and we know that investigation shows that negroes, as a race, are prone to suppuration; and they are freer from the later manifestations of syphilis. When a negro has either a soft or a hard chancre, in eight cases out of ten it is followed by a suppurating bubo, and he is very apt to escape those manifestations of the disease which would follow like conditions in the white race.

I shall advise this patient to use mercurial inunctions in preference to the radical treatment of enucleation of the glands. In this case they seem to occupy the whole base of Scarpa's triangle. While they commence above Poupart's ligament, they also extend below it and reach

out beyond the anterior spine of the ilium, and there is no question that the operation of enucleation would be a bloody one and extremely tedious. It is doubtful whether the long period he would have to remain in a recumbent position after the operation would not result in more debility and depression than would follow if he were to walk about while undergoing inunction. In the mean time, under the use of inunctions of the mercurial ointment, and constitutional treatment internally, the swelling of the glands will disappear, and the active inflammation will be subdued.

VARICOCELE.

In the next patient you will notice that there is an elongation of the testicle on the left side, and when he stands up I find a tortuous and twisted condition of the spermatic veins. They are in a state of varix, and on account of this condition the patient comes to us for an operation, as he desires to enlist in the army. Varicocele is generally considered as a disqualification for enlistment on the police force, and for the army or navy in almost all countries of the world in times of peace. In war-times they are not so particular; and unless the varicocele is pronounced, it will not disqualify the recruit from enlistment. The disease, as you know, consists in the breaking down of the valves of the spermatic veins. The veins, holding a great deal of blood, thus become twisted, knotted, and tortuous. They are liable at any time to produce great pain and disqualify a recruit from active work, especially in cavalry service, where horseback-riding is absolutely necessary in the ordinary performance of duty. The diagnosis is easy. When the patient is in the erect position the veins are seen to stand out with great distinctness.

The treatment is either palliative or radical. The radical treatment consists (1) in the extirpation of the veins, (2) in the subcutaneous ligation of the veins, (3) in the open ligation of the veins, and (4) in retrenchment of the scrotum (which is a very old treatment). In most of these cases you will see that the scrotum is elongated. The testicle seems to extend it, until on the affected side it hangs considerably lower than on the side which is normal. Furthermore, the weight of the blood itself in the vein has been conducive to its development and increase. So retrenchment of the scrotum was formerly proposed as a means of bringing up the scrotum to support the testicle and cord, in order that the direct pressure that the skin would exercise on the vein might result in a permanent cure. That is known as the Sir Astley Cooper method of treatment of varicocele. In practice, however, it

was found that the scrotum again became elastic and elongated, and that the veins again filled up: so that this operation is to-day not much practised. Excision of the veins is the favorite method of operating. Subcutaneous ligation may be practised, but it is quite likely to be followed by a severe grade of inflammation; it is attended with a good deal of pain, and the patient perhaps does not make so good a recovery as where the vein is obliterated by the bloodless method. Before giving you a description of that operation, I will mention the palliative treatment, which consists in the application of either an elastic bandage or an elastic suspensory bandage, by which more or less compression is continuously maintained on the veins, and thus the amount of blood in them is reduced.

Excision.—The veins may be excised under antiseptic precautions. We first make a longitudinal incision over the cord, through all the fasciæ; next the veins are caught up and separated from adjacent cord and other tissues by forceps passed under them; then a ligature is pulled through, and the vein, after tying, is cut directly across, and its free end seized with forceps; it is then withdrawn as far as practicable, and removed after ligation. Under antiseptic precautions there is but little trouble after the operation; the wound usually heals by primary union, the collateral circulation becomes established by other veins, and the disease is cured. An incision is now made directly over the line of the cord, extending from the external ring, which I will locate with the index finger. It is through this ring, remember, that the cord passes. You see here the mass of knotted veins, which I have turned out from the cord. We now divide the cremasteric fascia, and the spermatic veins are thoroughly separated from the adjacent tissues. You see the vein which I hold out with the forceps. I pass a ligature around it; it is tied very tightly, and with a firmness sufficient to approximate its walls.

The next step in the operation consists in cutting off the proximal end of the vein with the scissors, and it is tied below and cut off. I have now taken out about three inches of the spermatic veins, and tied them with catgut ligatures, which will be cut short and left in the wound. The wound will now be mopped carefully with sublimated gauze, or if there should be by accident much hemorrhage from the operation, which there has not been in this case, we would use irrigation. The little wound is now closed with catgut sutures, and we may expect primary union. The operation having been completed satisfactorily, let us hope that the patient's desire for military glory will be fully gratified.

CYSTIC GOITRE.

The neck of this patient presents a typical case of cystic bronchocele, or cystic goitre. As I press over the prominent portions of the swelling, I find it is distinctly fluctuating. If we were to tap this goitre we should find that it contained a glairy white-of-egg-like fluid, quite albuminous, without odor, and slightly alkaline in its reaction. These cysts of the thyroid gland are usually multilocular. Sometimes, however, a single cyst enlarges until it presents very nearly the appearance we see in this case.

There are two methods of operating for the removal of cystic goitres. One consists in the injection of a weak solution of carbolic acid, and the other in the removal of one-half or three-quarters of the gland. The removal of the goitrous gland entire, while attended with some difficulty, is not impracticable; but the entire removal has been abandoned on account of the danger of extirpation being followed by myxœdema, a condition in which the mental character of the patient becomes completely changed. The mental powers are much weaker, and finally the patient becomes almost an imbecile. Myxœdema is the thing most feared in total extirpation of goitrous glands. It is now believed that the removal of one-half of the gland is unattended by any such difficulty. I have performed the operation several times without observing myxœdema, and some surgeons even go so far as to say that the leaving *in situ* of any portion of the goitrous gland will prevent this disastrous result occurring after the operation.

The goitre in this case is quite large, extending on both sides of the neck, evidently growing with considerable rapidity. You notice how the swelling pulsates. Distinct pulsation is felt in almost all parts of the tumor. I shall now inject it in the usual way with a carbolic acid solution. There are two or three cases in the habit of coming to the clinic regularly for injections, and they are doing well under this treatment. In one case, a little girl, the goitre has been almost cured by injections of carbolic acid. I have seen a number of cases that were greatly relieved by the injection.

LIPOMA.

You notice in this case quite a prominence just below the seventh cervical vertebra, extending across from one shoulder to the other, a circumscribed swelling, which is painless, and has been growing for seven years. Its painless character and its slow growth exclude carcinoma or other form of malignant disease. It is not a sarcoma,

because, if it were, it would be painful and its growth more rapid; we should have veins showing distinctly through the skin, coming out of this enlargement. I have no doubt that this is a flat lipoma, a so-called diffuse lipoma, owing its shape to various causes, but particularly to the action of the muscles of the back. The patient declines an operation to-day, and will return, she says. The tumor is a solid one.

APPENDICITIS; COLLES'S FRACTURE; FRACTURE OF THE PATELLA.

CLINICAL LECTURE DELIVERED AT THE ST. LOUIS MEDICAL COLLEGE.

BY H. H. MUDD, M D.,

Professor of Clinical Surgery, Special, Fractures and Dislocations, St. Louis Medical College, etc.

APPENDICITIS.

GENTLEMEN,—The two cases which I now present to you have both been before you for observation. They are cases of appendicitis, or rather were cases of appendicitis. The histories of the two cases are quite different. The one, a young man aged thirty, suffered a severe attack of colic in April, 1889. Soreness and local tenderness were present for some days after the onset. In January, 1890, he suffered a second attack, which kept him in bed for two weeks. The pain at this time was well localized in the right iliac region. General distention of the abdomen, with fever and persistent pain and constipation, attested the severity of the attack. A third attack occurred in April, 1890. At this time a hardened mass developed in the right groin, which persisted for a number of months after convalescence. He was in bed with this attack also about two weeks. Persistent tenderness has been present ever since the third attack, the patient never having made a complete recovery. At the time we first saw him, in May, 1890, there was a distinct indurated mass in the iliac fossa. This line of induration was about as thick as the two fingers, well marked, and tender. He was unable to work much during the summer of 1890, but returned to his occupation as storekeeper in the fall. When he could remain comparatively quiet he did not suffer much pain, but any unusual exertion provoked a return of the tenderness and pain. In January, 1891, he suffered a fourth severe attack, and was confined to bed over two weeks. There was a general peritoneal inflammation at this time, at least so the physicians in attendance believed. He again came under our observation in May of the present year. At this

time I could detect no induration in the iliac fossa. There was, however, more or less pain on exertion, and there was a distinctly tender spot on the line between the umbilicus and the spine of the ilium. The sensitiveness at this point varied from day to day. In view of the frequent recurrence of the inflammatory action and the disabled condition persistently present, the patient submitted to operation three weeks ago, May 9, in an interval between attacks. I found upon opening the abdomen with the usual incision that the head of the colon was somewhat reddened, and after searching for a time I found the appendix turned outward along the lower extremity of the colon, held in place by a fold of the peritoneum and by some bands of adhesion. The appendix itself was about the size of a lead-pencil, fully two and one-half inches in length, hard and unyielding in character, somewhat redder at its base than in its body. It was removed without much difficulty and its very broad mesentery ligated in two sections. A simple catgut ligature was placed about the base of the appendix, the mucous portion of the stump clipped out with the scissors, and the abdominal wound approximated with a suture. The patient made a complete recovery without any evidence of inflammatory action about the abdomen. He suffered, however, a severe attack of catarrhal pneumonia, which I believe was in part due to the influence of the ether used at the time of operation.

The second case, a man, aged about forty, persisted in saying that he had not suffered any pain or discomfort in the abdomen until May 7, when he was taken, while at his business, with pain in the abdomen and with nausea. There was no fever during the first day. On the second day there was a slight rise in temperature. I saw him first on the third day, at which time the temperature was 101°, pulse about 80, some distention of the abdomen, with marked tenderness in the right iliac fossa, and with induration and thickening inside of the anterior superior spine. The operation was done May 11, on the fourth day of his sickness. The usual incision exposed the colon, and upon disturbing the adhesion of the colon to the iliac fossa there escaped at once offensive gas and thin purulent fluid. Carefully separating the adhesions of the colon and lifting it from the iliac fossa, the appendix was observed on its posterior surface. It was gangrenous, with a broad well-marked mesentery, the appendix itself being about two inches in length, with a broad base fully opening into the bowel. The appendix was gangrenous and had ulcerated so as to expose a large concretion, which was removed with it. Two separate silk ligatures were placed, one about the base of the appendix and the other enveloping the

mesentery. These ligatures were left long, protruding through the external wound, which was carefully cleansed by sponging. Iodoform gauze was put into the cavity and a drainage-tube placed in position, only a small portion of the external wound being approximated by sutures. This patient made an uninterrupted recovery.

In the first case the operation was performed between the attacks, and the appendix was found in a comparatively good condition, although I think it unquestionably the source of the recurring dangerous sickness with which the patient had been afflicted during the past two years. The wound was at once closed, and the patient was practically well in two weeks. The other had already suppurated, and he was in the midst of an acute attack of appendicitis.

The great majority of the cases which have come under our observation at the clinic have been during the presence of an acute attack, and have been operated upon, where operation was needed, during the acute process. The great majority of these cases have had a circumscribed pus-cavity in the peritoneum. The experience of the past few years has taught me how to open these abscesses to a better purpose than formerly. I confess to have opened many abscesses in the right iliac fossa, in my earlier experience, without recognizing what I believe now to have been the true source of the abscess,—viz., a suppurating appendix. Many of these abscesses I opened by a small incision just below and to the inside of the anterior superior spine of the ilium, passing well up along the iliac fascia and muscle, inserting a tube and allowing the part to drain until a cure was effected. We can open such abscesses, where they are dependent upon the appendicitis, with equal safety and to better purpose by the ordinary incision for the removal of the appendix. Great care should be exercised not to break up the circumscribed adhesions which limit the pus-cavity. If the general peritoneal cavity is opened, it should be carefully guarded, by sponges and by cleanliness, from infection. If a careful dissection is made and the separation of the adhesions about the appendix is cautiously done, the drainage and the escape of pus can usually be easily controlled, the appendix removed, the abscess-cavity cleaned, and the part put into shape for free drainage and rapid healing. In all cases where the pus has been in any considerable quantity, I pack the cavity with iodoform gauze and establish free drainage through the external wound. The removal of the appendix becomes an incident in the operation for the relief of the abscess and the proper cleansing of the cavity. Unfortunately, the great majority of these cases still come to the surgeon after an abscess-cavity has formed. It is then too late

to make a clean and perfect laparotomy and close the wound for primary union. It is true many cases of appendicitis recover without operative measures, the necessary treatment being rest in bed, attention to the bowels, and moderate diet,—the abscess, if there be one, opening into the bowel and discharging through the intestine. Induration and thickening about the head of the colon do not necessarily imply suppuration, for not infrequently the inflammation subsides without the formation of an abscess. It must be admitted, even by the most conservative, that operative interference in these cases is very generally judicious, conservative, and efficient.

The early operations have given us a clearer conception of the pathological changes which accompany the inflammatory attacks. It is astonishing how rapidly the destruction of the appendix is sometimes accomplished by the inflammatory process and how quickly suppuration occurs. I have found the appendix ulcerated and the bowel perforated within three days from the time of the apparent onset of the disease. It is sometimes difficult to get the full history from our patients. For instance, in the last-described case the patient persistently denied any pain in that region until the first day of his confinement to bed, and yet, after the operation had been made, he admitted that he had suffered for many months with pain in the right loin and back, a pain that he had ascribed to the kidney, but which no doubt was due to the presence of this enlarged and inflamed appendix.

COLLES'S FRACTURE.

This young man has a fracture of the lower end of the radius. The injury was received in the usual way by falling upon the palm of the hand, and the displacement of the lower fragment is backward. This gives us the peculiar deformity which is so characteristic of the fracture, a deformity which results largely from the displacement produced by the violence which effects the fracture, muscular action having but little to do with the displacement. As you see, the fulness on the dorsum is just above the wrist-joint, the joint itself being carried backward and forming a part of the dorsal projection. The prominence on the palmar side is a little above the line of that upon the dorsum. The lower fragment is inclined outward towards the radial side. It is sometimes difficult to determine the presence of a fracture in such a case, for if displacement is slight a sprain of the wrist presents symptoms which simulate, in part at least, this deformity. A careful comparison of the relative positions of the styloid process of the radius and that of the ulna will very generally lead to a correct

diagnosis. In this fracture the end of the radius is tilted backward and towards the radial side, the styloid process being carried with it, and it is thus found on a level with or above the styloid process of the ulna, whereas in the natural position of the bones the styloid process of the radius should present below a transverse line drawn from the process of the ulna.

The one important point in the treatment of this fracture is to obtain a perfect adjustment at the first dressing. The fracture is, as you know, through cancellated bone, or the expanded extremity of the radius. Ordinarily, if the fracture is once well adjusted and the position made perfect there is but little disposition towards a secondary displacement. The fracture is by no means uniform in its line. It is a rare thing to find crepitus well marked, the diagnosis depending upon the history of the case and the deformity presented rather than upon the presence of the absolute sign,—crepitus. The fracture is occasionally an impacted one.

The method of reducing the fracture is important. The elbow of the patient being held by an assistant, a second assistant should grasp the hand as if to shake hands. The surgeon now, with his thumbs upon the dorsum, has the fingers of his hand in front of the radius above the line of fracture. Extension by the assistants, aided by firm pressure of the surgeon's thumbs on the fragment, will very generally carry it forward and inward into perfect position. If there is difficulty in moving it into position, the assistant grasping the hand should carry the hand back to extreme extension, then with extension applied in this position the impaction may generally be broken, and the bone carried forward into position. I do not believe the complicated splints that have been devised for the treatment of this fracture are necessary. A small, thin board, a portion of a cigar-box, which should reach from the tip of the fingers to the epicondyle, padded with cotton, affords, I think, the best splint for the fracture. The padding in the palm of the hand should not be very abundant. The hand should rest nearly flat upon the splint. A moderate pad of cotton on the palmar surface should be applied above the line of fracture. The hand resting upon the splint, with fingers extended, is now bound to the splint by a roller bandage. A dorsal pad of cotton is placed directly over the lower end of the radius so as to hold the fragment in position. The splint and bandage should extend to the elbow. No dorsal splint is needed.

The results obtained in the treatment of this fracture should be better than one might expect from reading the ordinary text-books on

fractures. I think the deformity which should result after such fractures should be slight. I have found it more difficult to obtain the full length of the radius than to avoid the deformity which results from the posterior displacement. In order to avoid the stiffening of the fingers, which is very apt to occur in patients past middle life, it is a matter of extreme importance to free the fingers from restraint by shortening the splint at the end of the first week, or certainly at the end of the tenth day. The splint then should extend only to the distal ends of the metacarpal bones. The fingers can be moved freely without danger of displacement of the fracture. This precaution should be practised in all cases in elderly patients. The stiffening of the fingers and the loss of motion in the flexor tendons is ordinarily due to an inflammation in the sheaths of the tendons, or to a rheumatic stiffening of the joint and surrounding tissues.

I make it a habit to remove the dressing and inspect the parts two or three times during the first two weeks, and I remove the splint in the fourth or sixth week, the time being determined somewhat by the age of the patient. The patient should carry the arm in a sling after the removal of the dressing, and I inspect it every few days to see that no displacement has occurred and that the fracture is firmly united.

FRACTURE OF THE PATELLA.

The patient now presented is a man, aged about thirty years, who suffered eight weeks ago a fracture of the patella. The fracture was transverse, with the line of fracture running much nearer the lower extremity of the patella than the upper; in other words, the lower fragment is the smaller one of the two. There was considerable contusion of the joint, and a slight wound in the skin, which communicated with the fracture of the patella. The swelling of the joint was very marked during the first few days. The leg was put into an extended position, and secured by a plaster-of-Paris splint which enveloped the entire limb from the ankle to the groin. The leg thus enveloped was placed in a Hodgens suspension splint, with the foot elevated so that the thigh was flexed slightly upon the body. A large opening was made in the plaster-of-Paris splint on its anterior surface, so as to expose the knee. This opening was carried well up above the level of the capsule of the joint and extended down below the tubercle of the tibia. It should also be cut laterally, so that the margins of the opening may be placed well back upon the lateral surface of the leg. The support of the plaster splint and the pressure of the cotton batting over the knee, with the antiseptic dressing, no doubt aided mate-

rially in the rapid subsidence of the effusion into the joint and of the swelling about the knee-cap. About the fifth or sixth day I applied over the patella a soft metal ring pessary covered with india-rubber tubing moulded so that it encircled the patella. I then passed a loop through the ring upon either side and carried it back around the plaster-of-Paris splint, putting some batting above and below the knee-cap. Sufficient traction was made on the ring to secure a retention of the fragments of the patella in fairly good approximation. The two fragments of the patella should be approximated by the fingers before applying the ring. The ring should be watched, to avoid undue pressure. I usually keep the patient in bed four or five weeks. The patient is then allowed to move about on crutches. After eight or twelve weeks the splint may be removed and moderate motion of the knee-joint be permitted. The patient should not use the leg in walking for at least three months, and not freely for four or five months.

The results following such treatment have been fairly good, free motion being obtained, and a close fibrous union of the fragments being the rule. I resorted to this treatment in preference to the exposure of the fracture and the wiring of the fragments.

Genito-Urinary and Venereal Diseases.

HYPERTROPHY OF THE PROSTATE.

LECTURE II.

BY FRANCIS SEDGWICK WATSON, M.D.,
Instructor in Genito-Urinary Surgery, Harvard Medical School.

GENTLEMEN,—The urine in cases of patients with prostatic hypertrophy furnishes important indications of their condition. The most significant sign of all is the specific gravity. If a patient with prostatic hypertrophy has a urine with a specific gravity of 1010 or less, no matter whether the urine be free from any other evidences of renal trouble or not, this fact alone indicates danger for the patient, and is an evidence of what has been termed, in the absence of further proof of organic renal disease, *renal insufficiency*, by which term is meant that, although the kidneys are not the seat of sufficient pathological change ordinarily to produce death, nevertheless death is likely to ensue under very slight provocation, from suppression of urine or urinary fever and their consequences. This fact is, to my mind, the most important one in connection with the urinary examination. Other than this the urine, of course, presents, in a case in which cystitis exists, the ordinary characteristics of that disease,—viz., alkaline reaction, pus, mucus, blood-corpuscles, alkaline deposits, such as precipitates of the triple phosphates, etc. It is in these cases of cystitis in connection with hypertrophied prostates that we often see the most severe forms of the disease, the reason for which is the inability of such bladders to empty themselves, as has been already described, and that, as a consequence, pus and mucus and decomposing urine remain within the bladder as a source of ever-increasing trouble; so that not infrequently the latter part of the urine is made up almost wholly of such tough, stringy mucus and pus that it will only flow through a large-eyed catheter, and then often with difficulty. Beyond the facts just pointed out, there is nothing of special importance in connection with the urine of these patients. It is surprising how long a chronic urinary obstruction, going even to the point of urinary retention and an overflow

rially in the rapid subsidence of the effusion into the joint and of the swelling about the knee-cap. About the fifth or sixth day I applied over the patella a soft metal ring pessary covered with india-rubber tubing moulded so that it encircled the patella. I then passed a loop through the ring upon either side and carried it back around the plaster-of-Paris splint, putting some batting above and below the knee-cap. Sufficient traction was made on the ring to secure a retention of the fragments of the patella in fairly good approximation. The two fragments of the patella should be approximated by the fingers before applying the ring. The ring should be watched, to avoid undue pressure. I usually keep the patient in bed four or five weeks. The patient is then allowed to move about on crutches. After eight or twelve weeks the splint may be removed and moderate motion of the knee-joint be permitted. The patient should not use the leg in walking for at least three months, and not freely for four or five months.

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BY FRANCIS SEDGWICK WATSON, M.D.,
Instructor in Genito-Urinary Surgery, Harvard Medical School.

GENTLEMEN,—The urine in cases of patients with prostatic hypertrophy furnishes important indications of their condition. The most significant sign of all is the specific gravity. If a patient with prostatic hypertrophy has a urine with a specific gravity of 1010 or less, no matter whether the urine be free from any other evidences of renal trouble or not, this fact alone indicates danger for the patient, and is an evidence of what has been termed, in the absence of further proof of organic renal disease, *renal insufficiency*, by which term is meant that, although the kidneys are not the seat of sufficient pathological change ordinarily to produce death, nevertheless death is likely to ensue under very slight provocation, from suppression of urine or urinary fever and their consequences. This fact is, to my mind, the most important one in connection with the urinary examination. Other than this the urine, of course, presents, in a case in which cystitis exists, the ordinary characteristics of that disease,—viz., alkaline reaction, pus, mucus, blood-corpuscles, alkaline deposits, such as precipitates of the triple phosphates, etc. It is in these cases of cystitis in connection with hypertrophied prostates that we often see the most severe forms of the disease, the reason for which is the inability of such bladders to empty themselves, as has been already described, and that, as a consequence, pus and mucus and decomposing urine remain within the bladder as a source of ever-increasing trouble; so that not infrequently the latter part of the urine is made up almost wholly of such tough, stringy mucus and pus that it will only flow through a large-eyed catheter, and then often with difficulty. Beyond the facts just pointed out, there is nothing of special importance in connection with the urine of these patients. It is surprising how long a chronic urinary obstruction, going even to the point of urinary retention and an overflow

bladder, may persist in cases of prostatic hypertrophy without cystitis occurring, and with the urine showing no signs whatever of trouble, except a low specific gravity. In connection with the condition of the kidneys which I have designated by the name of renal insufficiency, and also in a more marked degree in those cases of cystitis with or without hydro- or pyo-nephrosis superadded, there are certain constitutional disturbances which have also marked significance, and which are due to a chronic form of uræmia. The more noticeable symptoms of this condition are a coated tongue,—the tongue being cracked, dry, and brown, the mouth parched, or sometimes a very red tongue; flatulence; an occasional feeling of nausea; more or less muscular weakness, especially of the legs; thirst; and an irregular action of the bowels. This group of symptoms, in connection with the local conditions just referred to, is very significant of danger to the patient, for it indicates, as a rule, that the renal insufficiency, even if no more grave disease of the kidneys is present, has advanced to a dangerous point.

TREATMENT.

In concluding this lecture I shall briefly summarize the more important points on the *palliative* treatment of this disease. Bear in mind the fact that it is the presence and the increasing amount of residual urine in the bladder, and their consequences, that are primarily the most important factors in this malady, and you can see at once that the treatment which will rid the bladder of its unnatural load and allow it to empty itself again entirely will constitute the most important feature; and *it is the use of the catheter* which is, indeed, the most important element in the treatment. The *amount of residual urine* which should determine the use of the catheter varies in the practice of different surgeons, and is an arbitrary one. My own rule is this. The residual urine may be as much as six ounces without the catheter being necessary, provided the presence of that quantity in the bladder has not caused the patient to get up more than once at night to urinate, and has not caused urination during the daytime more frequently than once in two or three hours, and if the urine is clear; in other words, if no cystitis be present. In any case in which the residual urine exceeds six ounces I have found it better to use the catheter once in twenty-four hours at least. And here let me say that catheter life is not to be entered upon lightly. This point cannot be impressed upon you too strongly, for, in spite of the innumerable warnings scattered through all the writings and teachings on prostatic hypertrophy against the practice of carelessly drawing off suddenly a large quantity of residual

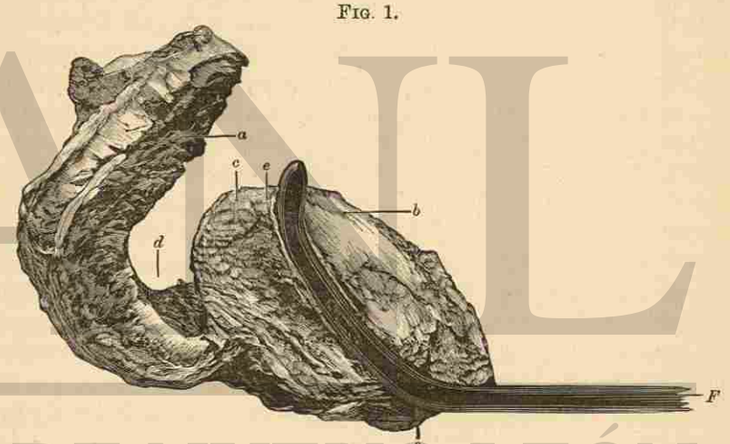
urine with a catheter which is not thoroughly clean, such remains the bad and frequent practice with many physicians. While it is perfectly true that many cases will have no trouble following such a procedure, it is equally true that every year where such practice occurs a certain number of deaths may be directly laid to its door; and special precautions should be taken in beginning the use of the catheter with patients whose urine has a low specific gravity, and when the residual urine is already large,—*i.e.*, from eight ounces upward. It is in that special class of cases that the sudden withdrawal of the urine is sometimes followed by urinary suppression and death, or by the constitutional disturbances characterized by chills, fever, dry tongue, mild delirium, or presenting itself in another form, with a weak pulse, fainting, tendency to collapse, and so on. It is never a mistake to be on the safe side and to do the best we can to avert these misfortunes, and if you will observe the following rules you will have done your best to avoid them. When it is intended to begin the use of the catheter, confine the patient to the house for at any rate the first five or six days of its use. Pass whatever instrument is used with great gentleness. Never use force in trying to make a catheter enter the bladder. If the residual urine is more than eight ounces, do not withdraw it all at once; stop when eight ounces have been evacuated, and if that quantity should represent *all* the residual urine, even then inject two ounces of a four-per-cent. warm boracic acid solution into the bladder, instead of leaving it quite empty. Where the residual urine does not exceed six or eight ounces and no cystitis is present, it is unusual to be obliged to use the catheter more than once in twenty-four hours. If, however, with that quantity (whether in the presence of a cystitis or not) the frequency of urination previous to the passage of the catheter has been marked,—*i.e.*, every one or two hours in the daytime and three or four times during the night,—and it is not relieved by the single use of the catheter each day, the irritability of the bladder may be relieved by the passage of the catheter as often as three or four times in the twenty-four hours. If, however, at the beginning of catheter life the passage of the instrument creates a marked irritability of the bladder, this irritability having been previously absent, then suspend the regular daily use of the catheter, and allow one or two days, or such a time as may be required for the bladder to become less irritable in the absence of the instrument, to elapse previous to making a second attempt. There is, however, one condition in which the rule just laid down cannot be observed, and that is when the surgeon is dealing with a case in which there is retention present or threatened, and especially if there be a

large residual urine: if the regular use of the catheter has been begun in such a case and cystitis with its local and general symptoms occurs, it is, I think, safer to tie a soft rubber catheter into the bladder instead of interrupting its periodic use, and to let it remain, changing it for a clean one every three days, until the urine shows a decreasing amount of pus or becomes again clear, and fever and such constitutional symptoms as may have been present have greatly improved or ceased, the catheter may then be removed, and its regular daily use resumed. So long as cystitis is present it should be treated by the use of bladder washes in the manner to be described at the end of this lecture. It sometimes happens that the first one or two attempts with the catheter are followed by great irritability of the bladder and by constitutional reaction and disturbance, and yet a subsequent attempt may be crowned with entire success. If an over-distended bladder is present, it must be approached with the utmost caution by the catheter. The patient should be confined to bed, put upon a light diet, and kept warm. The catheter should be gently introduced and the over-distended bladder relieved very slowly and of but comparatively small portions of its contents at a time. Such over-distended bladders may hold anywhere from one quart to two or three quarts of urine, and in any case, at the first passage of the catheter, not more than eight or ten ounces of the urine should be removed, and in the manner already described,—*i.e.*, a portion of the fluid removed should be replaced by a couple of ounces or so of a four-per cent. boracic acid solution. These greatly over-distended bladders occurring in the course of urinary obstruction rapidly refill, and polyuria almost invariably follows upon the withdrawal of a large amount of urine. Therefore it becomes necessary within a very few hours to relieve the bladder again. In the first twenty-four hours the catheter may be used as often as four or five times, withdrawing a slightly larger quantity at each sitting; for instance, beginning with eight ounces and increasing the amount by two ounces at each of the four uses of the catheter. In this way in the course of a few days, as a rule, the bladder may be emptied; but I have seen one or two instances where it required between two and three weeks of such daily use of the catheter before the bladder was wholly emptied.

Another method of emptying such over-distended bladders, and one which promises, I think, greater immunity from the occurrence of urinary fever in these cases, is that of frequent aspiration by means of a fine needle introduced above the symphysis pubis. The urine may be withdrawn by repeated aspirations (to the number of four or five a

day) in the same quantities as if the catheter was used, and the bladder thus gradually emptied. After three or four days of the aspirations, if the contents of the bladder have become largely reduced and its over-distention relieved, the catheter may *then* be substituted, and I think with less risk of setting up urinary fever than had it been used at the outset. If abscesses should form about the points of puncture, the aspirations should not be continued. Another rule of importance is that of keeping the catheter as clean as possible. The soft red rubber catheters can be easily kept in an aseptic condition thus. Let a stream of warm water run through them immediately before and after use, and after use syringe through them a solution of carbolic acid of the strength of one part to twenty. The catheter should then be once more washed with warm water, and placed in a tube or bottle (which can be corked) containing a saturated solution of boracic acid,

FIG. 1.



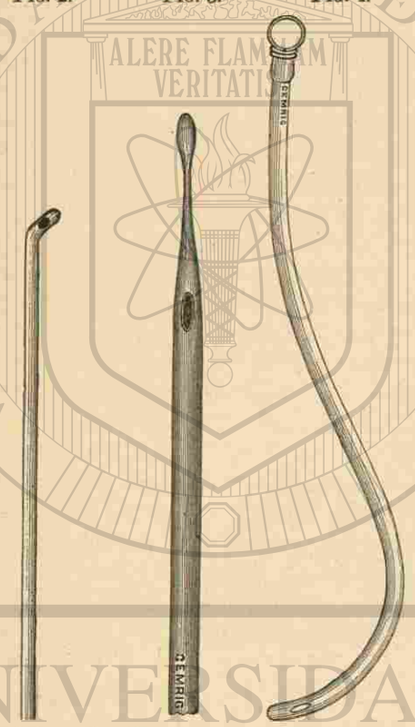
Represents a perpendicular section through the bladder, median enlargement of the prostate, and prostatic urethra. In the prostatic urethra lies a Mercier bicoudé catheter (*F*), the instrument having its natural form. *a*, greatly thickened bladder wall; *b*, left lateral lobe of prostate; *c*, middle prostatic lobe; *d*, bas fond of interior of bladder behind the hypertrophied middle lobe; *ee*, represents the course of the prostatic urethra. (Photographed from fresh specimen by the author.)

which neither rots the catheter nor causes it to irritate the urethra when again passed. The boracic solution should be frequently changed. The webbing catheters which are shellacked are spoiled by being *kept* in fluids, and therefore the best treatment is to wash them thoroughly with warm water before and after use, and then to syringe a quantity of corrosive sublimate solution through them, dry them, and keep them

in a clean place until next required. It is a frequent custom for patients to carry their catheters in their pockets, where they necessarily become dirty, and then are liable to carry septic materials into the bladder.

Next in importance to the *method of using* the catheter is the form of the instrument to be used. The statement which follows will sound, I am aware, arbitrary. I give it to you only as a matter of my personal preference. I have never been obliged, and do not think I ever shall be obliged, to use a metal catheter to enter the bladder over a prostatic obstruction. I advise you against this instrument, because, except in skilled hands, it is dangerous and likely to make false passages. The form of catheter which will enter the bladder in these cases more readily than any other is, I think, that called the *Mercier bicoudé*, or double-elbow catheter, which is shown in the accompanying illustration (Fig. 1). Fig. 1 illustrates admirably how this double-elbow catheter is adapted for its purpose in some cases. It will be seen in this figure that the catheter conforms very exactly to the shape of the posterior portion of the urethra in cases of large obstructing middle lobe hypertrophies of the prostate, which are those which, as a rule, present the greatest difficulties to the passage of the instrument. Next to the bicoudé the coudé catheter (Fig. 2) is most serviceable. Occasionally the finely-tipped bougie catheter (Fig. 3) will enter more readily than those just referred to. Another good way of entering the bladder is to

Fig. 2. Fig. 3. Fig. 4.



Mercier's coudé catheter. French bougie catheter. Over-curved flexible catheter.

pass the ordinary English red-webbing catheter armed with a stylet down to the point at which it meets the obstruction: by then withdrawing the stylet for about two inches and at the same time pushing the catheter gently forward, its tip, which the withdrawal of the stylet has thrown upward in the curve represented in Fig. 4, will surmount the median enlargement and often enter the bladder readily. If you cannot pass one of these forms of catheter, the chances are very much against your being able to pass any. The greatest advantage, besides that of adaptability to their purpose, of the above instruments is their freedom from danger. More serious trouble occurs through ill-advised and strenuous efforts to pass metal catheters with so-called prostatic curves than through any other interference. It is of frequent occurrence, I am sorry to say, to see cases of prostatic hypertrophy in which false passages have been made in the prostatic urethra by the injudicious use of metal catheters by physicians. If a false passage *is* made in this way it is generally situated at the anterior end of the prostatic urethra, at the point at which the prolongation of the third lobe into the posterior urethra is met with. When a false passage is made it announces itself generally by a more or less copious hemorrhage from the urethra following the withdrawal of the instrument, and also by the failure of the urine to flow through the instrument,—unless, indeed, the catheter has, as has sometimes happened, been forced upward through the prostatic enlargement, tunnelling it, and thus made to enter the bladder. If a false passage be formed it is frequently the source of septic infection of the patient, owing to the fact that it becomes filled with stagnant urine and decomposing pus for which there is no free drainage; and lastly, it often makes an insurmountable obstacle to further use of the catheter. It therefore constitutes a serious accident in the course of catheterism, which need never occur if the catheters of the kind I advise are used and properly used.

In regard to the *size* of the instrument, it is best to begin with one of moderate calibre, say No. 16 of the French scale, and if that meets with marked resistance, smaller sizes can be used until one is reached that will pass readily. A small instrument will sometimes excite spasm and fail to pass as easily as a larger one.

The best lubricant that I have found is *mineral glycerin*, although liquid vaseline or glycerin is perfectly good.

After the patient has been started favorably upon his catheter life he can be readily instructed, in most instances, in the use of the catheter himself.

On the treatment of urinary fever I can say only a word or two, as

it forms a subject by itself. It occurs in connection with this disease as with other diseases of the urinary tract. I shall only outline a few general principles. Chills should be combated by stimulants (and of these I prefer rum or whiskey) and by heaters. Morphine, which may be given in ordinary doses to younger patients, or to patients in whom there is no suspicion of kidney-disease or renal insufficiency, should in cases occurring with prostatic hypertrophy be used with caution, but may be beneficial if so used. All cardiac depressants should be carefully avoided. Digitalis may be given when there is no contra-indication and when the character of the pulse indicates its use.

It is a very common custom among physicians to treat patients with prostatic hypertrophy, who present symptoms of irritable bladder, with diluents and diuretics, such as citrate of potassium, various mineral waters, etc. If the residual urine is being drawn off regularly by the catheter the symptoms of bladder irritability may be relieved by the proper use of such means, but if the catheter is not being used regularly the patient's trouble is only being added to by diuretic treatment, for the reason that more water is poured into the bladder by the kidneys, and the pond of residual urine is consequently constantly increased.

A number of years ago a good deal was said in favor of the use of ergot and strychnine in cases of prostatic hypertrophy. By the use of ergot the gland was thought to be made to contract, and strychnine was said to give an increased power of expulsion to the bladder. I have never seen any marked effect from the use of ergot, except in the early or congestive period of the disease to which I have referred, and I cannot, therefore, say much in favor of its use. Strychnine has seemed to me to give some benefit in cases of atony of the bladder by aiding its expulsive power.

The treatment of cystitis when it coexists with prostatic hypertrophy is of much importance. More or less benefit may be expected to follow the use of certain internal medicines. Of these, those which I have seen work the most good are sandalwood oil in capsules, in doses of five to ten minims three or four times a day. It should always be borne in mind that with the larger doses of sandalwood oil there is a liability to renal congestion. I never like to employ in these cases sandalwood oil for a long period, but prefer to be on the safe side and interrupt the use of the drug for one or two days in every week, and not to continue it longer than four or five weeks at any one time consecutively. The following drugs sometimes do excellent work in rendering

the urine more clear and diminishing the bladder irritability in cases of cystitis: benzoate of sodium, acetate of potassium, each in doses of ten grains t. i. d., well diluted with water; the combination of a decoction of ulmus fulva and succus hyoscyamus is recommended by Reginald Harrison in the acute or subacute attacks of cystitis which frequently follow the first early use of the catheter. Mr. Harrison also speaks favorably in cases of chronic cystitis of the fluid extract of sabal serrulata (saw-palmetto) in doses of one drachm three or four times a day. The infusion of pareira brava is sometimes also beneficial. One other drug I have not infrequently found to produce strikingly beneficial results, and that is boracic acid, taken best in the form of pills, from three to six grains t. i. d. Under its use the urine may from being exceedingly foul become in the course of two or three weeks quite clear. An excellent regimen is a milk diet combined with the use of Vals water. Between two and three quarts of milk should be taken in the course of the twenty-four hours. The milk should be warm, and each tumblerful should be diluted one-quarter with Vals water. Boiled fresh fish and dry toast or Graham wafers may be allowed in the course of this diet. More important, however, is the local treatment of the bladder. Let me say at once in regard to bladder-washes that the attempt to cleanse the bladder by the use of the stronger antiseptics has proved in my hands, after thorough trial, to be distinctly deleterious. Not so, however, with the milder ones, and of these I have had the most beneficial results from the use of a four-per-cent. solution of boracic acid, or a one to four-thousand solution of permanganate of potassium. In the milder cases of cystitis the solution of borax and glycerin recommended by Sir Henry Thompson is admirable. Recently I have obtained in two cases of very obstinate and long-standing cystitis very marked benefit by the use of myrrh wash, beginning with a solution of the strength of the tincture of myrrh one part, water fifty parts, and increasing the strength up to one to sixteen.

The way in which bladder-washes are used is important. I have never seen any benefit result from attempts to distend small contracted bladders by forcible injection of fluids, as has been sometimes recommended. On the contrary, increased irritability has invariably resulted in my hands. The method which yields the best results is that recommended by Sir Henry Thompson,—viz., the injection (after emptying the bladder) of the fluid selected in quantities of from two to four ounces at a time, letting it run out again at once, and repeating this process until the returning fluid becomes clear. All bladder-washes

should be used warm, and oftentimes much additional benefit is gained by heating them to a temperature of from 110° to 120° F. In some cases of long-standing chronic cystitis with very foul urine, injections of a weak solution of nitrate of silver, one-half grain to one and one-half grains to the ounce, or of a solution of acetate of lead, sometimes work very well.

In the next lecture we will discuss the operative treatment of the hypertrophied prostate.

THE RESISTANCE OF THE BLADDER TO INFECTION.

SURGICAL CLINIC AT THE NECKER HOSPITAL, PARIS.

BY PROFESSOR FELIX GUYON.

THE patient we desire to study to-day you will find in No. 19 bed, Laugier ward. She first came here last November, went out, and then came back to us in February. She is twenty-four years of age, and has no hereditary predispositions of interest. She came here simply because for the past three years her urine has been thick (exhibits a large deposit on standing, and smells badly). We examined her urine and found that it contained pus. The patient has no other functional symptoms,—no frequency in urination, no pain in passing water,—but she says that she has shooting pains in the right iliac region. This caused us to examine her, and we found in the right portion of the body of the uterus and adhering to it a large mass, which held on to the walls of the pelvis and had a prolongation going down to the bladder.

What connection can such a tumor have with the state of the urine? The endoscope gave us an answer to this question. As it lit up the bladder we found a small orifice on the right side, from which pus was escaping. Two months ago pus came away in considerable quantity when we pressed on the tumor, but now it is difficult to obtain any even after considerable pressure. So, then, there is a direct communication between this tumor and the bladder, but there are no signs of cystitis, as we usually see it. The aspect of the bladder under the endoscope shows that the mucous membrane is normal, the color being that of a healthy bladder, and when any instrument is passed around the surface of the bladder there is no sensitiveness. As to the sensation by distention, we passed four hundred and fifty grammes of water (nearly a pint) before there was the slightest sign or desire to urinate; she did not empty the bladder until it had received five hundred and thirty grammes: so that this organ is normal, and there is not the slightest

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sign of any anatomical modification of the mucous membrane of the bladder. We must therefore conclude that there is no cystitis.

The bacteriological examination of this urine presents some interesting points. Two inoculations were made with it on the 29th of February, one in a guinea-pig and one in a mouse. The first animal died in five days without any particular lesion, and the mouse is still alive (April). The cultures made with the blood of the kidney and heart of the dead animal remained sterile. Remember this point, as we can draw a lesson from it later. Then with the same urine cultures were made, which gave us ovoid forms of bacteria sometimes united in chain form. On the 3d of March these cultures were repeated, and gave positive results, and on the 9th of March the culture was inoculated in a guinea-pig's peritoneum. The animal died the same night, and at the autopsy we found that there was a thick liquid in the folds of the peritoneum and some perihepatic adhesions. This liquid contained a large number of ovoid bacteria, and cultures were made of the blood taken from the heart and kidney, which all gave the well-known characters of the bacillus pyogenes.

Such are the facts of this case: on one side a virulent pus that has been thrown into the bladder for three years, and on the other side a bladder that has remained healthy under this. The question is put to us, Is this usual? or is it abnormal? or does the bladder resist infection? To my mind it is normal, and clinical observations, as well as bacteriological experiments, confirm this view of the matter, which I have held for some time.

That a cystitis can be produced only by the intervention of a microbe is certainly an established fact. One of our internes, Dr. Reblaud, confirms this in his thesis on "The Etiology and Pathogenesis of Non-Tubercular Cystitis in Women." A microbe certainly plays the most prominent part in the production of cystitis, but its presence alone is not sufficient to produce the disease. You see by our present case that the bladder will remain quite indifferent to it, and will continue so just as long as the mucous membrane has not undergone such modification as to allow of the micro-organisms fixing themselves in it and hatching there. These conditions must be properly studied, otherwise you will not have a true idea of the pathogenesis of cystitis. The case we have given shows you the resistance of the bladder to infection, and brings up the question as to what are the conditions that will permit this resistance to be overcome and allow of the implantation of the micro-organism in the mucous membrane and the subsequent appearance of a cystitis. The morbid conditions in question are those that clinical experience has enabled us to observe for a long time back, and their study will once more prove that, in most cases of disease, for a morbid sowing of unhealthy seed to prosper it is absolutely necessary that the ground be properly prepared. It is not enough that the seed be sown; it must take root. The proper conditions in question are of both a physiological and a pathological nature, and these two must be kept in sight in making a study of any infection.

Let us first of all examine into the question as to what are the necessary conditions for microbian invasion. As far as cystitis is concerned, they may be stated as being three in number,—*retention*, *congestion*, and *traumatism*. Let us start with the last, which is least in importance. M. Reblaud has made a number of experiments in regard to this matter, and he found that small traumatic lesions, even with micro-organisms in the bladder, did not cause cystitis. We have frequently scratched the surface of the mucous membrane of the bladder with a sound and pinched it with instruments without causing inflammation. You often see us open the bladder, scrape it, burn it, and drain it, without producing any unpleasant results; and when we do cause cystitis it will yield rapidly to treatment.

With regard to *congestion* it is quite a different matter. We constantly speak of the important part that it takes in the pathology of bladder-diseases. Its influence in the etiology of cystitis we have insisted upon. No matter how important you may fancy the traumatic lesions to be, you will always find them less so than congestion, which will modify the whole extent of the mucous membrane in depth at least, and will make the smallest vessels permeable, and no other condition of these parts is so favorable to the penetration of germs. M. Reblaud made some interesting experiments in regard to this point. Having produced an artificial congestion of the mucous membranes of a rabbit by giving it cantharides, and having found the urine quite free from micro-organisms, he made an injection of pure culture liquid from a preparation of the staphylococcus pyogenes albus, and two days afterwards the urine of the animal gave the characteristic signs of this organism. I must here remind you that the injection of such a culture liquid into healthy veins would not produce this result, and as to the action of cantharides you are all aware that a cystitis can be produced by it, but that the inflammatory condition will rapidly subside, even without the usual injection of an opium product. I, indeed, have never seen this kind of cystitis persist, except in two cases, where there was tuberculosis or where the bladder was already infected by disease.

Let us now examine the third condition necessary for the microbes to enter the system by this route,—that is to say, *retention*. Its importance is considerable, and it can be divided into four heads. 1st. *Stagnation of urine*. 2d. *Congestion*, which may be total and extend to a parenchymatous hemorrhage. 3d. *Loss of the epithelium*. 4th. *Vesical paresis*, which is owing to the distention of the bladder. So that retention alone can produce such alteration of the anatomical parts of the walls of the bladder that it is left open to the action of the micro-organisms. It will do everything necessary to cause infection and keep it up. I am speaking at present of the bladder only, but my studies made with M. Albarran show that the same thing happens for the ureters and even for the kidneys. The resistance, then, of the bladder to infection is broken up by retention: all those who have made experiments on this subject are agreed on this point, and that simple injection into the healthy bladder will not produce disease. Schnitzler claims that he has produced intense cystitis by simply injecting into a rabbit's bladder a culture of urobacillus liquefians septicus, but we have never been able to accomplish it. The virulence of the microbe is, however, an important point, for if all the various kinds produce a cystitis after a retention for twenty-four hours, still they by no means produce the same intensity of inflammation. M. Reblaud has shown that it requires a varying amount of time for retention to produce these changes according to the variety of microbe present. Thus, the micrococcus albicans amplus and the diplococcus subflavus do not cause any reaction after twelve hours' retention, while the urobacillus and the bacterium pyogenes will act in six hours. So that we must admit on the one hand the virulence of the microbe, and on the other that retention is necessary to infection. Our patient, whose bladder is full of the bacterium pyogenes, one of the most virulent, showed that just so long as there is no retention there will be resistance to infection. Here, then, the regular and complete evacuation of the bladder was sufficient to prevent contagion and resist infection. This is a practical point well worth your attention. Let me give you the history of another case.

I operated in 1889 on a man thirty-five years of age who had a pyonephrosis of the right side, and his urine was purulent. By the second day after the operation the urine was clear, much to the astonishment of the patient and of some of the students, who had seen this patient always with purulent urine. I have also called your attention to cases of intestinal trouble where there was a direct communication with the bladder, and notwithstanding the flood of coli

bacilli into the organ it resisted infection, and no cystitis took place. Professor Bouchard has shown that infectious maladies may cause nephritis and the microbes are eliminated by the urine, and yet how seldom have such troubles been followed by nephritis! We do not, however, mean to say that the conditions of infection are never influenced by the general state of the patient.

After what I have said it seems difficult to accept any idea tending to prove that cystitis varies according to the *kind* of microbe that produced it; no such classification of cystitis can be accepted. There is nothing in the symptoms or in the evolution of this disease that shows that it is different according to the *kind* of micro-organism causing it. The fact that there are microbes involved is, of course, important, but they are not indispensable to its production, while they certainly contribute to keep it up and preside over its birth, so to speak. When they disappear the inflammation begins to subside. The theory, then, of the etiology of cystitis should continue as it is accepted at present, and serve as the basis of all classifications, while we may add to it the pathogenic theory; but we should not substitute one for the other.

INTRA-VESICAL FATTY TUMORS.

CLINICAL LECTURE DELIVERED AT THE JEFFERSON MEDICAL COLLEGE HOSPITAL.

BY JOHN H. BRINTON, M.D.,

Professor of the Practice of Surgery and Clinical Surgery at the Jefferson Medical College, Philadelphia.

GENTLEMEN,—The case which I shall now bring before you is one of great interest. The patient is a Hebrew peddler, about forty years of age, and comes to us from Honesdale, in the interior of Pennsylvania. His trade or business requires him to be constantly on his feet, and he frequently walks many miles in the course of a day. He first came to this hospital in 1884, and was then treated by Dr. Barton, one of the hospital surgeons. This was his history at that time. He had suffered for several years with hæmaturia, accompanied by vesical irritation. His urine contained pus and phosphates, and there was evidently cystitis, but microscopic examination failed to detect any shreds of foreign tissue. There was no enlargement of the prostate, nor could any calculus be detected. With the sound, however, several elevated and irregular places were discovered. Examinations of the bladder were usually followed by bleeding. Under injections of borate of sodium he improved rapidly, and left the hospital, but again returned in September, 1885.

He was at that time brought before the class by Dr. Barton, whose clinical remarks were published at length in the Philadelphia *Medical Times*, September 5, 1885, p. 905. From these I learn that the patient was then urinating more than fifty times in the twenty-four hours, with frequent passage of blood; the bleeding, occurring at intervals for more than four years, was the first symptom noticed. The blood was always fresh, of bright-red color, and, when floated in water, assumed the form of flattened, irregular patches. The absence of calculus and of marked prostatic trouble, the age of the patient, the comparatively healthy condition of the urine, and the want of evidence of ulceration

on the walls of the bladder, all tended by exclusion to refer the difficulty to some form of bladder-tumor.

Acting on this supposition, Dr. Barton performed the median section, and, having introduced his finger, was able to detect a soft, velvety mass springing from the upper surface of the bladder, which when removed by the forceps afforded the characteristic appearances of papilloma. Some slight hemorrhage followed the operation; this, however, soon ceased, normal micturition was soon resumed, and the patient left the hospital entirely relieved. For six years he carried on his business of peddling, but during 1891 his vesical symptoms gradually returned.

In January of the present year he again entered the hospital, under the care of Professor Forbes, who on the 20th of that month performed a left lateral lithotomy, and removed from the bladder several masses of what appeared to be papillomatous growths. After this operation he improved somewhat, and was discharged from the hospital towards the end of February. Since then the vesical irritation has returned, and in the early part of the present month he was readmitted for treatment. He complains now of excessive irritability, which is increased when he is on his feet and walking. He passes his water every few minutes, and there is some pain in the head of the penis and above the pubis. His urine is phosphatic, and contains a little pus. The wound in the perineum from the last incision is not entirely healed; there is a perineal fistula, and occasional urinary dribbling. The bladder is contracted and very small. I have carefully filled it by simple hydraulic pressure from the douche, and find that its utmost capacity is about two ounces.

It seems to me that the only mode of relieving this man will be by the performance of a supra-pubic cystotomy, careful exploration of the bladder, and the removal of any growths which may be found. This treatment is indicated by the present symptoms and by the past history of the case. I shall doubtless find some sort of tumor, papillary, sessile, or polypoid. In the supra-pubic section, as most often practised, the bladder can be made prominent above the pubis by injection with eight or nine ounces of water, and by the insertion of the rectal rubber bag, holding ten or twelve ounces. In this case we can use the latter, but, as the capacity of the bladder is so greatly reduced, I shall be obliged to attack it deeply seated in the pelvis, the peritoneal fold being carried downward and forward with it. I shall therefore introduce a metallic catheter, dilate the bladder to the greatest extent that its contracted state and the existence of the perineal fistula will permit, and then, cutting through the abdominal walls and pre-vesical fat, open

the vesical wall upon the catheter. This must be hooked close to the pubis, so as to endanger as little as possible the fold of peritoneum.

This external incision I have now made; the bladder-walls are exposed anteriorly; I pass through them two strong sutures, open the bladder between them, and, introducing my finger, I feel some soft pendulous masses; I bring them into view, and find that they are three in number, pedunculated, with vesicular bases, and apparently fatty. I tie them with fine silk to prevent bleeding, excise the masses, insert an india-rubber drainage-tube, close the wound, and send the patient to the ward.

[On May 20 the patient was again brought before the class by Dr. Brinton, who stated that the three tumors he had removed from the bladder in this case had been microscopically examined by Dr. Coplin, and found to be purely fatty. A few hours after the operation the drainage-tube was removed, as the urine passed readily by the urethra.

There was some tympanites after the operation, which passed away under drachm doses of Epsom salts, a little Dover's powder, and rectal injections of Epsom salts and turpentine. After this all unpleasant symptoms disappeared, the patient's appetite returned, pulse and temperature became normal, and the wound healed.]

The peculiarity in this case was the character of the tumors, which were undoubtedly fatty, but which hung down from the upper and left vesical wall: one of these growths was pedunculated, while the others were sessile, with comparatively broad bases. The main vessels, which were distinct and largely injected, ran transversely at the base of the growths. In this case it might have been possible to ascertain the nature of the affection before operation, by electrical cystoscopy, had the condition of the urethra been such as to permit the necessary instrumentation. The tenderness of the urethra, the result of previous operations, however, forbade the attempt.

THE TREATMENT OF CHANCROID.

BY DR. BALZER,

Physician to the Hôpital du Midi,[1] Paris.

GENTLEMEN,—The first indication in the treatment of soft chancre consists in the destruction of the ulceration, which is a real focus of infection, producing, as it does, a highly-contagious pus that is auto-inoculable. It is also the point of departure of secondary lymphatic infection. But in undertaking to crush out the virulence of this venereal ulcer it is well to remember that its influence does not extend below the surface, as a rule. It may extend over a considerable surface, but it does not usually do so: so that it is possible to act against it with energetic agents, such as caustics, the hot iron, and the like. It is also possible to make a complete ablation of the chancre; and Dr. Humbert has lately performed this operation for a soft chancre of the arm. Rollet used the actual cautery, but nowadays we employ the thermo- or the galvano-cautery. As to caustics, Ricord's carbo-sulphuric paste was formerly much used. It was made of powdered charcoal and sulphuric acid, ten grammes of the former to four grammes of the latter, making a half-solid mass. It is not much used at present. Chloride of zinc paste, like the pâte de Canquoin, has been much recommended by Dr. Diday. It destroys the virulent ulcer completely, and does not spread much, but it sometimes goes deeply into the tissues, and has been known to reach the blood-vessels and cause hemorrhage. We recommend the following paste:

℞ Chloride of zinc, 1 part;
Oxide of zinc, 9 or 10 parts;
Distilled water, a sufficient quantity.
Mix, and make a paste.

This may be applied directly on the ulcer, or, better, on a small bit of antiseptic cotton. Its action will soon be manifest, as it causes pain, and very often a little swelling; but it is not dangerous, remains always superficial, and does not need watching. The pain can be borne,

[1] The Hôpital du Midi is a venereal hospital, where Professor Ricord once taught.

and the dressing should be taken off in twenty-four hours, when a slight white covering will be found on the ulcer, formed by the tissues on its surface. One application of this is enough, as a rule, but sometimes it will be found necessary to make two or even three. In certain places, like the fingers or the penis, it is useful to make a permanent bandage of it, which should be changed daily. In fact, this form of paste holds a rank between the real escharotics and the caustics. In a certain number of cases we use weaker caustics in a liquid form, and apply antiseptic agents as well. These caustics are simply applied by drops, every two or three days, and an antiseptic dressing put on in the mean time. Among the caustics, one of the best is the chloride of zinc, either in a saturated or in a ten-per-cent. solution. It is slightly painful, but sure. Iodoform or aristol may be used for the dressings. We have also applied zincated ether or zincated alcohol (1 in 10), and get the same results from them as from the chloride of zinc. Dr. Ducastel used a solution of carbolic acid in alcohol (1 in 10). This is a good method for home or office practice, as it is not painful, and you have not only a caustic action but an antiseptic and an analgesic one besides. This may be followed by the application of an antiseptic powder. Professor Fournier still uses nitrate of silver in a three-per-cent. solution. Sometimes a solution as strong as fifty per cent. has been used. With Dr. Fournier, we prefer weak solutions, which we apply on cotton, and keep on permanently. It is good to remember, however, that these dressings do not always remain limited to the place intended, and the adjoining parts may be also acted upon. This is rare, however, with well-made dressings. Dujardin-Beaumetz has obtained good results with a five in-twenty solution of chloral. Dr. Marc Sée uses silicate of sodium in three-per-cent. solutions. Resorcin has been much praised, in the powder form, or one-to-twenty solutions. We tried camphorized naphthol and salolized camphor when we were at the Lourcine Hospital, as well as the salts of iron,—the perchloride, the citrate, and the tartrate (of iron and potassium). This last, both internally and externally, was much used by Ricord, who said it was the sworn enemy of phagedæna. Like nitrate of silver, these salts are used in strong solutions for touching these ulcers, or in weaker solutions for more permanent dressings. Dr. Besnier, in 1866, recommended iodoform, and it is still much used, notwithstanding its smell. It may be depended upon to cure soft chancre without any other treatment. Every means has been employed to overcome the odor of iodoform, but the best methods do not exert their influence long, and the characteristic odor will return.

Iodol and aristol merit attention. This last is the biniodide of thymol. While they have the great advantage of not being disagreeable in smell, still they are not so effective as iodoform. Iodol forms a sort of covering on the surface of the ulcer which retains the pus. Aristol acts better, and in city practice we often advise iodoform applications at night, and aristol during the day. Two newer substances, europhen and biniodide of thiophen, are without odor, but have not been used as yet sufficiently long for us to reach a definite conclusion in regard to them. The salicylates and salol have not given good results. We use in this hospital a powder composed of chloride of zinc, one part, to oxide of zinc, nine parts. It has no smell, and seems to meet all the indications for the treatment of soft chancre. It is easy of application and costs almost nothing. If found too caustic, we add oxide of zinc in the proportion of fifteen parts to one part of the chloride. Dr. Terrillon endorses pyrogallic acid, one part, and starch-powder, three parts. It is an energetic agent that was first advised by Dr. Vidal. Some of the bismuth preparations have been used in late days, such as sub-benzoate of bismuth, and the gallate base of bismuth called "dermatol." This we tried, but in too small a number of cases to give an opinion as to its merits.

The following was used at the Lourcine Hospital for Women, combined with iodoform, and succeeded very well:

℞ Crystallized acid nitrate of bismuth, 1 gramme;
Distilled water (acidulated with nitric acid), 10 grammes.—M.

About the same time we tried sulphocarbol. This product is a mixture of sulphuric acid and carbolic acid, in the shape of an oil, made by M. Charlard-Vigier. When brought in contact with the skin it did not cause any trouble. Employed on chancres it quickly modified them, and it compares favorably with the best of liquid preparations for this purpose. It may be used pure on small chancres, or in a solution of one in ten on larger ones. Last year we tried, without much success, the application of chloride of methyl and acetanilide. We have more confidence in antipyrin, especially in cases where there may be hemorrhage.

To sum up: any of the antiseptics may succeed in the treatment of chancroid, and, as there are so many to choose from, it may be well to formulate a few rules as to the treatment of chancroid, as follows:

First. Great cleanliness should be observed, and asepsis as complete as possible of the environs of the purulent focus. Local hot baths of 40° C. often give good results.

Second. After the local bath, apply caustics in solution or in paste, and continue them until the virulent ulcer has been transformed into a simple wound.

Third. When this effect has been procured, and in the interval of making the caustic applications, apply dressings of weak antiseptic powders. These should be continued until the wound has been cicatrized.

Besides this, advise rest, and remove every condition that will cause any irritation of the sore. If the patient is weak, of course use tonics.

Thus the rapid and complete destruction of the ulceration by caustics, antisepsis, rest, and tonics is the sum total of the treatment of soft chancre.

Gynæcology and Obstetrics.

POST-PARTUM HEMORRHAGE; ITS USUAL CAUSES AND TREATMENT.

CLINICAL LECTURE DELIVERED AT THE PHILADELPHIA POLYCLINIC HOSPITAL.

BY EDWARD P. DAVIS, A.M., M.D.,
Professor of Obstetrics in the Philadelphia Polyclinic.

GENTLEMEN,—The subject of which I shall speak to-night will be better understood if we first inquire what are the factors which prevent serious hemorrhage after normal labor; and the study of these factors will assist us in understanding the reasons for, and the treatment of, hemorrhage.

A potent agent in preventing the escape of blood in all portions of the body is the condition of the blood itself; its peculiar property of coagulation may be taken as a great safeguard against hemorrhage. You will remember that this property becomes apparent only when a solution of continuity occurs in some one of the tissues of the body; thus, in health, blood circulates freely through the vessels, but should a vessel be wounded or injured by the formation of pathological products within its walls, coagulation readily occurs. The blood of the pregnant woman is especially fitted for coagulation by the increased amount of fibrin which develops as gestation proceeds, so that although the separation of the placenta produces an open wound of considerable size, yet the blood which oozes from this wound coagulates in the healthy woman more efficiently than in the patient in the non-pregnant condition.

Again, the arrangement of the muscular fibres of the uterus is such as to provide an efficient series of constricting bands for the sinuses left patent at the separation of the placenta. "These living ligatures" are ordinarily most efficient, and exist in the same perfec-

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Again, the arrangement of the muscular fibres of the uterus is such as to provide an efficient series of constricting bands for the sinuses left patent at the separation of the placenta. "These living ligatures" are ordinarily most efficient, and exist in the same perfec-

tion in no other organ of the body. Further, the lining membrane of the uterus, although normally the seat of periodic hemorrhage, still is possessed of a certain firmness and resisting power which in health prevents the oozing of blood after the uterus is empty. A pathological condition of this membrane, however, removes this resisting power, and a gradual and even serious leakage of blood may result. There are certain mechanical conditions which predispose to the cessation of hemorrhage after normal labor. In many cases the uterus becomes anteverted and even anteflexed, and considerable compression is exercised upon the vessels which traverse the anterior and lateral portions of the uterus. Again, the conditions in the patient's general circulation are markedly changed by the delivery of the child and its appendages; it is as if, in a system of water-pipes through which fluid is forced by a central engine, a considerable area were to be removed from the circuit, and at the same time a considerable amount of the circulating fluid were to be withdrawn. The natural inference would be to reduce the propelling power of the central engine, and this cutting off of steam and lowering of pressure are seen very clearly by the sudden fall in the frequency and force of the heart-beats after labor. During labor, the general activity of the muscular system is so great that the heart shares the vigorous contractions of other muscles. So soon as delivery has occurred, a period of comparative quietude ensues, in which the heart-beat is very greatly diminished. The nervous system, which during labor is in a condition of general activity, rapidly passes into a state of repose, and thus the stimulus to increased circulation is removed. All of these factors, when taken together, constitute a most perfect provision for the avoidance of hemorrhage in the normal patient.

The occurrence of post-partum hemorrhage can be naturally referred to some perversion or lack of the factors which usually render this complication impossible; thus, a depraved condition of the blood predisposes to post-partum hemorrhage. Profound anæmia, the result of long-continued infection by the germ of malaria, the disordered blood which is peculiar to syphilitic infection, and the rapidly disintegrated blood which is present in acute infectious diseases, such as typhoid or small-pox, all predispose to the occurrence of hemorrhage; thus, a woman who aborts during an acute infection is especially liable to severe hemorrhage.

The most common and immediate cause of post-partum hemorrhage lies in the failure of the muscular fibres of the uterus to contract and to close the sinuses left open by the separation of the placenta.

This failure of muscular action may result from a deficient development or impaired condition of the muscular substance itself; thus, a woman whose general muscular development is very deficient is predisposed thereby to hemorrhage. Again, a woman who has borne a number of children, and in whom the muscular structure of the uterus has become thinned by repeated increase and retrograde change, is especially liable to post-partum hemorrhage. Again, a woman who receives during labor a severe shock to the nervous system, or whose nervous system is exhausted by long and difficult labor, is very likely to suffer from paresis or paralysis of the uterine muscle and post-partum hemorrhage. It sometimes happens that the uterine muscle, although healthy, becomes so completely exhausted as to fail utterly to respond to appeals for contraction.

Disease of the lining membrane of the uterus, endometritis, often occasions the most persistent and intractable hemorrhage; there may be present contraction of the muscular substance, the uterus may be emptied of its contents, and yet a persistent oozing of blood occurs which threatens the safety of the patient. Such a woman is very liable to septic infection, either directly from without, or from the absorption of diseased tissue in the endometrium.

A patient who has been safely delivered, but who is the victim of sudden and profound excitement, may suffer from post-partum hemorrhage; in these cases the usual slowing of the circulation does not take place, but the heart, roused to increased activity by nervous impressions, causes a flow of blood from the site of the placental tissue.

The frequency of post-partum hemorrhage has been greatly lessened as a better knowledge of the physiology of labor has become common; thus, it is rare to see in the hands of intelligent obstetricians and in well-appointed hospitals a case of serious post-partum hemorrhage. I have never seen a patient die from this cause, although I have observed serious cases. The method of delivering the placenta by expression has had much to do with this favorable condition of affairs. When time is given to the uterus to separate the placenta by the formation of a thin but extensive clot, and when the uterus, from twenty minutes to half an hour after the delivery of the child, is gently but firmly compressed from before backward in the median line, the placenta is usually expelled without difficulty, and its expulsion is followed by a condition of uterine contraction which rarely relaxes. If, however, an effort is made to expel the placenta immediately after the birth of the child, failure is not uncommon, because the placenta has not become partially or wholly separated. By such ineffectual efforts the uterine

muscle is exhausted, and relaxation after the delivery of the placenta is favored.

Again, the more common use of anæsthetics during labor, preventing exhaustion of the nervous system, relieving the patient of mental pain and distress, favors a better action of the uterus, and thereby a more efficient contraction after labor. A better understanding of the use of the forceps has resulted in diminishing post-partum hemorrhage. By a knowledge of pelvimetry, the obstetrician becomes aware of conditions which render the proper use of the forceps impossible, and is able to spare the patient useless and exhausting manipulation. On the other hand, the wide diffusion of the knowledge of the advantages and methods of securing axis-traction, with the common use of improved instruments, enables the obstetrician to interfere promptly and successfully where a continuance of labor would strongly predispose to post-partum hemorrhage. I may illustrate this remark by describing the following case. A young woman of ill-developed general physique had a tedious first labor; although the pelvis was of normal size, and the child not too great to enter the pelvic brim, the head delayed at the brim of the pelvis from failure in the mother's expulsive effort. When exhaustion seemed imminent, the attending obstetrician applied the Tarnier forceps, and delivered a living child without especial difficulty; the placenta was normally delivered, and the patient seemed doing well. Some eight or ten hours after delivery she was observed to be faint and pallid; there was no external hemorrhage. When her physician arrived, he found the birth-canal filled with clotted blood; when the clots were removed, a profuse hemorrhage occurred from the relaxed and distended uterus. The patient succumbed to syncope and sudden anæmia in half an hour. Here the mistake lay in not employing the forceps as soon as progress had definitely ceased during the labor; if the uterus had not been allowed to become thoroughly exhausted, secondary relaxation and fatal hemorrhage would, in all probability, not have occurred. The intelligent use of the forceps is, then, a distinct factor in preventing post-partum hemorrhage. I need but mention that a faulty method of delivering the placenta, sometimes met with among midwives and ignorant practitioners, not infrequently causes severe hemorrhage. Pulling upon the cord and inversion of the uterus are flagrant examples of such wrong practice. Certainly, as knowledge becomes better diffused, these fatalities must steadily diminish.

As regards the symptoms of post-partum hemorrhage, I will not detain you with a repetition of the classical picture. I may mention,

however, some symptoms upon which you may base an estimation of the imminence of such hemorrhage: most significant of these are the rapidity and the character of the pulse. It has been well said that a pulse above one hundred, after labor, should awaken suspicion in the mind of the attending obstetrician. A rapid compressible pulse should certainly put the practitioner upon his guard. The most frequent physical signs of hemorrhage are the presence of blood about the patient, and the relaxed condition of her uterus. In place of the hard, semi-globular body at the brim of the pelvis, the well-contracted uterus, the hand pressing upon the abdomen finds an indefinitely outlined, doughy tumor, whose boundaries cannot be clearly detected. Such a condition of the pulse as has been described should at once lead to a careful palpation of the abdomen, and to ascertaining the size and consistence of the uterus. The appearance of blood beneath and about the patient should not be waited for to establish a diagnosis of hemorrhage. It is not infrequent to observe cases of concealed post-partum hemorrhage in which no blood may emerge, and yet a profuse bleeding occur within the uterus. Such may be diagnosticated by the condition of the pulse and of the patient's abdomen. Symptoms of lesser importance are the patient's pallor, her thirst, restlessness, dimness of vision, and sensation of lack of air. In cases where bleeding occurs from a diseased endometrium, the condition of the pulse and continuous oozing of blood may be the only symptoms recognizable. Where hemorrhage occurs from lacerations of the genital tract, the uterus may be well contracted and still profuse bleeding be going on. In these cases, again, the only symptoms to be observed are the flow of blood, the rapid pulse, and the patient's general condition of acute anæmia.

The treatment of post-partum hemorrhage consists in remedying those disordered conditions which produce a hemorrhage. First in order of time should be the effort to control as rapidly as possible the contractions of the uterus; the hand should seek the patient's abdomen, and rapid but gentle friction should be made over the fundus. It is to be noted that the uterus will contract more promptly under light but rapid friction than from deeper but slower manipulation. So soon as the uterus is felt to contract, it may be gently but firmly grasped in the hand of the obstetrician, the thumb resting in the median line of its anterior surface, while the fingers cover the posterior wall. Care should be taken to exercise compression in the median line, as the ovaries which are enlarged during pregnancy may be compressed if the hand slips to one or other side, causing sudden and

severe shock. If the practitioner be without assistance, he may inject into the skin of the abdomen, or deeply into the thigh, one or two hypodermatic syringefuls of a solution of ergotin, or fluid extract of ergot. Having cleansed the hand which is not holding the uterus, it should then be introduced into the birth-canal, and if a mass of clot is found filling the vagina and cervix, this clot should be at once removed. If the cervical canal is patulous, the fingers may be inserted to the fundus, and any portion of retained placenta or membranes removed. It is not well to scrape the uterine wall free of clot, for a thin layer of coagulated blood is always to be found closing the sinuses at the site of the placental tissue. If the attending physician can procure the materials for an injection of hot antiseptic fluid, such an injection may now be given to advantage. Boiled water at a temperature of 110° F., a saturated solution of boracic acid, a two-per-cent. solution of creolin or carbolic acid, a one-per-cent. solution of thymol, or a solution of sodium chloride and sodium bicarbonate added to boiled water in sufficient quantity to give a saline taste,—any one of these as most convenient may be employed. The quantity should be several quarts. Such an injection is best given with a fountain syringe, and if the mouth of the uterus is open no intra-uterine catheter is absolutely necessary for such an injection. A free return-flow can be obtained with a simple nozzle, or with the rubber tube of a fountain syringe without a nozzle. An ordinary case of post-partum hemorrhage will yield promptly to such treatment; should, however, the bleeding recur, a strip of iodoform gauze, four inches wide and four feet long, should be carried to the fundus. The uterus should be lightly tamponned with the gauze, a very moderate amount being left within its cavity, and the rest packed with moderate firmness about the cervix and os. If gauze is not at hand, a strip of clean old linen dipped in boiling water may be used instead. This method, to be successful, requires that not too much gauze be used, the strip without doubling being often sufficient; that the strip of gauze or cloth be carried to the fundus, and that violence in introducing it be carefully avoided. There is no one expedient so efficient as this when the uterus has been emptied of clots. If the physician has the assistance of a nurse or intelligent person, the hot intra-uterine injection may be very conveniently employed. In many cases, however, he must dispense with this, and then the prompt use of the tampon, after the hand has emptied the uterus of clots, is of inestimable service.

If the uterus shows a persistent tendency to relax, it may be necessary for the obstetrician to remain beside the patient for some time;

the hand should rest lightly upon the abdomen, and when the uterus is felt to relax, it should be brought back to its condition of contraction by gentle massage. In cases of threatened hemorrhage it is well to delay the application of the binder until permanent uterine contraction has occurred. In obstinate cases the faradic current of electricity will sometimes secure and maintain uterine contraction when other means have failed. Thus, we recall a case in the person of an anæmic, weak multipara whose uterus refused to remain contracted; after the use of ergot, hot douches, the tampon, and massage, a brief application of the faradic current secured prompt and lasting contraction. One electrode should be placed over the uterus, the other over the spinal column at the lumbar region. The current should be as strong as the patient can conveniently endure, each application not usually occupying longer than half an hour.

When the uterus has ceased to bleed, the patient's general condition of anæmia demands attention. A familiar expedient of raising the foot of the bed is of value when efficiently carried out, but to plâce a few books beneath the bedpost, or several bricks, amounts to very little. The most efficient way of raising the foot of the bed in these cases is to place a small table beneath the foot-board; if the patient's hips are to be raised at all, they should be elevated very considerably, so that she lies with the head very much lower than the feet. The injection of hot whiskey and milk, an ounce of each, into the rectum is an efficient method of securing stimulation. The application of warmth to the surface of the body by hot bottles or hot bricks is also of use. The hypodermatic injection of stimulants is of great value; sulphate of strychnine in solution, in doses of one-twentieth to one-tenth, may be employed to great advantage. The hypodermatic syringe may be filled with ether several times, and its contents injected deeply into various portions of the trunk and lower limbs. Where the patient's nervous shock is profound, as evinced by restlessness and great cerebral depression, a hypodermatic injection of one-sixth of a grain of morphine and one one-hundred-and-twentieth grain of atropine will be found of decided value. Auto-transfusion by tightly bandaging the limbs from the extremities towards the trunk is a method worthy of use in severe and protracted cases. The application of heat to the cerebellum should not be neglected; thus a rubber bag filled with hot water, or a hot bottle carefully covered with flannel, should be placed beneath the nape of the neck, where it will give the patient a considerable degree of comfort and stimulation. Inhalations of oxygen are of great value in cases of profound anæmia; whether the forced respiration of the gas

under pressure benefits the patient, or whether the oxygen itself is responsible for improvement in the patient's condition, is not definitely known.

So soon as reaction occurs, the patient may be given a small quantity of hot water, by the mouth, containing brandy, whiskey, or wine. Freshly-made black tea, taken hot, to which has been added a teaspoonful of rum, forms a most stimulating and efficient beverage. Freshly-made black coffee, to which brandy has been added, is also useful. Such stimulants must be given in small quantities. All freshly-made and hot broths, koumys, and raw oysters are available forms of nutritious material which may be taken as soon as the patient's stomach tolerates the presence of food. While champagne may allay nausea and vomiting, it is far inferior in stimulating power to the substances already mentioned.

Post-partum hemorrhage has an allied danger besides death from acute anæmia and syncope, in the predisposition which a patient suffering from hemorrhage has to septic infection. It is well, then, to exercise the most scrupulous care in guarding against this complication; the physician's hands, whatever touches the patient, fluids used for injections, all should be most thoroughly cleansed, and in default of assistance, and when time cannot be spared, soap and hot water are quite sufficient in place of chemical antiseptics. The tampon of gauze may be allowed to remain twenty-four hours if necessary; a free serous discharge will commonly take place, the gauze draining from the uterus as it drains in the abdominal cavity. When it is removed, the vagina should be thoroughly washed out with bichloride solution, one to five thousand, and if the gauze presents the slightest odor of decomposition the uterus should be douched with a creolin solution, two per cent., and an iodoform suppository, containing sixty grains, should be placed within its cavity. Such suppositories may be made with cacao butter, or may be ordered after the following formula:

Iodoform, grs. lx;
Gum arabic,
Glycerin,
Starch, āā grs. vi.
Make one suppository.

If, when the gauze is removed, a decided odor is present, the uterus should be curetted, thoroughly douched, and the iodoform suppository left within its cavity. The administration of small doses of quinine and iron and nux vomica, together with ergot, is useful when uterine

involution is tardy and deficient. Quinine may be administered to advantage as follows:

℞ Quininæ sulphat., ʒi;
Pepsin pur., gr xx.
In capsules, twenty in number. One four times daily with food.

The use of pepsin with quinine in these cases is desirable to obviate nausea, which frequently is a distressing complication of septic infection. There are many cases in which quinine seems to stimulate uterine involution in a more marked degree than the administration of ergot. Where hemorrhage has been excessive and involution is retarded, the following will be found of decided advantage:

℞ Ferri sulphat. exsiccat., gr. l;
Ext. nucis vom., gr. v;
Pepsin pur., gr. xx.
In capsules, twenty in number. One three or four times daily with food.

The convalescence of a patient after post-partum hemorrhage requires the most careful attention to nutrition. Feeding with small quantities of the most easily digested foods is indicated for a week or ten days. Then larger quantities at longer intervals may be substituted. Massage will be found a most useful adjuvant in securing proper involution of the uterus. The abdomen should not be included in this manipulation for the first week or ten days after delivery, the massage being confined to the extremities and back. After this, however, general massage will be found beneficial.

Among the agents not uncommonly employed in the treatment of acute anæmia is the injection into the connective-tissue spaces of a sterilized saline solution. The apparatus required is a hollow needle, considerably larger than the ordinary hypodermatic needle, a piece of rubber tubing two or three feet long, and a glass funnel. Water which has been boiled should be used, and sufficient sodium chloride and sodium bicarbonate added to this water to give it a faint saline taste. The needle, carefully sterilized, should be thrust to a depth of an inch and a half into the outer surface of the thigh, or the regions of the back, or diagonally in the abdominal wall, if the tissues are thick. As much of the fluid as can be induced to enter by gentle rubbing over the point of insertion should then be gradually poured into the funnel. Several ounces of fluid can be introduced in this manner without inconvenience, the resulting tumor, which is often as large as a goose-egg, disappearing readily under continued rubbing. The effect upon the pulse is

immediate and decided. Its frequency is slightly diminished, and its volume perceptibly increased. Such an injection may be repeated several times, if desired.

It may have been noted that nothing has been said regarding the employment of two agents for checking hemorrhage which are often recommended and available in domestic practice,—vinegar and ice. There can be no objection to swabbing the interior of the empty uterus with vinegar, or to squeezing the juice of a lemon upon its surface, if necessary; but in the experience of the writer the means advocated have been quite sufficient and seemed better calculated to afford prompt relief with antiseptic precautions. While acetic acid may be inimical to the growth of many bacteria, yet domestic vinegar is hardly an aseptic substance, nor is the ordinary lemon. The same criticism applies to ice, unless the ice has been especially prepared from sterile water. If we are to be consistent in following the light which bacteriology gives us, we shall do well to limit our employment of agents to those which we are reasonably certain may be made aseptic.

It should take but a few moments' reflection to demonstrate the extreme danger attending the use of preparations of iron in the bleeding uterus. It is but a few years since, in amputations where capillary oozing and secondary hemorrhage from small vessels threatened the patient's life, that surgeons occasionally had recourse to opening the flaps freely and smearing the surfaces with a preparation of iron. The usual results of this procedure were suppuration in these flaps and the death of the patient from pyæmia. The same danger exists in the use of iron in the uterine cavity. Small but firm coagula are formed which may be carried into the circulation, resulting in multiple embolism. If the solution employed be strong, necrosis of the cellular elements of the endometrium and uterine muscle follows the application of the iron, and an irritant necrosis is produced which readily causes suppuration. The employment of a non-irritating, aseptic tampon, such as iodoform gauze, should supersede entirely the employment of corrosive styptics.

Post-partum hemorrhage occurring from a wounded uterine artery or from a blood-vessel in the lower portion of the genital tract requires prompt surgical treatment. If the patient's strength permits and the circumstances are favorable, ligature by the curved needle and aseptic ligature material are indicated. If the patient is too weak to endure such manipulation, and the light be such that the vessel cannot be recognized, the application of hæmostatic forceps, or, better, the employment of the antiseptic tampon, should be chosen. Post-partum

hemorrhage from inversion of the uterus is amenable to treatment, first, by the prompt restoration of the uterus to its usual condition and location, or, if this be impossible, to compression by the tampon and a bandage.

In obstetric literature are found the records of a few remarkable cases of hemorrhage in which a radical operation, such as the removal of the uterus, has been performed for this complication. Such cases must necessarily be very rare, and yet conditions may arise, such as rupture of the uterus, followed by hemorrhage, in which an abdominal incision and the suture or extirpation of the uterus may be indicated. The perfection of obstetric surgery will render these operations comparatively successful if promptly and skilfully performed.

SOME OF THE COMMON DISEASES OF THE FEMALE URETHRA.

CLINICAL LECTURE DELIVERED AT CHARING CROSS HOSPITAL TO THE POST-GRADUATE CLASS.

BY AMAND ROUTH, M.D., B.S.,

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WHILST many of the causes of painful and difficult micturition in women are of reflex origin, due to uterine or ovarian displacements, uterine or peri-uterine inflammation, renal calculus, and so on, there is quite a variety of vesical and urethral diseases, which, owing to the difficulty of making a definite diagnosis, are very apt to be ignored. To-day I propose to discuss briefly a few of the commoner diseases of the female urethra, which is usually supposed to be prone to few troubles as compared with the male urethra, owing to its relatively shorter length, greater diameter, and simplicity.

On the board I have drawn out a list of urethral diseases, and some of them will form the subject of to-day's lecture.

- Malformations . . . { Absence—complete or partial. / Atresia.
- Functional diseases . { Neuralgia. / Spasm.
- Organic diseases . . { Inflammations . . . . . { Simple. / Gonorrhœal. / Of Skene's ducts.
  - Granular urethra.
  - Vesico-urethral fissure.
  - New growths.
  - Vascular caruncle.
  - Prolapse of mucous membrane.
  - Dilatation—urethrocele.
  - Dislocation.
  - Diverticulum.
  - Stricture.
  - Fistula (urethro-vaginal).
  - Foreign bodies.

A few words as to the anatomy may be useful. The urethra is about one and a half inches long, and is a musculo-membranous tube lined by a mucous membrane, with a pavement epithelium near its external meatus and a columnar epithelium elsewhere. It runs from the neck of the bladder downward and forward, approximately in the curve of the true pelvis, and opens anteriorly at the base of the vestibule by the meatus externus. It is supported by being embedded, as it were, in the connective tissue round the anterior vaginal wall, piercing the triangular sub-pubic ligament. There are numerous glands in connection with the urethra. Thus, Littré's glands, which are little more than mucous-membrane reduplications, are described as being about twenty-five in number, running nearly parallel with the urethral floor, and opening into its anterior half to two-thirds by apertures admitting a bristle. The follicles of Morgagni, again, are numerous small racemose glands opening by minute ducts at right angles to the urethral floor. Skene's ducts, which some think are the efferent canals of Max Schuller's glands, are two in number, run parallel to the anterior third or half of the floor of the urethra, and open into it just inside the meatus.

I have had two cases of vaginal cysts which seem, however, to point to Skene's ducts being really the anterior opening of unusually patent Gartner's ducts which run from the Wolffian body in the broad ligaments.

The upper portions of the urethra are formed from the allantois uniting with Müller's ducts, whilst the lower end, lined by pavement epithelium, is formed by the junction of Müller's ducts with the incurving of the uro-genital cleft.

MALFORMATIONS.

Occasionally the urethra is entirely absent, owing to its posterior wall not having been formed. The bladder is then continuous with the vagina, which is practically an undeveloped uro-genital sinus. I have seen this condition exactly imitated by the destruction due to syphilitic ulceration. Plastic operations are here almost impossible, but some good may be done by narrowing the vesical opening into the bladder, and so bringing into use the few fibres of the sphincter vesicæ which are almost invariably present.

Sometimes the lower half of the urethra is absent, or rather its posterior wall, and the effect produced is as if the whole urethra had been drawn up as in retroversion of the gravid uterus, the external meatus being found posterior to the pubic arch. The diminished length of

the urethral canal is, however, easily ascertained by the use of a bulbous-ended sound, whose head is grasped by the sphincter vesicæ, enabling the exact distance from the outlet to be measured by the finger in the vagina. If the hymen is unusually complete, urine is passed into the vagina and passes very slowly out over the hymen. I recently saw a case where the posterior fourchette or anterior margin of the perineum was continued forward almost to the clitoris, causing similar symptoms.

The main symptom of defective formation is incontinence, and this is absolute if the sphincter vesicæ is absent, but slight where the defect is only in the lower parts of the urethra.

Atresia of the urethra may also be complete or partial, the resulting symptom being, of course, retention of urine. This retention leads to a retrograde distention of the bladder, ureters, and kidneys, and causes death *in utero*, or soon after birth, the only escape from this result being where the urachus remains patent and urine passes along it from the bladder through an umbilical fistula. If no water be passed in the twenty-four hours after birth, a careful examination should be made, and if everything appears normal a catheter should be passed, as it not infrequently happens that no urine is being secreted, or it is so loaded with urates that, whilst causing great irritation, it cannot be passed. If no opening into the bladder be found, and especially if vesical distention be evident, a channel must be made and kept open, either in the proper position of the urethra or above the pubes.

FUNCTIONAL DISEASE OF THE URETHRA.

I have named two varieties of functional disease in the table. It is, however, very doubtful whether *neuralgia* exists apart from organic disease either of the urethra itself or of some organ in its neighborhood. I have also seen cases where so-called neuralgia was entirely due to very acid urine, the result of dyspepsia and gout, and speedily relieved by ordinary therapeutic remedies.

Spasm also is usually superadded to true organic mischief in the urethra or elsewhere in the pelvis, and, so far as I have been able to judge, is never present in hysterical retention, which is mainly due to spasm of the sphincter vesicæ muscle, or to a temporary failure of the normal polarity of the vesical muscles.

ORGANIC DISEASES.

The first of this group is *inflammation*.

Urethritis in the female is much less common and much less severe than in the male, owing partly to the urethra's protected position, but

mainly to its shorter length, greater diameter, and straighter course, so that its irrigation is more effectually secured at each act of micturition. There are also fewer lacunæ to harbor irritating or infecting products. The diagnosis of urethritis is easy. After vulvar sponging, pass the finger (not immediately after the woman has micturated) into the vagina, and the urethra can then be felt along its roof to be tender, swollen, and hard, and, on gentle pressure from behind forward, pus is seen to ooze out of the meatus externus, stained, maybe, with blood in acute cases.

Simple acute urethritis is very rare as a primary manifestation, being then due to injury, or sometimes to irritating urine affecting a urethra already granular or dilated, and a course of sedatives, alkalines with purgatives, such as hyoscyamus, buchu, citrate of potash, and Epsom salts, will usually put things right. Direct local measures are rarely called for, but hot hip-baths or hot vaginal douches are often useful. Where simple acute urethritis occurs secondarily by extension of inflammation from the vagina or the bladder, its treatment will be merged with that adopted for the primary disease.

Gonorrhœal urethritis is usually said to be more severe than the simple acute form, but this is incorrect, for a gonorrhœal vaginitis or vulvitis, one of which is almost always the primary manifestation of the disease, may be extremely mild, subacute, or chronic, sometimes being entirely unsuspected by the individual affected. Indeed, it is not rare to find that the only symptom noticed at the time, or recalled afterwards, is the scalding during micturition common to all forms of urethritis at their onset. If there is a history of gonorrhœa or gleet on the other side of the house, the diagnosis is easy, but this history is often not forthcoming, nor is it wise, as a rule, to endeavor to obtain it. The presence of gonococci in the urethral or vaginal pus is also a definite proof of gonorrhœa, and the presence of an acute metritis, salpingitis, and perimetritis is strong presumptive evidence to the same effect, especially if there is a history of such attacks having been recurrent.

In chronic urethritis, a chronic endocervicitis, with a dilated or matted pair of Fallopian tubes, points to a specific origin, but by no means so certainly as was once thought, it being abundantly clear now that a simple vaginitis may cause endocervicitis, endometritis, and salpingitis very readily if the vaginal discharges are allowed to become septic. As a rule, a gonorrhœal urethritis needs no active treatment other than that adopted for the vaginitis, for gonorrhœa in the female attacks almost entirely the generative tract, rarely spreading along the urinary as in the male.

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Very good results are obtained by salicylic acid in fifteen-grain doses every four or six hours, and better still by salol in ten-grain doses. The latter is broken up into salicylic acid and carbolic acid, and carboluria may become evident in a couple of days. The urine should, unless contra-indicated by kidney-disease, be allowed to remain slightly colored by it until the urethritis is cured.

If urethritis tend to become chronic, no matter whether simple or specific in origin, a moderate antiseptic dilatation of the urethra by graduated bougies, such as Hegar's, may be effected; and if this does not suffice to cure, dilatation followed by the application of a rather strong solution of nitrate of silver (ten to twenty grains to the ounce) along the whole urethra, through a Bryant's or Reeves's speculum, will often cure a chronic urethritis at once. It is a good plan to keep in a Goodell's retention-catheter for thirty-six or forty-eight hours after doing this. Some prefer injecting about ten drops of a weaker solution of nitrate of silver, or of sulphate of copper or of zinc, into the urethra, either directly or through a reflux syringe; but the mucous folds and glands are not reached unless dilatation is first adopted, and the catarrh is then apt to recur constantly.

In very obstinate cases of chronic urethritis, where the mucous membrane is much thickened and thrown up into ridges, Emmet's button-hole operation, to be afterwards described, may be required.

Inflammation of (Skene's) Urethral Glands.—These glands are almost invariably involved in urethritis, and often remain inflamed after the urethritis is cured. They cause a fulness at the lateral aspect of the urethral orifice, exude a thin purulent fluid, and lead to dysuria and dyspareunia, and sometimes to vaginismus. The diagnosis has to be made from uncomplicated urethritis on the one side, and from vascular caruncle, if there is much bulging at the urethral orifice, on the other.

The treatment is to pass fine probes into the exposed orifices of the ducts and lay the ducts freely open posteriorly. Immediate union is prevented by freely applying tincture of iodine to the part. A cure is thus almost always effected.

Granular Urethra.—The female urethra is occasionally the seat of patches of so-called granulation, the pathology of which is doubtful. They may be almost painless and simply cause pruritus at the external meatus, or they may be exquisitely sensitive, causing terrible pain during micturition and rendering a complete examination impossible without local or general anæsthesia. Indeed, local anæsthesia, even by injecting two or three drops of a twenty-per-cent. solution of cocaine

on each side of the urethra, often fails to control the agony caused by the passage of a sound along the urethra. Nothing short of dilatation will render the diagnosis certain and at the same time render the local treatment easy. After partial dilatation by a Reeves's or Bryant's speculum or by Hegar's bougies, anæsthesia being carried just as far as the abolishment of the palpebral reflexes, the passage of a sound along the urethra to the point where the granulations begin will at once set up a pelvic reflex. A better way still is to have a strong illuminant, such as electric light, so that the color of the urethral lining may be noted through the gap in the side of the speculum. If no force has been used in the dilatation, so that bleeding has not occurred, a small-sized glass test-tube passed into the urethra enables a good view of its whole length to be obtained if a strong light be present. Skene's endoscope is practically a test-tube with a small movable mirror resembling a laryngeal mirror to throw the light on all points. Having thus seen where the patches of granulation are, the test-tube is withdrawn, a Bryant's speculum introduced, the granulation scraped with a very small ear-curette, and either pure carbolic acid or nitrate of silver solution (3i to ℥i) applied. Sometimes, if the patches can be readily brought into view, the galvanic cautery is the most effectual treatment.

These cases are not common, but when seen are not easily forgotten. I recently saw a lady who had suffered thus for two years; uterine disease was present, and all was put down to that, whilst the urethra was ignored. She then went to a stone specialist, who dilated the urethra and explored the bladder. A general surgeon then thoroughly explored both the rectum and the bladder, but still forgot the urethra. I was more lucky, and, proceeding as above advised, found the granular patches, scraped and cauterized them, and cured her at one sitting, and for the last six months she has had no return.

Vesico-Urethral Fissure.—This is another cause of excessive pain during and after micturition with severe tenesmus. The pain most resembles that in acute cystitis, except that in the latter the pain is decreased after micturition, instead of being increased as in fissure. The pain caused by this fissure at the neck of the bladder may well be compared with that due to a fissure of the anus.

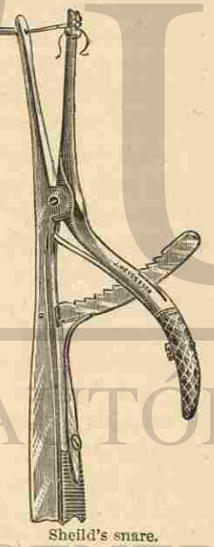
The diagnosis can be pretty accurately made by the effect of passing a hollow bulbous catheter, which is found to cause acute pain just at its entrance into the bladder, known by its bulbous end suddenly escaping into the bladder through the sphincter, and by urine beginning to pass through the catheter. Attempts have often been made to see

the fissure, but this is difficult even with an endoscope. Incising an unseen fissure is also unsatisfactory. Fortunately, however, a cure is almost always produced by dilating the urethra and the neck of the bladder by bougies, up to No. 18 English, unless it is desired to pass the little finger up to the bladder for further diagnostic purposes, when No. 24 is needed. Dilatation beyond this may produce subsequent incontinence.

New Growths.—These are of various kinds,—condylomata, cysts, adenomata, polypi (mucous or angiomatous), fibromata, sarcomata, epitheliomata, etc.

The symptoms are mainly pain during the act of micturition, and a frequent desire to micturate, yet often a dread of doing so resulting in postponing the act. Occasionally there will be slight hemorrhage at the end of micturition, especially if the neoplasm be vascular and near the neck of the bladder. If the tumors be relatively large, some obstruction may take place, and if ulceration occur there will be a constant pinky purulent discharge from the urethra. The obstruction is in most cases due to superadded spasm. If the tumor involve the neck of the bladder incontinence may result.

FIG. 1.



Sheild's snare.

The *diagnosis* of an intra-urethral neoplasm can be made only by careful instrumental examination, including dilatation and the use of some form of endoscope or speculum under cocaine or general anæsthesia. Roughly speaking, the cysts, condylomata, and fibromata are painless and non-vascular, whilst the angiomata and malignant growths are very painful and very vascular.

The treatment consists in the removal or destruction of the growth. Having exposed the growth, it can be dealt with by the curette, scissors, galvanic cautery, electrolysis, or by caustic, bromine, nitric or chromic acid, or acid nitrate of mercury. Some form of Simon's or Reeves's or Bryant's speculum exposes the growth well after its position has been localized by the test-tube or other endoscope, and at the same time protects the rest of the urethra from injury.

If the growth be pedunculated, one of the wire nooses used in ear-surgery, such as Marmaduke Sheild's snare (see Fig. 1) is extremely useful, or a small ear-curette may be sufficient. In either case some caustic should be subsequently applied. After removal a microscopical examination should in all cases be made. Primary or secondary hemorrhage is easily controlled by plugging the vagina.

Vascular Caruncle of the Meatus Urethræ.—[Some account of this common and important neoplasm was given, but, as the subject has been recently so ably and exhaustively treated in these pages by Dr. Herman, the remarks are here omitted.]

Prolapsus Urethræ.—This is a protrusion of the mucous membrane from the external meatus, and is usually secondary to intra-urethral neoplasm, chronic urethritis, or urethral displacement, such as occurs in cystocele and procidentia uteri. The protrusion may be circular, with the orifice in the centre, or limited to one side, with the orifice often then overlapped. The protruded part becomes congested, and often ulcerated and inflamed, and very sensitive. It has to be distinguished mainly from urethral caruncle and the bulging of the orifices of Skene's ducts.

The treatment is to remove the cause and replace the protruded membrane, using astringent applications afterwards. If this fail, the protruded part should be cut off and the edge of the mucous membrane sutured to the edge of the meatus.

Dilatation; Diverticulum; Dislocation.—This is an important group, each member of which is readily mistaken for another, all having many symptoms in common. As, however, the treatment of each essentially differs from that of the others, a correct diagnosis is most important.

Dilatation.—There may be dilatation of the whole urethra, or of any portion of it. Dilatation of the whole urethra is very rare, though cases are recorded where coitus has taken place in the urethra owing to absence or atresia of the vagina. The only two cases which have come under my notice were as follows:

CASE I.—The first was in a girl whose spinal cord had been injured, resulting in paraplegia with paralysis of the bladder. A very large calculus, shown afterwards at the Obstetrical Society of London, became impacted in the urethra and was eventually drawn forward. Here dilatation and incontinence persisted for some weeks. Ordinarily, when the urethra is dilated to admit the little finger, the dilatation is recovered from almost completely in a few hours, though atony of the circular fibres at the neck of the bladder may remain and cause some amount of incontinence for two or three days.

CASE II.—The second case was in a young girl with aggravated

hysteria, who closely simulated renal colic and passage of renal calculi by passing daily into her own bladder by means of scissors (!) small pebbles from garden gravel. She was able thus to imitate admirably, and for a time successfully, the colic, the sickness, the hæmaturia, and subsequently the clicking expulsion of the stone into the china utensil. She was also an inveterate masturbator. Her urethra easily admitted the little finger up to the neck of the bladder, and was much congested and full of large veins and petechiæ.

The anterior third of the urethra may be dilated as a result of external manipulation, or by the presence of foreign bodies or new growths. It may be accompanied by eversion of the edges of its orifice, secondary to chronically inflamed Skene's ducts, or to very chronic procidentia uteri with dislocation of the urethra and bladder.

The posterior third is not often dilated unless there is marked cystocele, or when the functions of the sphincter vesicæ are destroyed by central disease, or by local mechanical distention, such as results from the passage and impaction of a renal calculus. Dilatation of the middle third is much the most common form, and is generally called—or, I should say, *mis*called—*urethrocele*. It would be far better to reserve the term for a dislocation downward of the urethra, just as cystocele is the term given to dislocation downward of the bladder.

Urethrocele (Fig. 4), to use the ordinary nomenclature, is dilatation of the posterior or inferior wall of the urethra in its middle third, the anterior or superior urethral wall remaining in its normal position.

This dilatation is due mainly to an organic or a congestive narrowing of the anterior portion of the urethra, the result of urethral or periurethral inflammation. The floor of the urethra, being loosely supported, yields easily, and its mucous membrane becomes hypertrophied and redundant. More rarely urethrocele is due to injury (bruising and stretching) during the passage of a child's head, or to the prolonged pressure of a badly-fitting vaginal pessary, or to careless catheterism or dilatation, and also to a puckering of the anterior vaginal wall in cases of prolapsus uteri or retroversion with prolapse.

The symptoms of dilatation vary somewhat according to the part dilated. When the anterior third is dilated there are no symptoms apart from those of the cause of that condition. When the upper third is dilated there is always some amount of incontinence, especially in coughing, sneezing, laughing, etc., or even in the erect position.

In urethrocele there is frequent micturition, with a constant feeling that the bladder is not empty. If the urethrocele be large, it may contain nearly a drachm of urine, which speedily decomposes, owing to the

presence of a chronic catarrh, and much irritation and tenesmus result. Patients often find relief from this by passing the finger into the vagina and, by pressure, emptying the urethrocele of its contents after each act of micturition.

The diagnosis of urethrocele has to be made from dislocation of the urethra, dislocation with urethrocele, and diverticulum. The differential points will be noticed further on.

Anterior dilatation becomes obvious when a large sound passes easily at first and is arrested higher up. In *dilatation of the upper third,* urine flows from an introduced catheter before the real neck of the bladder is reached, and the end of the catheter can be felt per vaginam not to be grasped by the urethra at its dilated portion.

Treatment.—Much may be done to remedy minor degrees of dilatation by removing the cause and by astringent applications, as referred to under urethritis. If there be much congestion, local or general depletives are indicated, and vaginal tamponnings to afford rest to the urethra may be occasionally advisable.

If the mucous membrane be redundant it may be scarred with a fine-pointed galvanic cautery, after the urethra anterior to the part affected has been dilated. Emmet has practised an operation which goes by his name, which is especially useful in urethrocele, where the lining mucous membrane is always thickened and redundant. He makes an incision into the bulging portion by a pair of button-hole scissors, the probe-pointed blade being introduced into the urethra, the other blade into the vagina. The redundant portions of the mucous membrane are then drawn through the vaginal wound. If drainage is indicated, the edges of the urethral mucous membrane are stitched to the vaginal edges. If not, the mucous membrane rapidly recovers its healthy appearance and the vaginal wound closes. Where applications per urethram and vaginal support fail to cure, Emmet's operation should certainly be tried.

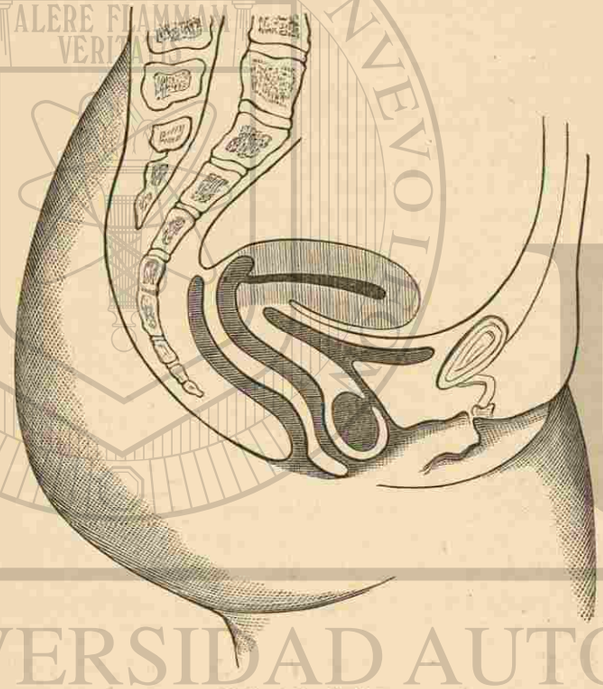
Diverticulum.—Urethral retention-cysts opening into the urethra are by no means rare.

A reference to the diagrams (Figs. 2 and 6) will show that a urethral diverticulum is essentially a urinary pouch or cyst occupying the urethro-vaginal septum communicating with a urethra of normal calibre, usually in the middle third of its floor, by an orifice relatively narrow.

Roughly speaking, the symptoms are progressive discomfort and frequency of micturition, the appearance and growth of a tender lump projecting at the vaginal orifice, the passage of thin irritating pus either at the end of micturition or on pressure, and sooner or later cystitis, etc.

The physical signs are as follows. Per vaginam, a rounded, tender, tense swelling is found in the urethro-vaginal septum opposite the middle third of the urethra. Its smoothness and elastic hardness are characteristic, the rugæ of the mucous membrane over it being obliterated. In some cases the mucous membrane moves freely over the underlying sac, in others it is adherent by inflammation. A sound

FIG. 2.



Urethral diverticulum.

passed along the anterior wall of the urethra goes directly into the bladder, proving that the case is not one of urethral dislocation.

As proving further that these cases are not simple dilatations of the middle third of the inferior wall of the urethra (simple urethrocele), it is found that a large-sized sound will not enter the pouch, but will pass on into the bladder, whereas a small sound or probe will find its way through the narrow opening (which is often valvular) if it be passed along the posterior wall of the urethra. The exact size of the

opening (usually from No. 4 to No. 8 English catheter) is ascertained by a graduated bulbous sound.

With a bougie lying in the urethra the sac is felt to be quite distinct, and even when emptied by pressure it remains a distinct, thick-walled cyst. When full these cysts vary in size from that of a pea to that of a hen's egg.

After being emptied, it only partially refills at the next act of micturition, taking six or eight hours to become tense by exudation from its own lining membrane.

As regards its etiology, I have mentioned numerous ducts and glands which are in connection with the urethra. Any of these ducts or glands may become retention-cysts by closure of their orifices from urethritis, peri-urethritis, or even accidental plugging. As a result of suppuration or rupture, the cyst then opens again into the urethra, and the inflammation is kept up by urine finding access to its cavity at each act of micturition, and, owing to the small and often valvular character of the opening, the distention of the cavity increases. As these diverticula appear to be always opposite the middle third of the urethra, it seems improbable that they can be due to occlusion and distention of Skene's ducts, which are opposite the anterior third, unless Skene's ducts are the anterior termination of Gartner's ducts, which are certainly responsible for many vaginal cysts nearer the cervix uteri. Max Schuller's glands, from which Skene's ducts are usually supposed to lead, seem, however, to be the most likely origin of these retention-cysts.

Pregnancy, with its greater local activity, seems to be a starting-point of their formation, and the impending labor causes them to rupture into the urethra, in that case anticipating the slower effects of a subsequent suppuration if only inflammation ensued.

The treatment varies. If the cysts do not communicate with the urethra they may be dissected out, or merely incised and drained per vaginam, but when urine is obtaining intermittent entrance the following seems the best treatment:

1st. Where urethritis or cystitis already exists, the cyst-wall should be dissected out, and the vaginal wound left wholly or partly unclosed to allow of free drainage.

2d. Where the urine is normal and the urinary passages are healthy, the cyst should be dissected out, the opening into the urethra enlarged to allow of urethral drainage, and the vaginal wound at once closed with wire sutures, which should not enter the urethra itself.

Until union is complete, the vagina should be kept aseptic and the

urine should be drawn off either at regular intervals or by a self-retaining catheter.

In dissecting out the diverticulum the presence of a large bougie in the urethra is helpful, and the contents of the cyst should not be pressed out, lest it become flaccid.

Dislocation of the urethra requires but little explanation after what has been already said.

The four diagrams (Figs. 3 to 6) may further aid the diagnosis. Dislocation of the urethra is often present in procidentia uteri and

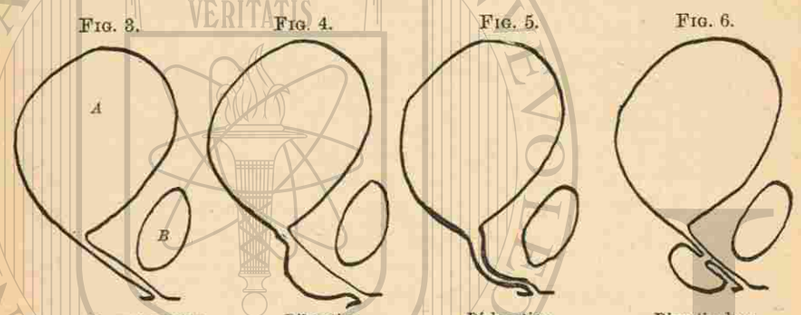


FIG. 3. Normal urethra. FIG. 4. Dilatation. FIG. 5. Dislocation. FIG. 6. Diverticulum.

A, bladder; *B*, symphysis.

cystocele, the sound going at once downward and forward into the bladder lying on the protruded uterus, covered by the inverted vaginal wall. Sometimes, however, cystocele occurs whilst the urethra remains normally situated. The urethra, again, may be dislocated or drawn upward in cases where the bladder is displaced upward by adhesions to the front or back of a growing ovarian cystoma, and in cases of retroversion of the gravid uterus. In such cases the urethral orifice is drawn up with the anterior vaginal wall and is absent from the base of the vestibule, and may be seen only by using a Sims speculum.

In partial cystocele the upper end of the urethra is dislocated, the vesical end then lying at a lower level than the meatus externus. When the whole urethra is dislocated, apart from cystocele, there has usually been a difficult labor as a cause, the advancing head dragging the anterior vaginal wall and urethra away from its attachments. This condition is very apt to persist if the perineum is torn, the anterior vaginal wall being thus deprived of its posterior support.

The symptoms do not differ much from those of dilatation. The treatment consists in supporting the anterior vaginal wall and keeping the uterus at a proper level by a suitable pessary, or by tamponning

the vagina, and, if necessary, by restoring the integrity of the perineum by surgical means.

Lack of space allows me to say only a few words on *stricture*, which, as compared with stricture in the male urethra, is less frequent and important. It is mostly produced by peri-urethral inflammation, or by cicatrices the result of injury, or following operations by caustics or the cautery.

Frequency of, and slowness and difficulty in, the act of micturition are the main symptoms, with retention by superadded spasm or congestion.

The diagnosis needs only a series of bulbous-ended sounds to make the exact position and extent obvious.

The treatment consists in dilatation by bougies, followed by the use of a retention-catheter, such as Goodell's, and, if need be, the division of any cicatricial bands by some form of urethrotome. Atrophic contraction, such as is seen in long-standing vesico-vaginal fistula, requires patient and gradual dilatation to allow the tissues to become accustomed to the changed conditions after the cure of the fistula.

If these imperfect remarks should lead those present to study with more thoroughness urethral conditions in the female, much good may ensue, for from considerable experience in gynæcology I am constantly meeting with cases of urethral diseases the existence of which has been absolutely ignored by men whose experience in female pelvic diseases is far greater than my own.

It almost seems that if, on inspection, no gross lesion, such as vascular caruncle, is discovered, it is at once assumed that the urethra must be healthy, and that the cause of the dysuria, etc., must be looked for in the condition of the other pelvic organs. That common urethral diseases should not be thus overlooked is the object of this rather hurried sketch of the subject.

ENDOMETRITIS.

CLINICAL LECTURE DELIVERED AT THE MEDICO-CHIRURGICAL HOSPITAL.

BY WILLIAM EASTERLY ASHTON, M.D.,

Professor of Gynæcology in the Medico-Chirurgical College of Philadelphia.

GENTLEMEN,—The patient I bring before you this morning presented herself at the dispensary for diseases of women with the following history:

Mrs. E. S., white, aged twenty-three. She has been married two years, but has never been pregnant. She enjoyed good health up to within six months of marriage. At this time, while carrying a large bucket full of water up a flight of steps, she felt a sudden and severe pain in the lower abdomen. She was subsequently confined to her bed for several days, and has not been in good health since. Menstruation, which had previously been normal, now began to be more or less profuse, and at the present time the flow continues from eight to ten days. The great loss of blood occurring at the periods has gradually made her weak and anæmic. She also suffers with a leucorrhœal discharge, profuse in amount, and opaque white in color. There is a feeling of pain or weakness in the lumbo-sacral region, accompanied with a bearing-down sensation in the pelvis. The bowels are constipated, and there is a feeling of pressure upon the upper part of the rectum. The appetite and digestion are poor, and she is more or less nervous and irritable.

Introducing now my index-finger into the vagina, I find there is no indication of pelvic disease, but the uterus, although movable, is retro-displaced, and by examining well up in the posterior cul-de-sac, the fundus is felt resting upon the rectum. Exposing the cervix through the speculum, those of you who are near will notice that the neck of the uterus is slightly enlarged, the os patulous, and the altered uterine secretions are seen oozing from the cervical opening. Taking into consideration the evidence offered by the subjective and objective symptoms in this patient, our diagnosis is a retro-displacement of the uterus

complicated with an endometritis. We have, therefore, two conditions to deal with: first, the displacement, which is the primary disease, and, second, the endometritis, which is a secondary result.

Endometritis, as you already know, is the term applied to an inflammation of the uterine mucous membrane. In the light of modern investigations upon the pathology of pelvic disease, endometritis becomes at once a subject of vital importance, as it is the starting-point, in the majority of instances, of all inflammatory conditions affecting the pelvic organs and peritoneum. With a knowledge, therefore, of this fact before you, you will not only be better able to appreciate the necessity for holding clear and definite ideas upon the causes of this disease and its results, but you will also keep well in mind the urgency, in some of its varieties, for prompt and radical treatment. In dealing, therefore, with endometritis, we must forget the teaching of the past, and remember only the lessons taught us through the advances made in the pathology of pelvic diseases.

Inflammation of the endometrium may be conveniently divided into four varieties,—the congestive, the constitutional, the septic, and the gonorrhœal. The first two varieties are usually spoken of under the term "simple endometritis," and they are the least dangerous forms of the disease, as they seldom cause grave pelvic complications, unless improper treatment be employed for their relief. The septic and gonorrhœal varieties, on the other hand, are dangerous to life, or, if they do not end fatally, the health and future usefulness of the patient are, in many cases, forever sacrificed.

In studying the etiology of endometritis, we find that it is the result of micro-organisms infecting the mucous lining of the uterus. In the septic and gonorrhœal forms, this infection is the direct cause or primary condition, while in the congestive and constitutional varieties, the inflammation, which is also due to the development of germs, is secondary to an exciting cause. To make this clear, let me call your attention to the fact that normally the uterine cavity and cervical canal contain micro-organisms, but it is only through the result of an exciting cause that the germs develop and produce disease. Remembering, therefore, that endometritis is always caused by the development of micro-organisms, but that the germs are the secondary and not the primary condition in the simple forms, we are in a position to appreciate the pathology of this disease. The existing causes of the congestive form are, displacements of the uterus, cold, narrowing of the cervical canal, subinvolution of the uterus, lacerations of the cervix, the exanthemata, constipation, sexual excess, and pel-

vic tumors and adhesions. The result of all these conditions is pelvic congestion, and following this the uterine secretions become more profuse. To make clear to you the process by which disease is produced under these conditions, I cannot do better than quote to you from an article by Dr. Laplace[1] upon "Endometrial Micro-Organisms." He says, "It is plain that the mere presence of the micro-organisms does not suffice to constitute disease. Disease is the reaction upon the system, local or general, or both, resulting from the developing organism. In the uterus the normal secretions are a poor culture medium for germ life, and at the same time keep the micro-organisms at a distance from the blood-vessels. If given the proper opportunity, however, if furnished with blood or serum retained any undue length of time within the uterine cavity, micro-organisms develop therein with as remarkable rapidity as they do upon artificial culture media in the laboratory. Now the conditions will have changed, and enormous hordes of bacteria soon develop from those already present, and infect the tissues. In our observation, judging from the reaction of tissues under the influence of developing bacteria elsewhere, we would say that cold is, perhaps, the most frequent cause of the initial process; the congestion which soon follows the action of cold upon the tissues being familiar to us all. Next follows the exudation of serum, which is soon contaminated by the bacteria in the neighborhood; these finding their most favorable soil develop rapidly, producing a chemical irritant or ptomaine which is the decomposition of the serum incident to their growth; this acts as a direct chemical irritant which keeps up indefinitely the irritated condition of congestion and hence hypernutrition of superficial cells,—proliferation of cells resulting, which cells naturally find their protoplasm inoculated from the first with the bacteria under whose impulse they developed."

Referring for a moment to the history of our patient, we find that she dates her troubles back to six months before marriage, when she was seized with a sudden and severe pain while carrying a heavy weight. Now, what happened at this time to cause her subsequent symptoms? The explanation is, I take it, a simple one, and bears out most strikingly the process by which congestive endometritis is produced. In all probability her bladder at the time of the accident was full of urine, and the extraordinary effort she made in lifting the bucket caused an acute retro-displacement of the uterus. This malposition

[1] The Relation of Micro-organisms to the Diseased Endometrium. The American Journal of the Medical Sciences, October, 1892.

having once occurred never properly restored itself, and she has since suffered with a backward dislocation of the womb. Now, a retro-displacement of the uterus must of a necessity interfere with the circulation of blood in the organ, and if the condition be a permanent one, chronic uterine congestion follows.

Passing now to the constitutional variety, we find that the disease occurs in scrofulous and anæmic women, and also in patients suffering from phthisis. In these cases the endometritis begins as a simple hypersecretion of the glands of the uterus, thus producing a culture medium for the development of the micro-organisms found normally within the uterine cavity and cervical canal. The causes of septic endometritis are—sepsis following labor or abortion, sloughing polypi, malignant degenerations of the uterine mucous membrane, and the use of dirty instruments or their improper application in the diagnosis and treatment of intra-uterine disease. The common practice among physicians of making applications to the interior of the uterus in their offices, and the use of the sound, are, in my judgment, dangerous both to the health and life of their patients, as septic endometritis with pelvic complications is likely to follow. The larger my experience grows the more convinced I am that a good and safe rule to follow is, *never enter the uterine cavity or the cervical canal unless it be done under an anæsthetic and with strict operative asepsis.* I cannot too strongly impress this upon you, for irreparable damage is often done through ignorance or forgetfulness of this fact on the part of the physician. It may be that I take too radical a view upon the treatment of intra-uterine disease, yet I feel it is better to err upon this side of the question than to teach you the opposite one.

The gonorrhœal or specific variety of endometritis is caused by the gonococcus of Neisser. This form of the disease is especially dangerous to life on account of the rapidity with which the specific inflammation spreads to the pelvic structures. I do not believe in the theory of a latent gonorrhœa in the male causing a specific endometritis in the female years after the original attack. The assertions of Noeggerath and also those of Tait are not borne out by facts. The histories of patients they bring forward to uphold this theory show in the majority of instances post-puerperal sepsis and not a gonorrhœal infection. And, again, if we believe in latent gonorrhœa, how are we to explain the fact that such a vast number of marriages are followed by conception?

The symptoms of endometritis depend upon the form of the disease and the cause which produces it. In the simple varieties the

inflammation in the majority of instances comes on slowly and is chronic in character. The subjective symptoms complained of are caused, as a rule, by the various conditions producing the disease. Thus uterine displacements, lacerations of the cervix, pelvic tumors and adhesions, etc., are responsible for the symptoms, and not the endometritis, which is a secondary condition. On the other hand, however, the profuse uterine discharge, the digestive symptoms, and the tendency, in some cases, to sterility and abortion are due primarily to the disease of the mucous membrane. In the gonorrhœal and septic forms the inflammation begins acutely, and there are present constitutional symptoms, as indicated by a chill, elevated temperature, and increased pulse-rate. If the disease extends to the pelvic organs, there are also present the evidences of a local or general peritonitis. In cases of chronic endometritis, septic in origin and following labor or abortion, we find that uterine hemorrhage is occasionally a symptom of the disease. This is due either to the uterus being incompletely emptied of its contents or to pathological changes taking place in the decidual membranes.

The physical signs in chronic cases of endometritis are practically the same in all forms of the disease. With the speculum we see the characteristic discharge coming from the os uteri. The uterus upon palpation is not, as a rule, tender to the touch nor is it enlarged unless the disease be associated with a metritis or the uterus occupied by a foreign growth. If the pelvic organs have become infected, an examination of these structures will reveal their condition. Again, the uterus may be found displaced or the cervix lacerated, but these as well as other conditions are complications or causes, not the disease itself.

In acute inflammations of the endometrium, on the other hand, the uterus is enlarged and tender to the touch. The uterine secretions are profuse and the cervix is swollen and congested. These conditions are not only present in the septic and gonorrhœal forms but also in the congestive variety, if the disease be sudden in its outset.

The prognosis of endometritis depends upon, first, its cause, and, second, as to whether or not the disease has extended to the pelvic organs. The gonorrhœal and septic forms are actively dangerous to life, while the other varieties seldom of themselves tend towards a fatal issue. Again, the prognosis is influenced, in some cases, by the promptness and thoroughness with which local treatment is instituted. Of course in the constitutional forms we cannot hope to materially influence the disease while the cause is actively present. In a general way we may safely state that there is no natural tendency towards a cure

in cases of chronic endometritis and that the simple forms are not of themselves necessarily dangerous to life; on the other hand, however, the septic and gonorrhœal varieties frequently result in death or at best produce chronic invalids.

The treatment of endometritis, based upon the germ theory of disease, is directed first to the cause and second to the removal of the results of the inflammation upon the uterine mucous membrane. In the constitutional varieties of the disease we must not lose sight of the fact that the general condition of the patient is primarily responsible for the pathological changes taking place within the cavity of the uterus. Hence our first effort must be to correct those vices of the constitution to which I have referred in discussing the etiology of endometritis. It would be useless, therefore, in these cases to attempt a cure by means of local treatment. If, however, after the patient's general health has been restored, the uterine secretions still continue to be profuse, it is an indication that the micro-organisms have infected the deeper structures of the mucous membrane, and hence the local inflammatory condition can only be relieved by direct treatment to the endometrium. Again, these principles of treatment hold good also in the congestive forms of the disease when the outset is sudden,—as, for example, exposure to cold and the like. Here the acute congestion increases the uterine secretions, thus favoring the rapid development of germs. If, under these circumstances, the congestion is relieved by treatment, no damage is done, and the endometritis subsides. On the other hand, should the congestion become permanent, the endometrial inflammation assumes a chronic condition, and we must, therefore, restore the status of the pelvic circulation before directing our attention to the local uterine disease.

In those cases of endometritis congestive in form and coming on slowly and running a chronic course, there is always a definite cause to be found. For example, if the uterine congestion be due to a retrodisplacement of the uterus, to a lacerated cervix, or to a pelvic tumor, the indication is to remove the cause, and at the same time, or subsequently, treat the local disease within the uterine cavity.

In the septic and gonorrhœal varieties the disease begins acutely as a primary local condition. Therefore we at once direct our treatment to the endometrium, and by thus removing the cause we at the same time cure the disease. The local treatment of endometritis is of great importance, and you must have clear and well-defined ideas upon the subject if you hope to cure your patients. You will recall what I have already told you in reference to the danger and uselessness of

routine intra-uterine medications. This method of treatment is dangerous, because it cannot be carried out under strict asepsis, and it is useless in chronic cases, because the deeper structures of the mucous membrane are infected, and we are unable to exert any influence upon them with applications made directly to the surface of the endometrium. Therefore to cure the disease we must remove the infected tissues, and this is best accomplished by means of the curette. The operation of curetting consists in (1) dilatation of the uterus with the heavy dilators of Goodell; (2) thorough removal of the mucous membrane with the curette; (3) flushing the uterine cavity with an "acid" solution of corrosive sublimate (1 to 2000); (4) the application of Churchill's iodine to the interior of the uterus; (5) introducing into the cavity of the womb a narrow strip of iodoform gauze and leaving it in position for twenty-four hours.

I shall not have the time during my clinic this morning to enter into a discussion of the antiseptic and operative details of this method of local treatment, therefore we will pass at once and briefly to a consideration of the contra-indications for the operation. Curetting of the uterus should never be performed if an inflammation exists of the ovaries, tubes, or peritoneum. Therefore, in all cases where curetting is indicated we must first carefully examine the pelvis for the presence of disease before undertaking the operation. A neglect of this precaution will not only bring discredit upon you, but in many instances will be the direct cause of a fatal peritonitis resulting.

Before dismissing you I will refer for a moment to some special points in the treatment of acute septic and gonorrhœal endometritis. Grave pelvic complications can only be prevented in the gonorrhœal form by prompt and effective treatment. If, therefore, during the course of a specific inflammation, the uterine cavity becomes infected, you must dilate and curette the uterus. This operation will at once remove the diseased mucous membrane and at the same time destroy the germs of infection, thus reducing to a minimum the chances of pelvic disease resulting.

In septic cases following abortion or labor the uterine cavity is irrigated three times during twenty-four hours with an "acid" solution of corrosive sublimate (1 to 4000). If at the end of that time the symptoms have abated, the irrigation is kept up twice daily for two or three days. Should, however, the symptoms at the end of the first twenty-four hours show no signs of improvement, the uterus must be then thoroughly curetted.

PROLAPSUS OF THE OVARIES, CHRONIC OÖPHORITIS, AND PELVIC PERITONITIS; ABDOMINAL SECTION.

CLINICAL LECTURE DELIVERED AT THE LONG ISLAND COLLEGE HOSPITAL.

BY ALEXANDER J. C. SKENE, M.D.,

Professor of Gynæcology, Long Island College Hospital, and Dean of the Faculty.

THIS patient is twenty-nine years of age, and has been married five years. She has suffered more or less from dysmenorrhœa, and is sterile. The pain, which is severe, begins some time before the menstrual flow, continues during the flow, but in a much modified degree, and persists in a severe form for about three days after the flow. In the interval she has continual pelvic tenesmus. She has been treated for a year before coming to us. For several months she has been treated here as an outpatient for diseased ovaries and tubes, but without getting much relief. It is the opinion of my associates (and I agree with them) that we can do little to relieve her except by removing the offending organs. I believe I have often enough said to you that we should not hastily open the human abdomen, or attempt to remove any of the pelvic organs for any degree of suffering without first giving general treatment a fair trial. That has been done here, and without success. We have given her some relief, it is true, but it was only temporary, and she has come back again. I will now operate on her.

You will observe that we arranged the patient and performed the operation in the method which has already been described to you on previous occasions. The operation was a double ovariotomy. You will notice the difference in size between these ovaries; one is nearly twice as large as the other. The rule is that the ovary becomes very much enlarged, and finally undergoes a kind of atrophic degeneration and hardening. One of these has advanced further in this process than the other. In the large one there is considerable swelling and œdema; the other is small and atrophied, so that it is in the condition resembling that of a contracted kidney after a long-existing interstitial nephritis. Still, both of these ovaries look as though they hardly ought

to have been removed. I am always very critical about ovaries that have been removed. Had this woman been differently circumstanced and had not so much been dependent upon her, we might still have waited and tried other means. If we had waited until both the ovaries became atrophied, then this patient would have been in the same position as a woman of fifty-five or sixty, who has passed the menopause and the final atrophic changes in the ovaries. But this takes a long time, and, as she is a poor woman dependent on her own exertions for a livelihood, we decided that it was better to operate.

Again, both of these ovaries were prolapsed. An inflamed ovary which remains in its normal position behaves infinitely better than one in the same condition that has become prolapsed, so that when a dislocation exists in addition to the chronic inflammation, that is a strong argument for removal. In this patient we found the ovaries in the most dependent part of the sac of Douglas. So marked was the prolapse that it was a little difficult to tell which was the right and which was the left one. They were close together, the one a little behind the other, and we could find them only by pushing them between the examining finger and the sacrum through the vagina. Taking all the circumstances into consideration, I think we were justified in giving this patient the relief which I believe will follow. I see no reason why she should not get well. We kept the abdomen open a little longer than we sometimes do, but during that time it was well protected by sponges. You noticed how the patient was protected by a rubber sheet, about which I have already spoken to you. When you take that rubber sheet off, you leave her garments and everything perfectly dry and clean, and do not have to change the clothing. If pus or other animal material gets on the clothing and the patient is put to bed, you have all the conditions present that favor a rapid decomposition.

A word with reference to the use of sponges. You will remember I told you that in former times we used to operate in a very high temperature, almost as high as 100° F., which was very unpleasant, both for the operator and for the patient, but it was then deemed necessary, because the abdominal cavity was exposed. Now we protect the patient from shock with hot sponges,—just as hot as you can comfortably handle them. You should also see that your hands are warm. I have often, during large operations, crowded in a considerable number of big sponges, so that the abdomen was distended by them. Besides protecting the patient from shock, these sponges check the oozing and keep the intestines from coming down. We keep the sponges hot by putting a smaller pail inside of a larger one; the inner one is thoroughly sterilized

and filled with sponges, and the outer one is filled with hot water, and a lid is put over both. In cases where some of the septic material has got into the abdominal cavity, you can take a quantity of sterilized water at the proper temperature and flush the whole abdomen, and this will prove a good stimulant as well. I have resorted to that measure, and shall do so again in difficult cases where the whole abdominal cavity is open to possible contamination; but if you simply have a little leaking from the sac, you can do quite as well by carefully sponging it.

In the operation I did here to-day, the difficult part of it was not seen by you,—namely, the adhesions. An ovariotomy without firm adhesions is a very simple thing,—so simple, in fact, that a double ovariotomy has been done in a few minutes; by some in seven or eight minutes; I am inclined to think it has been done in this institution in less than ten minutes. When there are adhesions, it becomes much more difficult. To-day I encountered adhesions. The omentum was adherent to the abdominal wall as far down as the pubes.

With reference to breaking up adhesions, I have a rule which I have established for myself, and it may be of some use to you. We encounter sometimes both old and recent adhesions. Recent adhesions can generally be broken up with perfect safety. When I can break up an adhesion with the side pressure I am able to exert with my middle and index fingers, I consider it safe to do so, but I do not care to exert any greater force than that. I much prefer to make a little larger opening and find such adhesions, divide them, and thus work my way down to the ovaries and tubes.

This operation looked easier to you than it was, yet it was not easy. I have always been unwilling to show my class a very easy operation of this kind, because it gives them the wrong impression. Opening the abdominal cavity is an awful necessity, and I start out with the idea that every case is going to do badly. In every other relation of life women are reliable, but you cannot tell how they are going to act after such an operation, and you must do your very best.

As far as dressing the wound is concerned, I find the following very simple and efficient. I take cheese-cloth, which is cheap and clean, and saturate it in a solution of carbolic acid and glycerin (1 to 8); the glycerin takes away the caustic properties of the carbolic acid. The cloth is wrung out immediately before it is applied. It is spongy and porous, and any oozing will be mopped up and disinfected. Decomposition cannot take place, and the surface is kept moist and not at all irritated. Then, to protect it from outside influences, I apply sterilized cotton, which answers every purpose.

CONTRA-INDICATIONS TO MINOR GYNÆCOLOGICAL OPERATIONS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY HENRY C. COE, M.D., M.R.C.S.,

Professor of Gynæcology at the New York Polyclinic; Gynæcologist to the New York Cancer Hospital; Assistant Surgeon to the Woman's Hospital; Obstetric Surgeon to Maternity Hospital.

I WISH to direct your attention to-day to a subject which is of great practical importance to you, although I fear that you hear little said about it. I shall illustrate it by reference to two or three patients upon whom I operated several months ago, and who are not as much benefited as I expected they would be. It will be interesting to try to discover the reason in each instance. Unfortunately, by reason of your short stay here, and the fact that you see only occasional operations by individual surgeons, you are not in a position to follow the cases afterwards. You see an operation performed rapidly and smoothly by an experienced hand in a few minutes. With trained assistants and all the appliances of a well-appointed hospital there is no hitch, and the procedure seems to be simplicity itself. So it is when you know how to do it. The wound looks perfect, the dressing is applied in a scientific manner, and you are merely told a few days or weeks later that the result was good and that the patient was discharged "cured." Now, this is a very imperfect picture of the case, as you will find when you come to do the same operation in private practice. You have learned nothing about the precise indications for the operation, the long course of preparatory treatment which may have been necessary, and the condition of the patient six months or a year later. After all, the best test of the value of a minor gynæcological operation (I exclude cases of abdominal section) is not the appearance of the parts when the stitches are removed. The patient does not regard these matters from a purely æsthetic stand-point. She cannot share the surgeon's enthusiasm over

a "beautiful result" if several months elapse and she does not experience the promised relief. The perfect restoration of a torn cervix or perineum does not appeal to her as it does to the artistic eye of the operator or the spectator. She would never have consented to the operation unless she had confidently expected that certain definite symptoms which had become unbearable would be eliminated by it. These may have been one or many—pain, hemorrhage, sterility, or other troubles which prevented her from fulfilling the duties of a wife and mother. Now, when these symptoms remain unchanged after the operation there must be some reason for it. Either there is a failure from an anatomical stand-point, or the case was an improper one for operation. Failures of union are, or should be, rare with our present technique, hence we may regard the latter cause as the more common. I hold that it is quite as important for you as practising physicians to learn when *not* to operate as it is to become perfectly familiar with the details of gynæcological operations. Unless the patient is thoroughly prepared for the operation she will not be benefited by it. In a general way it may be said that surgical interference should be the last, not the first, step in your treatment. The patient will ask, "Shall I be entirely cured by the operation, so that I shall never need any more treatment?" Be exceedingly careful how you answer this question. The manner in which you do it stamps you as a conscientious or a careless gynæcologist. I do not see how any man can give a positive promise of cure in a case of pelvic trouble, since the condition is so complicated. How seldom do we meet with a laceration of the cervix without an accompanying laceration of the perineum, subinvolution, and endometritis! The symptoms which we attribute to the former lesion may in reality be due to one of the accompanying troubles, so that an operation would give little, if any, relief. The same result is seen after removal of diseased appendages when the uterus remains retroflexed and fixed by adhesions. In every case you must make up your mind which is the principal trouble—the one that gives rise to the symptoms from which the patient seeks relief. It may not be of pelvic origin at all. Be careful about confidently locating the seat of an obscure neurosis in a slight laceration of the cervix when it may be a fissure of the anus. I have a private patient now under observation who illustrates this last error perfectly. I have operated upon her twice, repairing a bilateral laceration of the cervix and an extensive injury to the pelvic floor, and finally tying off several hemorrhoids. When examined six months later the result was perfect from an anatomical stand-point, but clinically there was little, if any, relief from the backache and dragging sensations of

which she complained originally. Finally I have come to the conclusion that the pelvic symptoms are reflex, since I can find no cause for them in or around the uterus. The lady's bowels are obstinately constipated, and defecation is attended and followed for several hours with severe throbbing pains in the rectum, which are also transferred to the uterus and ovaries. She must have proctitis or ulcer, or an undiscovered anal fissure, and I propose to etherize her and ascertain. There is a peculiar spasm of the sphincter, which I have noted in other cases of this character. [The above opinion was justified a month later, when the patient was thoroughly examined under chloroform-anæsthesia. The rectum was normal, but the grasp of the sphincters, especially the internal, was so powerful that it was literally impossible to paralyze them without nicking some of the muscles. The ultimate effect of the operation entirely confirmed the diagnosis. The patient has now a regular daily evacuation of the bowels for the first time in several years, and there is considerable relief of the pelvic pains.]

With this brief introduction, let us look at the patients.

Case I.—This patient illustrates well what I have been saying. She was operated upon six months ago, lacerations of the cervix and perineum having been repaired in the usual manner; the flap-splitting method was adopted in the perineum. She was discharged at the end of a month with the parts in a perfectly satisfactory condition, and received the usual cautions against over-exertion and too early resumption of her matrimonial relations, which, as you will infer, are seldom heeded. Now, let us see how the operations have stood the test of time and the various vicissitudes incident to her condition. The perineum looks all right, but when I introduce my finger I find that it has little supporting power. This is my criticism of Tait's operation as compared with other methods of repairing the perineum—it is an easy and ingenious method, but the ultimate results are not all that could be desired. The cervix, as you see, is anatomically perfect; at the first glance you can hardly tell that it has ever been torn. The os is of normal size, the uterus is small and in good position. Why, then, does the patient return for treatment? She complains of a constant pain in the right side, the same that she had before the operation. On examination I find a prolapsed ovary, not much enlarged, but quite tender. Its presence vitiates the effect of both operations, so far as the relief of pain is concerned. It was of course recognized before the operation, but its importance as the cause of the symptoms was underrated.

Case II.—The operation in this case was a surgical failure, as you

can readily see. The patient had a bilateral laceration of the cervix, with such extensive erosion and induration that I suspected commencing epithelioma, and accordingly I excised all the diseased tissue, removing a large wedge or core, so that I was obliged to insert a stem in order to maintain patency of the cervical canal. There was no sepsis or rise of temperature, but when I came to remove the sutures at the end of twelve or fourteen days the opposed surfaces simply fell apart, showing that there had been no attempt at union. A more careful inquiry into the patient's history developed the fact that a syphilitic taint existed. Under the usual specific treatment the wound began to granulate rapidly, and finally healed as you see it now, presenting a much better appearance than was hoped for at the outset. I have several times noted this same retardation of the healing process in syphilitics. The patient had an attack of pelvic inflammation after leaving the hospital, so that she is now much worse than before. I had intended to perform a second operation upon the cervix, but of course I cannot think of it now. I show the patient simply to emphasize the fact that before performing even a minor gynæcological operation one must be sure not to overlook any obstacle to success, whether it is a general or a local complication.

You have examined a young woman here who has a bilateral laceration of the cervix and needs an operation. There is no local contra-indication, and, after a superficial examination, I sent her to the hospital, expecting to operate at once. But a careful review of her history revealed the fact that she was an epileptic, evidences of cardiac trouble were discovered, and after keeping her under observation for a fortnight I decided that it would be unwise to subject her to the risk of anæsthesia, since the beneficial results of the operation would probably be either slight or *nil*. These are just the sort of cases in which a wise conservatism should temper our surgical enthusiasm. In private practice the proper appreciation of the contra-indications to an operation are even more important than technical skill. If you are convinced that it is not right to perform it, no mere pecuniary considerations should be allowed to influence you. If another surgeon differs from you and decides to operate, let him do it.

Case III.—Now, here is a patient upon whom I would not hesitate to operate at once. Why? She has a deep bilateral laceration of the cervix, which originally extended out into the vaginal fornix, resulting from the use of high forceps, "not wisely, but too well." The pelvic floor affords no support whatever, so that the heavy uterus sags downward until the cervix is almost at the vulva. The uterus is

movable and can readily be replaced; there is no tenderness in or around it, no prolapsed ovary. The patient is in fair general condition, with good muscular tone, and there is every reason to believe that the simultaneous repair of the cervix and pelvic floor (anterior and posterior colporrhaphy) would not only be successful anatomically, but would relieve the dragging sensations of which alone she complains.

Now, I have only time to make a few short deductions. How shall we decide in private practice when to advise an operation, and when shall we perform it? I do not think that it is always possible to arrive at a decision at the first interview. It requires only a superficial knowledge of gynæcology to recognize an extensive lesion of the cervix or perineum, a stenosis of the os externum, or a visible neoplasm, but the recognition of complications appreciable only to the practised touch is not so easy. It may be a great disappointment to the patient to tell her that she has some parametritis which must first be eliminated before it is safe to operate, and if you hint at a possible delay of weeks or months she may go to another gynæcologist, but you will have done your duty, even though from a selfish stand-point you may seem to be working against your own interests. I can assure you from bitter experience that it pays better to refuse to operate on a doubtful case than to yield to the solicitations of an impatient woman or to operate prematurely against your better judgment. If your patient is so fortunate as to escape a fresh attack of pelvic inflammation which may permanently cripple her, be sure that she will not have that benefit from the operation which she confidently expects, and which perhaps you were so unwise as to positively predict. Do not be guided by the wishes of the patient alone, nor by the opinions of others. If you are to bear the responsibility of an operation, make up your mind only after a careful review of the history of the case and a thorough examination, repeated if you have any doubt at the first. Analyze the symptoms, decide which are the most important, and seek to assign them to their true source, which may not be the prominent lesion that at once arrests your attention. Do not be on the constant lookout for reflex neuroses due to laceration of the cervix, for they are rare; if ovarian trouble is present the ovaries are far more likely to be the cause of such symptoms. Above all, be sure that if a laceration of the cervix or perineum is present it is sufficiently marked to require an operation. I cannot lay down any positive rules to determine this fact. It is largely a matter of conscience. If there be a subacute pelvic inflammation, prolapsed, tender ovaries, especially if they are adherent, or tubal disease, you will certainly not operate—at least with the expectation of curing the patient.

If the uterus is displaced and fixed by adhesions, you will certainly neither cure the displacement nor relieve the symptoms by restoring the cervix and perineum to a state of anatomical perfection. The operation will be an æsthetic procedure, nothing more. Finally, look well to the patient's general condition. If her entire nervous system is at fault, any operation may be more harmful than beneficial; or she may have some serious visceral affection, such as to render even a minor operation unwarrantable.

In conclusion, I would again remind you that the ultimate result of a minor gynæcological operation is of more practical interest both to the surgeon and to the patient than its skilful performance and a smooth, rapid convalescence.

AFTER-TREATMENT OF LAPAROTOMIES.

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY MATTHEW D. MANN, A.M., M.D.,

Professor of Gynæcology, Medical Department of the University of Buffalo.

In our last two hundred cases of laparotomy the deaths have been six or eight, and, considering that many of the cases were desperate and that two or three died on the table, I think we have a right to say that the methods which we employ in this hospital are good. I think it quite important that you should know something about the after-treatment of cases of abdominal section. The first principle I lay down is to let the patients alone. Immediately after the operation the less interference, the less medication, the less food, the less drink, the less everything except quiet, the better. If I find a great deal of shock—there is always a certain amount—I use a little stimulant until reaction begins.

I have adopted the plan of using drainage but seldom. Some men use it in every case, others do not use it at all. I look upon a drainage-tube as a nuisance, and use it just as little as I can. I would rather delay the operation a little and put sponges into the peritoneal cavity than insert a drainage-tube and close the abdomen immediately. There is always a certain amount of fluid effused into the peritoneal cavity, bloody at first, serous afterwards. This effusion is to a certain extent a source of danger, because it is a pretty good culture-medium, and the temperature in the abdomen is right for the development of pathogenic bacteria. In order to stimulate the lymphatics to their greatest absorbing power, we keep the patient on a very scant regimen, giving only a teaspoonful of water now and then, so that the lymphatics will remove this fluid from the peritoneum. The patients say that the greatest suffering they have to endure is the lack of water. Within a short time, I presume, the peritoneal cavity becomes perfectly dry. Many authorities recommend if there be any rise of temperature, an indication of septic trouble, that a saline cathartic or saline enema be

given to move the bowels and still further stimulate the lymphatics by depriving the system of water. I have never used these saline enemata for this purpose, perhaps because I so seldom see septicæmia. The only cases we have seen here have been of such a nature that saline enemata would have done no good; for example, one case that resulted from the dropping back into the peritoneum of a stump[1] after hysterectomy.

When there have been numerous adhesions and much tearing of tissues and a greater probability of the effusion of fluid, the strict regimen with regard to water is all the more necessary.

As to the control of pain, I always encourage patients to get along without morphine if possible. One bad result of morphine is its effect on the intestines, stilling peristalsis, and making the intestines liable to form adhesions. I have lost two cases from obstruction of the bowels after laparotomy, and in each case adhesions to the stump after operation made a sharp angle in the intestine. I think that if no morphine had been given to those patients, particularly during the first twelve hours, till the intestines had had time to rearrange themselves after the operation, the obstruction of the bowels would have been less likely. I speak of rearrangement of the intestines, for during an operation they are often necessarily handled to a greater or less extent and their position disturbed. Again, the after-effect of morphine is to cause vomiting in a considerable number of cases. My first laparotomy case vomited almost incessantly for two or three days. This was one of my worst cases. When she was under the influence of morphine she did not vomit, but as soon as she came out of it she would begin again. Finally I stopped the morphine absolutely, and after vomiting for a few hours she ceased, and made a good recovery. Another bad effect of morphine is the increased pain in the intestines after stopping the drug. The intestines are paralyzed by the morphine, and when its effect begins to wear off the intestines begin to come out from the quieting effects in spots, so to speak, and there are irregular contractions, the nerves have not full control of the muscular coat, and from the irregular movements of the intestines colicky pains result. I have noticed that whenever I have taken morphine myself, as soon as the effect has begun to wear off I have experienced irregular griping pains in the bowels, and I have observed the same phenomenon in patients. I have therefore arrived at the theory which I have just given you. If the patient suffers very much from pain in the wound on account of

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[1] Due to bending of the pins.

coughing, muscular movement, and so forth, I strap the abdomen very tightly and encourage the patient to get along without the morphine as far as possible.

The movement of the bowels is another important thing. When I was a hospital interne I had nine laparotomies in my service, which was considered to be an immense number. The internes here have more than that in a month. We had no trained nurses in those days, and we had to do the nursing ourselves. It was the custom then to keep the patients under the influence of morphine; in fact, we began it two or three days before the operation, thinking that it would quiet the nervous system. The bowels were kept bound up for five or six days, with the idea that it would be dangerous to have them moved sooner. When the bowels did move there was a terrible time. Now I do not care if the patient's bowels move within an hour after the operation. In fact, I give the nurses a standing order to give an enema if there is colicky pain in the bowels. A large enema, however, should not be given, since too much water would be absorbed into the system. A small glycerin enema should be used instead. If the bowels do not move within a day or two, we give Noble's enema, thrown far up into the bowel, and it hardly ever fails. The formula is:

℞ Magnesii sulph., ℥ii;
Ol. terebinthinæ, ℥ss;
Glycerini, ℥i;
Aquæ, q. s. ad ℥iv.

Sometimes compound liquorice powder or other drugs are given by the mouth.

Food is given only on the second or third day, and in very small quantities at first. If there is any vomiting I prefer to let the stomach alone, giving only water or a little tea or coffee till the stomach is settled. We begin with light broths usually. If the patient can take milk,—and there is no use in trying to force every one to take milk,—we begin with that, two ounces being administered, hot or cold, according to the taste of the patient, every three hours. After five days the patients are allowed to take almost anything. Fruit, oranges, grapes eaten without the seeds, and particularly Malaga grapes, peaches, etc., seem to agree very well.

As regards the removal of the stitches, I leave the dressings on for about seven days. The parts are soaked in an antiseptic solution before removing the stitches, so as to avoid any infection due to drawing the sutures through the wound.

The patient is not allowed to sit up under fourteen days, on account of the danger of tearing open some of the deep tissues and ultimately producing ventral hernia. After this time the patients are allowed to sit up, and are sent home at the end of the third week, or a little later. After the wound is entirely healed, I like the patient to wear an abdominal supporter for two or three months. The London, the Gray and Foster, and the Marvin supporters are very good ones. We use one here devised by Dr. Howard A. Kelley, of Johns Hopkins University. This is inexpensive and answers the purpose well. It has a band coming between the thighs to keep the bandage from slipping up, and it is fitted to each patient. After the patient goes home, I advise her to keep very quiet for a number of months, and after three or four months she can do about as she pleases.

The temperature in these cases has a very constant normal curve. Immediately after the operation there is a fall, and in the evening there is a rise, and the second evening it usually reaches the highest point, 100° or 101°. Then it falls to between 98.5° and 99.5°, and remains there till about the tenth day, or sometimes sooner, when it drops to normal. You may always look for a rise on the first or second evening. This depends somewhat on the impressionability of the patient, on the amount of shock, etc. The pulse varies greatly in different individuals, ranging between 80 and 120 for the first few hours, and then it begins to come down. If I find the pulse-curve on the chart downward, I do not feel any anxiety about the temperature unless it goes unreasonably high. The pulse is a much safer indication of the condition of the patient than the temperature. I seldom give any antipyretics if the temperature does go up. If there is very severe headache and much febrile disturbance, an antipyretic may relieve the trouble, but I prefer not to mask the course of the fever, on account of its value as a diagnostic point with regard to septic infection.

CARE OF PESSARIES; MOVABLE LIVER; CONSTITUTIONAL AMENORRHŒA; LATERO-FLEXION OF UTERUS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY PAUL F. MUNDÉ, M.D.,

Professor of Gynæcology.

Case I.—This patient is forty-four years of age; she is a widow, and has had eight children, the last seven years ago. She flows every three weeks, and has pain in the back and abdomen; there are also headache and constipation. There is a pessary in the vagina, and examination shows that the uterus is held by it in the proper position. The patient comes here to-day because I have made it an invariable rule that any patient with a retroverted or retroflexed uterus, after having a pessary introduced, is to return within about a week, in order that we may see, after this interval, whether or not the pessary is still in place and giving proper support. If it be in proper position, the patient can safely go from observation for two or three months, only taking care to keep the parts clean. A pessary for retroversion or retroflexion should never be so large that you cannot easily pass your finger between the instrument and the vaginal wall, nor should it be so small that with a slight movement of the finger you can dislodge it from its position. Be careful not to advise women wearing pessaries to use injections of alum-water, although it is recommended in many books, for my experience has taught me that alum will sooner or later produce an encrusted and roughened pessary, resulting in erosion and irritation of the vagina. Always remember that in retroversions and in prolapse the uterus must be placed in its proper position before the pessary is introduced. This does not apply to anterior displacements, for they usually do not require pessaries at all, and this is fortunate, for it is quite difficult to fit pessaries to such cases. There is no pessary, except a stem inside of the uterus, which will straighten an anteflexed uterus.

Case II.—Our next patient is forty-five years of age; she has been married twenty-two years, and has had eight children, the last

one eight years ago. Menstruation has been irregular for the last six months, the last period being two months ago. She complains of pain in the right side. Her abdominal walls are very lax, owing to the numerous pregnancies. She comes to us with a diagnosis of movable kidney. The tumor on the right side has a sharp, well-defined edge, and both palpation and percussion indicate that it is continuous with the liver,—that it is, in fact, a movable liver. This condition is occasionally met with in women whose abdominal walls have become very much relaxed, either by numerous pregnancies or by the distention of some other abdominal tumor. There are usually dragging pain and more or less discomfort, but ordinarily floating livers do not produce the symptoms which accompany floating kidneys. I once removed a displaced kidney, situated in the pelvic cavity behind and to the left of the uterus, mistaking it for an inflamed ovary and tube. The woman had recently had a peritonitis, and the left ovary could not be felt at first, even with the finger in the abdominal cavity. The patient recovered. This patient should be provided with a pad which will keep the liver in its proper position.

Case III.—The next patient is a single woman, twenty years of age; she flows every four weeks, and has pain just before menstruation. Her last menstruation was four months ago. She complains of backache and constipation.

The fact of the existence of amenorrhœa in any woman who has not passed well beyond the menopause should always lead us to be on our guard, and to be very suspicious of the case being one of pregnancy. I shall, therefore, proceed to assure myself that the size of the uterus does not correspond to that of a four months' pregnancy. On further inquiry, she says that she was born in Ireland, and has been in this country for three years. She was seventeen years old when she first menstruated, and then "it was brought on by medicine." You will find that women coming to this country, particularly from the middle and northern parts of Europe, very commonly skip one or two menstrual periods after arriving here. This is due to the change of climate, and probably also to change of occupation and habits of life. I find that this girl is intensely anæmic; the lips and gums are almost bloodless, and she suffers also from a cough. It is quite possible, therefore, that the amenorrhœa in this case is due to this patient's general anæmia as well as to the change of climate, and perhaps also to an irregularity from which some girls suffer until they are in the twenties. Inspection of the genitals shows that the hymen is intact, and our examination therefore tends to exclude pregnancy.

The treatment consists in the administration of general tonics, chiefly iron, along with aloes, or some other remedy to regulate the bowels. I prefer, in these cases, Blaud's mass to any other form of iron. I would recommend the following pill to be taken:

℞ Aloin, gr. ¼, or less;
Ext. of nux vomica, gr. ¼;
Powd. rhubarb, gr. i;
Blaud's mass, gr. iv.
This to be taken after each meal for some months.

This treatment will probably greatly improve this girl's health, and perhaps restore regular menstruation. In addition to this, of course, the girl should be allowed plenty of fresh air and exercise, and, if her circumstances will permit, let her make use of salt-baths and horseback-riding, and an abundance of the most nourishing food. She has a slight anteflexion of the uterus, but it is of no consequence, and is present in a large proportion of young girls. It does not give rise to any symptoms and does not require any treatment.

Case IV.—The next patient is forty years old, and is single. She flows every four weeks for three days, the last time being one week ago. She complains of pain during menstruation, and also of pain on the right side of the abdomen and in the back. She also complains of neuralgic pains all over the head, of pains in the epigastrium, and of vomiting. She is sent here by Professor Gray to see if there is any cause in the pelvis for her neuralgia.

Digital examination shows a peculiar and rather uncommon displacement of the uterus, but one which I think has nothing to do with her symptoms. Instead of lying in the normal position, it is tilted, with the fundus to the left side, and is sharply anteflexed, and the uterus cannot be readily returned to its normal position. She may have had an inflammation of the appendages on the left side, which would account for the displacement, but the displacement in itself does not give rise to any local or reflex symptoms. It is possibly even a congenital condition. Lateral displacements of the uterus, whether flexions or versions, are the results of one of two conditions,—viz., either a congenital shortening of the ligaments on one side or the other, or an inflammatory contraction of the ligaments on one side: thus, a contraction of the broad ligament on one side, or of the cellular tissue following inflammation, would cause the cervix to be dragged over to that side, and *vice versa*. The treatment by pessaries is very unsatisfactory, and, fortunately, the condition rarely calls for such interference.

Ophthalmology.

DISEASES OF THE EYE ASSOCIATED WITH DISEASES OF THE KIDNEY.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL.

BY WILLIAM OLIVER MOORE, M.D.,
Professor of the Diseases of the Eye and Ear, New York Post-Graduate Medical School and Hospital, etc.

Gentlemen,—I shall speak to you this morning of diseases of the eye occurring in connection with affections of the kidney. This patient, now under treatment, gives a fair clinical picture of this affection. Susan R., aged twenty-one years, for several months prior to coming under observation complained of severe headache with nausea. She is, however, able to attend to her work as a domestic, but her general health has become impaired to such an extent that she will be unable in a short time to fill her position. The examination of the eyes reveals V = $\frac{10}{100}$ in each eye. To external appearance they are normal in every respect, but the ophthalmoscope shows the well-marked signs of neuro-retinitis nephritica or albuminurica. The examination of the urine shows slight traces of albumin and granular casts. She has had at different times pain across the back; she is not married, and menstruation is regular.

Half a century before Dr. Bright published his discovery of the association of renal disease with albumin in the urine, cases of dropsy were recorded in which remarkable loss of vision occurred. These cases had been ascribed to lesions of the brain, but were in reality due to chronic renal disease. Although in 1827 Bright made his announcement, and in 1832 published a report of one hundred cases, in many of which ocular symptoms were present, very little attention was paid to this complication until Landouzy, in 1851, made it a

The treatment consists in the administration of general tonics, chiefly iron, along with aloes, or some other remedy to regulate the bowels. I prefer, in these cases, Blaud's mass to any other form of iron. I would recommend the following pill to be taken:

℞ Aloin, gr. ¼, or less;
Ext. of nux vomica, gr. ¼;
Powd. rhubarb, gr. i;
Blaud's mass, gr. iv.
This to be taken after each meal for some months.

This treatment will probably greatly improve this girl's health, and perhaps restore regular menstruation. In addition to this, of course, the girl should be allowed plenty of fresh air and exercise, and, if her circumstances will permit, let her make use of salt-baths and horseback-riding, and an abundance of the most nourishing food. She has a slight anteflexion of the uterus, but it is of no consequence, and is present in a large proportion of young girls. It does not give rise to any symptoms and does not require any treatment.

Case IV.—The next patient is forty years old, and is single. She flows every four weeks for three days, the last time being one week ago. She complains of pain during menstruation, and also of pain on the right side of the abdomen and in the back. She also complains of neuralgic pains all over the head, of pains in the epigastrium, and of vomiting. She is sent here by Professor Gray to see if there is any cause in the pelvis for her neuralgia.

Digital examination shows a peculiar and rather uncommon displacement of the uterus, but one which I think has nothing to do with her symptoms. Instead of lying in the normal position, it is tilted, with the fundus to the left side, and is sharply anteflexed, and the uterus cannot be readily returned to its normal position. She may have had an inflammation of the appendages on the left side, which would account for the displacement, but the displacement in itself does not give rise to any local or reflex symptoms. It is possibly even a congenital condition. Lateral displacements of the uterus, whether flexions or versions, are the results of one of two conditions,—viz., either a congenital shortening of the ligaments on one side or the other, or an inflammatory contraction of the ligaments on one side: thus, a contraction of the broad ligament on one side, or of the cellular tissue following inflammation, would cause the cervix to be dragged over to that side, and *vice versa*. The treatment by pessaries is very unsatisfactory, and, fortunately, the condition rarely calls for such interference.

Ophthalmology.

DISEASES OF THE EYE ASSOCIATED WITH DISEASES OF THE KIDNEY.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL.

BY WILLIAM OLIVER MOORE, M.D.,
Professor of the Diseases of the Eye and Ear, New York Post-Graduate Medical School and Hospital, etc.

Gentlemen,—I shall speak to you this morning of diseases of the eye occurring in connection with affections of the kidney. This patient, now under treatment, gives a fair clinical picture of this affection. Susan R., aged twenty-one years, for several months prior to coming under observation complained of severe headache with nausea. She is, however, able to attend to her work as a domestic, but her general health has become impaired to such an extent that she will be unable in a short time to fill her position. The examination of the eyes reveals V = $\frac{10}{100}$ in each eye. To external appearance they are normal in every respect, but the ophthalmoscope shows the well-marked signs of neuro-retinitis nephritica or albuminurica. The examination of the urine shows slight traces of albumin and granular casts. She has had at different times pain across the back; she is not married, and menstruation is regular.

Half a century before Dr. Bright published his discovery of the association of renal disease with albumin in the urine, cases of dropsy were recorded in which remarkable loss of vision occurred. These cases had been ascribed to lesions of the brain, but were in reality due to chronic renal disease. Although in 1827 Bright made his announcement, and in 1832 published a report of one hundred cases, in many of which ocular symptoms were present, very little attention was paid to this complication until Landouzy, in 1851, made it a

special study, and found that the lesion was in the retina, and not in the brain, as formerly held. It was in this same year that Helmholtz invented that most exact of all the aids to physical diagnosis, the ophthalmoscope, which opened such an era in general as well as in ophthalmic medicine. Instead of the visual disorders in kidney-disease being looked upon as interesting symptoms in the progress of the disease, they have now become an index of unsuspected kidney-affection, and a means of diagnosis. Frequently patients with failing vision present themselves to our notice complaining only of this defect, and by the ophthalmoscope we are able to diagnosticate albuminuria and renal disease, already well established, by the invariable retinal picture, and so characteristic is it that when once seen it cannot be mistaken thereafter for anything else. The peculiar changes seen in the retina are not found in any other disease of this tissue, although Wadsworth says that the same appearances obtain in neuro-retinitis from cerebral tumors. In speaking of the peculiar and characteristic picture seen in the retina in both acute and chronic renal disease, we exclude for the present that form of disturbance of vision due to uræmic convulsions, where the vision is impaired, as a rule, for only a few hours. The changes which take place in the retina in kidney-diseases are more commonly retinitis albuminurica, amaurosis uræmica, and detachment of the retina.

RETINITIS ALBUMINURICA.

This diseased condition may occur in every form of renal inflammation, and has been seen in amyloid degeneration, yet it is more commonly associated with the granular or contracted kidney. It occurs during and after scarlatina, and may be found in some cases of spontaneous origin. The frequency with which it is associated with renal disease is variously stated; for example, Galezowski found retinitis albuminurica in thirty-three per cent., Lebert in twenty per cent., Lecorche in twenty-one per cent., Earle in twenty-nine per cent., and Ayres in nine and a half per cent. Voelcker, in thirty cases, found two incident to pregnancy, and the rest associated with contracted kidney. It would thus seem that about twenty-three per cent. of patients with renal inflammation have disorders of vision at some period of the disease. In many cases the renal disease is well established before the eye-symptoms present themselves; but very often the eye-lesion is the first indication of the general disease. Patients with this complication of kidney-disease complain only of gradual failure of sight, everything appearing blurred and smoky. This failure

of vision may in some instances come on rapidly; it generally occurs in both eyes simultaneously, although I have seen it in only one eye, the other remaining normal. Such a case has recently been under my care, and the following is a brief summary of the history of this patient. I mention it because it is unique. Male, sixty-seven years old, has albuminous urine and hypertrophy of the left ventricle, and other well-marked evidence of granular kidney. The right eye was injured twenty years ago, resulting in a traumatic cataract with adhesions of the iris to the cornea. Owing to the pain in this eye produced by the iritic adhesions and the cataract, cataract extraction was performed, with good result. The retina and choroid, as seen by the ophthalmoscope after the operation, were found to be normal. One year later the patient returned with failure of vision in the left eye, and the fundus showed all the well-marked appearances of albuminuric retinitis; the right eye remained normal. The left eye presented later the symptoms of acute glaucoma, due to intra-ocular hemorrhage, and the patient died two years after this, the right eye never having become affected with albuminuric retinitis. This occurrence of one-sided retinitis albuminurica has been noticed by several authors. Yvert reports the case of a man whose urine contained albumin, who, after being under observation seventy-two hours, died. The autopsy showed only one kidney, and this of the large white variety. This patient had retinitis albuminurica on the same side as that on which the kidney was found, the opposite eye being normal. Yvert explains this one-sided eye affection by assuming an irritation of the sympathetic nerve on one side, due to unilateral affection of the kidney, and he cites five cases, reported by Potain, where there was anasarca of only one side in consequence of contusion of the kidney in which he considered the sympathetic of one side of great importance. In our case, we should rather consider the reason to be the difference of tension of the eyeball,—the right eye, the one from which the cataract had been removed, being less firm, and the vascular system less interfered with. The course of the disease is various, but rarely results in recovery.

Ophthalmoscopic Appearances.—We rarely have the opportunity of seeing the first stage of the disease, but those who have seen it describe the appearances as those of congestion. Subsequently, when the patient presents himself for treatment, we find cloudiness of the papillæ of the optic nerve and of the adjacent retina, the nerve looks gray and swollen, its margin is indistinct, the retinal veins are large and tortuous, and show points of different color, depending upon their varying depth.

The arteries are smaller than usual or normal in size; the retina about the nerve is cloudy and œdematous, with extravasations of blood here and there present, concealing the vessels in some instances. They are sometimes round, and again irregular in shape; we also notice white spots of various sizes from a pin's point to the size of the papilla itself. In the macular region we find on its temporal side many fine white dots radiating from it as a centre. These dots look very much as if a brush full of white kalsomine had been thrown upon the retina at this point. Often the previous cloudiness of the retina already mentioned increases, the exudations grow larger, coalesce, and surround the optic papillæ; the hemorrhages may increase, and the specks and dots about the macula also increase and become confluent. The refraction of the eye in these cases is usually hypermetropic, owing to the œdema of the retina. These appearances are usually present in both eyes alike, yet they may vary. In some cases they are much less marked, and we have only the peculiar stippling at the macula, without any swelling at the optic papillæ, and with only a few hemorrhages, or perhaps none at all. These changes may subside again, the white patches becoming smaller, and the vessels they cover becoming again visible, the cloudiness of the retina subsiding, and the outline of the optic nerve again appearing. Only in that form following scarlatina, and that which accompanies the nephritis of pregnancy, do we see occasional complete absorption; of course, when the optic nerve has had much infiltration in the beginning more or less damage has been done to it, and atrophy of its fibres will ensue. The changes just described are found in retinitis albuminurica occurring in pregnancy and in the various forms of renal disease.

Anatomical Appearances of Retinitis Albuminurica.—In this form of retinitis we find that portion of the retina in the neighborhood of the optic papillæ and macula lutea involved. In these regions all the parts of the retina suffer, and in many cases not only the blood-vessels and connective tissue but also the nerve-filaments are altered. Microscopically we find much swelling of the optic papillæ, which is due to œdema and hyperplasia of the connective tissue. In the macular region we find cells which contain numerous fat-globules having a nucleus. These are ganglionic cells undergoing retrograde metamorphosis. Numerous hemorrhages are also found in the nerve-fibre layer and in the neighborhood of the blood-vessels, or they may lie in the deep tissue of the retina. Crystals of hæmatoidin are never found after such hemorrhages. The hemorrhages are probably due to changes in the walls of the vessels. Dr. Johnson says that the muscular walls of the minute arteries in most of the organs are hypertrophied, owing to long-continued over-action in chronic renal disease, and that this contraction of the small arteries impedes the onward flow of blood, and calls for an increased effort on the part of the heart to carry on the circulation, causing hypertrophy of the left ventricle; hence, owing to the increased action of the heart, and the increased arterial resistance, great pressure is brought to bear on the arterial wall, and there is necessarily a greater risk of extravasation. This increased thickness of the arteries differs in various parts. The retinal hemorrhages have been thought to be due to this want of hypertrophy in the small retinal vessels, the heart-action not being counterbalanced by an increase in their thickness. Denissenko examined all parts of the eye, and found no evidence of inflammation, but all parts were infiltrated with albuminous liquid, and a general œdema prevailed, the interstices of the tissues being distended and torn. The albuminous exudation may coagulate, and even undergo fatty degeneration; only a few wandering cells were found in the swollen tissue. The cause of the exudation here, as well as in the rest of the body, must be said to be in the nervous system. According to this author, the exudation causes compression of the blood-vessels, and to this also he ascribes the extravasation of blood, and detachment of the retina, when present. The peculiar stellate dots referred to in the macula, in the ophthalmoscopic appearances, are owing to fatty degeneration of the radial fibres of the retina, the stellate appearance, according to Schweigger, being due to the anatomical arrangement of these fibres at the macula. The pathological process in the retina I do not believe to be an inflammatory one, but a tissue-metamorphosis, which produces permanent changes in the retina, such as are brought about by the changes in the vascular system generally, which in turn are caused by the effete materials circulating in the blood, due to improper action of the kidneys; this material may be urea alone, or other substances. The cardiac complication so frequently found naturally increases the difficulty in the vascular system already mentioned. These same phases are found in the retinitis albuminurica of pregnancy and scarlatina.

Retinitis Albuminurica in Pregnancy.—It has long been known that pregnant women are liable to suffer from disturbances of vision, from a slight impairment to complete and permanent blindness. This was known long before Bright's time, the cause being usually referred to cerebral origin. We now find it due in the majority of cases to renal disease. The same retinal changes are found as those already

described. It occurs at varying times during pregnancy from the second to the eighth month, but it is more apt to occur during the last four months; it may occur after abortions. Many cases have been reported where the retinal disturbance produced blindness for weeks, and then after labor complete recovery occurred, and others, of less fortunate women, who have remained partially blind. The occurrence of this disease during pregnancy opens up some very interesting points regarding the proper course to pursue. Eastlake reports an interesting case of a patient, thirty-four years of age, who had had nine children at full term, and no miscarriages, who at the birth of the first child had no ocular symptoms, yet on the second day after the birth of the second child, and after all the seven subsequent labors, suddenly became totally blind in both eyes, the blindness lasting from three to five weeks. Ophthalmoscopic examination of these eyes long afterwards demonstrated only the existence of contracted retinal arteries and blanched optic nerves,—the last stages of albuminuric retinitis. Dr. W. J. Scott, of Cleveland, Ohio, reports a number of similar cases, and says, in 1875, "What advice shall be given to patients who have suffered from these conditions in the first pregnancy? Would it be justifiable to advise against conception, or to remove the fœtus afterwards?" He answers by saying, "I think only after serious symptoms have presented themselves should we interfere."

The first case under our notice in which labor was induced for the reason that permanent blindness was feared was reported by the late Dr. E. G. Loring, in a very interesting article published in the *New York Medical Journal*, vol. xxxvii. Since this case was reported before the American Ophthalmological Society, I have had a case under my own care in which labor was induced at the eighth month for the sole purpose of preventing permanent blindness. The history is briefly as follows. A woman, forty-eight years old, was pregnant with her twelfth child, and had never presented ocular symptoms in the previous pregnancies. Three weeks before coming under observation she had a chill, and since then had not felt so well. Impairment of vision was noticed shortly after the chill, and she became nearly blind. When first seen she was anæmic, there was no anasarca, the urine contained one-third albumin with some epithelial casts, and the ophthalmoscopic picture showed the media clear, the retina much swollen, and a neuro-retinitis with exudations and well-marked hemorrhages, more especially in the macula of the left eye. R. V. $= \frac{1}{70}$; L. V. = fingers at one foot. The field of vision was much narrowed, so that the patient was unable to walk alone. *Without examination of the urine*, it was decided best for the future welfare of the eyes that premature labor be induced, and, as there were no proper facilities at hand in the institution where she was first seen, she was transferred to a neighboring one, where premature labor could be induced. An examination per vaginam showed the os dilated one inch, and the breech and foot of the fœtus presenting. By irritation of the uterine walls with the finger, labor was induced, and a dead fœtus was expelled, together with a fatty placenta. The patient made a good recovery after a convalescence of two weeks. Vision began to improve from the time labor was completed, and three months subsequently R. V. was improved to $\frac{20}{50}$, and L. V. to $\frac{20}{70}$. Her vision remained at this point up to the time of her death, two years later.

Here we see a case where, in my opinion, had the patient been allowed to go on to full term she would have been blind for some time before natural labor took place; very much more damage would have been done to the delicate tissue of the retina. It will be noticed that this patient was about to abort owing to the fatty placenta and the dead fœtus produced by the kidney-disease. Premature labor was advised solely for the preservation of sight, and on the assumption that the child was viable, the eighth month being considered a perfectly safe time. Had I known at the time that the fœtus was dead, the same treatment would have necessarily followed. Life is often endangered when vision is, and when the life of the mother is in danger there is no doubt about the weight of authority among obstetricians concerning the general question of abortion or premature labor. Dr. Howe, of Buffalo, in an analysis of cases occurring during a period of fifteen years, says, "These tend to show that when vision begins to be impaired only in the last two weeks of pregnancy, recovery follows almost invariably. Of those described as being in the eighth month, or thereabouts, when the retinitis commences, not one-half recovered, and several did not materially improve. Again, when this began earlier than was estimated, as the middle of the seventh month, when nature did not interfere by bringing on a miscarriage, and when the patient escaped with her life, it was only to remain blind for ever afterwards." From this array of facts I would conclude that where great failure of sight has occurred, and where it is progressive, the induction of premature labor is justifiable, and often demanded. Again, when in one pregnancy failure of vision has occurred which remained permanent, abortion in a subsequent pregnancy, with proper restrictions, is justifiable and often a necessity.

AMAUROSIS URÆMICA.

In this class of cases there are no intra-ocular changes except in some rare instances. There is œdema of the retina, which passes away in a few days, or more rarely it is so marked as to cause a separation of the retina by serous infiltration, as in the case reported by Heyl, where retinal separation in the right eye and uræmic amaurosis in the left occurred simultaneously. As to the cause of the blindness in uræmic amaurosis, Traube's theory seems at present to hold good. He considers the cerebral symptoms of uræmia to be due to acute anæmia of the brain, caused by œdema of the brain-tissue, due to the circulation of urea in the blood. The blindness in these cases is sudden and complete, and it usually is associated with a group of uræmic symptoms, —convulsions, etc. The treatment of this, as of the other ocular symptoms occurring in renal diseases, needs no special local measures; the treatment of the general disease is the best that can be done for the eyes. As regards the prognosis of retinitis albuminurica not associated with pregnancy, the duration of life does not, as a rule, exceed eighteen months. Thus it behooves us all, gentlemen, when we do not adhere to the code of health of the "school of Salernum," which says,—

> "At least six times in every fleeting day
> Some tribute to the renal function pay,"—

to consult some fellow-practitioner.

TREATMENT OF ACUTE PURULENT CONJUNCTIVITIS AND OPHTHALMIA NEONATORUM.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY THOMAS R. POOLEY, M.D.,

Professor of Ophthalmology in the New York Polyclinic, and Surgeon-in-Chief of the New Amsterdam Eye and Ear Hospital.

Gentlemen,—The other day we considered the treatment of the severer forms of purulent ophthalmia. Continuing this subject, I may say that considerable importance is to be attached to the proper cleansing of the eye, and whether an antiseptic solution is to be employed or not is for you to decide, and will depend upon the extent to which your mind or your imagination is carried away with the present views concerning the advantages of antiseptics. Personally, I think cleansing the eye with lukewarm water is as good as anything. It should be done by a competent nurse, the pus being wiped away with absorbent cotton every thirty minutes or oftener. The cotton should be destroyed after each washing. The cleansing of the eye by the use of syringes should be severely condemned, for it is obvious that the attendant is subjected to the danger of contamination with the secretions from the patient, and also because it is very difficult to control the force of a stream from a syringe, and if the cornea be weakened, as it often is, by the disease, you favor the rupture of the eyeball. A great variety of antiseptics are recommended; some use carbolic acid, others bichloride, and still others boracic acid.

Let us speak of the treatment of what may be termed the complications which arise in the course of severe purulent ophthalmia; and, first of all, let me urge you to examine such eyes with great care, for it is not always an easy matter to tell when the cornea is implicated. In a very severe case of gonorrhœal ophthalmia which I had under treatment, I told one of my colleagues I thought there was a perforating ulcer in the upper corneal margin, but he could not see it until I suggested that he raise the chemotic conjunctiva which

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Let us speak of the treatment of what may be termed the complications which arise in the course of severe purulent ophthalmia; and, first of all, let me urge you to examine such eyes with great care, for it is not always an easy matter to tell when the cornea is implicated. In a very severe case of gonorrhœal ophthalmia which I had under treatment, I told one of my colleagues I thought there was a perforating ulcer in the upper corneal margin, but he could not see it until I suggested that he raise the chemotic conjunctiva which

covered it. It is in this locality that you so often find these ulcers, and if seen when the ulcer is well formed it has a perfectly transparent base, making it difficult to determine that the ulcer is really present. If ulceration be not present, you may find infiltration,—*i.e.*, the cornea is rendered opaque by either serous or purulent infiltration. In the severer cases there is infiltration of the cornea nearer to its centre, or even the whole corneal membrane may be invaded. When the whole cornea is infiltrated with pus, sloughing is of course inevitable. Now, if the process of ulceration lead to perforation of the cornea, what is to be expected if this occur at the corneal margin? The eye is by no means lost. The perforation may be such that the iris is prolapsed, and if this occurs, one rule should always be followed here which is not applicable in other cases,—*i.e.*, to avoid abscission. If you excise the iris, you favor the invasion of the purulent material into the interior of the eye. If ulceration is imminent, it may be sometimes averted by the use of eserine. Instil a solution of eserine, one-half per cent. or weaker,—for a stronger solution has a tendency to provoke iritis. It probably acts by relieving the intra-ocular tension, and the danger, of course, is that the normal intra-ocular tension may cause rupture. Many advise the performance of paracentesis when the ulcer is very deep, but this I never do, for I think it is better to employ the eserine; and if rupture occurs in spite of this, I think I have done all that is advisable.

The purulent process may extend to the interior of the eye, and this is almost necessarily fatal to the integrity of the eye; for if there be considerable sloughing of the cornea, and the purulent process reaches the interior of the eye, in all probability there will be a panophthalmitis,—an extension of the purulent trouble to the interior of the eye,—and this is to be deplored on account of the certain loss of sight, and also because the pain is thereby greatly increased.

Another and not infrequent complication is iritis. In all of these corneal diseases, and in iritis also, it is well to employ atropine for the alleviation of pain, and, above all, in iritis, to prevent the adhesions between the iris and the lens. In two or three of my cases where iritis has complicated these purulent cases, there has been an extensive exudation into the interior of the eye,—into the anterior chamber and into the pupil. I am not sure that this is a special feature of this complication, but it would seem probable. The exudation was what is known as "spongy exudation." This was first described minutely by Dr. Gunning, of Amsterdam, and afterwards by Dr. Gruening, of this city. The latter gentleman showed that the gelatinous exudation was but one of the stages of a spongy exudation. This exudation forms very rapidly and completely fills the pupil, and often also the anterior chamber. It resembles very closely the structure of a sponge, and hence the name of spongy exudation, which I believe was first given to it by Dr. Knapp. It consists of fine filaments which cross one another in all directions. When examined by oblique light, it resembles very closely the microscopical appearance of a bone-corpuscle. Within twenty-four or forty-eight hours it will change to a whitish homogeneous mass, which looks like a cataractous lens dislocated into the anterior chamber, and then absorption begins at the periphery and continues until it is entirely absorbed. You may achieve considerable reputation by your prognosis in such a case, for it alarms the patient, and yet you can certainly predict that within a few hours it will have all disappeared.

Extending our remarks to ophthalmia neonatorum, we find that the manner of invasion of the cornea is not essentially different from that already described. The examination of the patient is somewhat troublesome, yet you must remember that it is your duty to examine the condition of the cornea in all these cases at least once in every twenty-four hours. In young children, corneal complications, especially infiltrations, are not so much to be dreaded as they are in the severe forms of purulent ophthalmia of adults due to infection.

If we turn now to a consideration of the conditions remaining after recovery from a purulent process in the conjunctiva, we find that a good many eyes may be saved from absolute blindness, both in adults and in the new-born. There may be an opacity of the cornea as a result of the purulent process,—*i.e.*, a change in the entire corneal structure, constituting what we call leucoma. This leucoma may be simple or adherent,—that is, the iris may be adherent to some part of the scar in the cornea. It results from the substitution for the corneal tissue of some newly-formed connective tissue, which remains perfectly opaque. If a portion of the cornea remains clear, you often secure fair sight for these patients by an iridectomy. In a great many cases of this kind, and especially after ophthalmia neonatorum, iridectomy is rendered very difficult, because the iris is adherent to the scar and is put on the stretch, and the anterior chamber is shallow; nevertheless, in very many cases you can make the distinction between a simple perception of light, or slight useful vision, and absolute blindness by carefully searching out some portion of the cornea which is clear and performing an iridectomy in this locality. The operation often cannot be done with the ordinary iridectomy knife, in which case

Graefe's knife will have to be employed. Often the iris cannot be grasped by the common iridectomy forceps, and then you must use the iris forceps of Matthieu, and if these fail you may use Tyrrell's hook. Besides these cases, there are some with staphyloma. I recently showed you a case where there was a total staphyloma. Here there can be no recovery of vision; but if only a part of the cornea be invaded by staphyloma, you may quite often restore vision to the eye. Thus, in the case of a boy recovering from gonorrhœal ophthalmia, where the whole upper third of the cornea was the seat of a dense leucoma, which afterwards became staphylomatous, and where the iris was transparent in the direction of the scar, I restored very good sight to the patient. The first thing I did was to perform Küchler's operation for staphyloma, which consists in dividing the staphyloma throughout its entire length in the vertical meridian. The lens may lie under the staphyloma, and if so you evacuate the lens; if not, you cut from each lip of the horizontal wound you have made a narrow strip of the leucomatous tissue. The eye is now to be kept persistently bandaged for several weeks, when it will be found that there is a flat leucoma instead of a protrusion. After this, you can perform an iridectomy, as I did in the case just alluded to, with the result of securing him very useful vision. As the outcome of long experience, I would say, never make a perfectly hopeless prognosis in ophthalmia neonatorum, except where there has been almost complete sloughing of the cornea.

Laryngology and Rhinology.

NASAL CATARRH.

CLINICAL LECTURE DELIVERED AT THE BELLEVUE HOSPITAL MEDICAL COLLEGE.

BY BEVERLEY ROBINSON, M.D.,

Clinical Professor of Medicine in the Bellevue Hospital Medical College, New York.

NASAL OBSTRUCTION.

GENTLEMEN,—This young lady came to me complaining of marked obstruction in the right side of the nose. With the Jarvis "nippers" I removed sufficient cartilage to relieve the obstruction partially, but, for some unaccountable reason, the tissue on the right side, instead of being cartilaginous down to the nares, seemed to be bony, and I found it necessary to use the Bosworth saw. The operation was done only a few days ago, so that some of the obstruction still present is due to the granulation-tissue; but more tissue will probably have to be removed.

IMPERFECT SPEECH-DEVELOPMENT.

This young man came to me the other day and said that a horse had kicked him in the temple seven years ago, and that following this there was trouble with both ears, but no loss of consciousness, and no paralysis of the limbs. Examination shows nothing wrong in the throat or nose. He has a marked impediment in his speech, which is probably due to a central lesion in the frontal convolution on the right side, and which is not within the reach of medicinal or operative treatment. It is not an instance of aphasia, for he knows what he wants to say, but his speech is imperfect. I shall refer him to an elocutionist, in the hope that by education his speech may be somewhat improved. I do not recall ever having seen such a case before.

Graefe's knife will have to be employed. Often the iris cannot be grasped by the common iridectomy forceps, and then you must use the iris forceps of Matthieu, and if these fail you may use Tyrrell's hook. Besides these cases, there are some with staphyloma. I recently showed you a case where there was a total staphyloma. Here there can be no recovery of vision; but if only a part of the cornea be invaded by staphyloma, you may quite often restore vision to the eye. Thus, in the case of a boy recovering from gonorrhœal ophthalmia, where the whole upper third of the cornea was the seat of a dense leucoma, which afterwards became staphylomatous, and where the iris was transparent in the direction of the scar, I restored very good sight to the patient. The first thing I did was to perform Küchler's operation for staphyloma, which consists in dividing the staphyloma throughout its entire length in the vertical meridian. The lens may lie under the staphyloma, and if so you evacuate the lens; if not, you cut from each lip of the horizontal wound you have made a narrow strip of the leucomatous tissue. The eye is now to be kept persistently bandaged for several weeks, when it will be found that there is a flat leucoma instead of a protrusion. After this, you can perform an iridectomy, as I did in the case just alluded to, with the result of securing him very useful vision. As the outcome of long experience, I would say, never make a perfectly hopeless prognosis in ophthalmia neonatorum, except where there has been almost complete sloughing of the cornea.

Laryngology and Rhinology.

NASAL CATARRH.

CLINICAL LECTURE DELIVERED AT THE BELLEVUE HOSPITAL MEDICAL COLLEGE.

BY BEVERLEY ROBINSON, M.D.,

Clinical Professor of Medicine in the Bellevue Hospital Medical College, New York.

NASAL OBSTRUCTION.

GENTLEMEN,—This young lady came to me complaining of marked obstruction in the right side of the nose. With the Jarvis "nippers" I removed sufficient cartilage to relieve the obstruction partially, but, for some unaccountable reason, the tissue on the right side, instead of being cartilaginous down to the nares, seemed to be bony, and I found it necessary to use the Bosworth saw. The operation was done only a few days ago, so that some of the obstruction still present is due to the granulation-tissue; but more tissue will probably have to be removed.

IMPERFECT SPEECH-DEVELOPMENT.

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NASAL CATARRH.

Hippocrates thought that nasal catarrh was a disease of the brain; Schneider was of opinion that it was a constitutional disease, or an irritable condition of the blood which manifested itself by these catarrhal symptoms. The French especially have been supposed to look upon the disease as of diathetic origin, while the Germans have been inclined to attribute it to local causes, and hence have devoted their attention chiefly to the local treatment. In spite of the philosophical way of looking at disease, which can be, I think, fairly attributed to the French school, the present trend of opinion is towards local treatment. There are many people who have an underlying constitutional condition which can be described only by saying that there is a predisposition to catarrhal inflammation, without the existence of scrofula, tuberculosis, syphilis, or gout. When I find nothing explanatory in the environment of a patient, I must naturally admit this etiological catarrhal state. Accompanying this, in very many cases, there is more or less obstruction in the nose, possibly from accidental causes, or from some defect of development. Thus, some individuals have a deviated septum, or some enlargement of the internal nasal structures, etc., producing a certain amount of obstruction on either side, and requiring local interference of an operative kind. If, in an endeavor to relieve an obstruction on one side, a perforation be made in the septum, it will be impossible to heal it up, and it will prove very much more annoying than the original disease.

We know that there are certain drugs which are eliminated through the mucous membrane of the nasal passages, which are more or less useful: one is sulphur, another cubeb, a third ammoniacum, and a fourth ammonium. I prescribed sulphur internally for a long time, and thought it was moderately useful, but the sulphur waters obtainable in this country usually cause dyspepsia when given internally, and they are eliminated from the mucous membrane only to a limited degree. The reason that the sulphur waters of Ritchfield, Sharon, etc., disturb the digestion, is that they contain a large quantity of sulphate of calcium, whereas those of Europe contain a large proportion of sulphate of sodium, which is not injurious. Cubeb is an excellent drug, and has done much good in catarrhal affections *per se*, and in certain forms of diphtheria which I regard as a croupous inflammation of the mucous membrane. We know that one kind of diphtheria is characterized by the presence of the Loeffler bacillus and the other is not, and that the one is genuine and the other is not. I saw a case

to-day which, although having the true clinical symptoms of diphtheria, did not show, on microscopical examination, the presence of the Loeffler bacillus in the secretions of the throat. Cubeb in large doses produces a rash, and sometimes causes gastric disturbance; nevertheless it is one of the best remedies for catarrhal inflammations of the throat. Fraser, of this city, prepares tablets containing about one-fourth of a grain each of chloride of ammonium and of powdered cubeb, with some liquorice. I usually prescribe one of these to be taken at short intervals,—every fifteen or thirty minutes, or every hour, depending upon the case. If there is much cough in connection with the catarrh, I sometimes add some codeia. The liquorice is a demulcent which is not unpleasant to the taste, and is a valuable adjunct. I commonly prescribe ammoniac in the form of the mistura ammoniaci. Carbonate of ammonium in acute catarrhal conditions I still believe is one of the most valuable of all remedies, to be given in large doses frequently repeated. I do not believe there is a drug in the Pharmacopœia which has more control over acute catarrh and cold in the head, and sometimes even when the larynx is involved. I prescribe it with syrup of acacia in doses of two grains, every half-hour or hour; or it may be given in the proportion of one grain of the carbonate of ammonium to each teaspoonful of the mistura amygdalæ, which makes a very palatable mixture. For adults, the dose of this mixture is a dessertspoonful every two or three hours. I am confident it is eliminated through the mucous membrane, and also that there is nothing quite so valuable as this drug. If the catarrhal condition be due to gout, give guaiacum and colchicum; if it be due to syphilis, give mercury and iodide of potassium; if it be due to scrofula or a "strumous" condition, give cod-liver oil, the hypophosphites, or iodide of arsenic, and advise a change of air. But suppose the patient's occupation causes a constant irritation of the nose and throat,—*e.g.*, in a tobacco-factory: you cannot conscientiously treat such a case, because you cannot do away with the cause of the trouble, and hence cannot hope to cure the disease. Again, if a patient is living in a locality in which there are bad sewage and drainage, and he has what are called malarial manifestations, you must not direct your attention entirely to local treatment, but you must explain to him the cause of the trouble, and give the remedies appropriate for this condition, instead of wasting time and money by using sprays. Sir Morell Mackenzie, my deceased and beloved master, used to say that all the catarrh here was due to the filth and bad drainage of the towns in America, and the absolute lack of attention to those conditions which we know are better looked

after in Europe. It is not surprising that so many in this city have catarrh, when nine-tenths of our houses are overheated by furnaces the air for which is taken from the cellars or from the surface of the soil.

Ten or fifteen years ago, almost every physician was spraying or using powders upon the congested and irritable mucous linings of the nose and throat, and for a time we had a good deal of faith in such medication, but the treatment did not long continue popular. Then the nasal douche was very widely used, until Roosa, Knapp, Pomeroy, and others told us that the douche produces only temporary relief and often sets up a purulent otitis. Some patients use this douche properly and with slight temporary advantage, but few can use it with marked and permanent relief. A few patients after using the douche for a long time have found that they have no smell or taste, and that they do not enjoy their food as they did formerly, and they have attributed all this to the catarrh, but in reality it is most commonly due to the douche itself, for its action, when long continued, tends to blunt the sensitiveness of the mucous membrane. The spray is less injurious than the douche, but neither sprays, douches, nor powders can, from the very construction of the nasal cavity, reach all the diseased parts, as the cavities are often small and the passages communicating with them blocked up with secretion. Sprays should not be used either very cold or very hot, and if the strength of the solutions employed is not properly proportioned, endosmosis will take place between the mucous membrane and the solution, with the result of increasing instead of diminishing the thickening. We must at times look upon these methods of treatment with some reasonable doubt as to their efficacy, and also mistrust the individual who ignores the important constitutional conditions underlying catarrh.

If there be an adenoid growth causing much annoyance, it must be removed, and likewise if there be any form of obstruction in the nose which causes much discomfort; but the point I wish to impress upon you is that very few people have perfectly free nasal passages on both sides. I should like to ask any one in the class to test the nasal passages and tell me the result. By actual count, I find that only two out of twenty-seven are able to breathe equally well through both sides of the nose; yet from the point of view of the average specialist the other twenty-five are fit subjects for an operation. If the obstruction of the nose causes aural trouble, it is rational to conclude that the removal of some of the obstruction by one of these methods will give relief; then by all means do it,—I do it every day,—but do not operate

in this way unless you have some definite object in view, or some positive indication for such interference. There is one specialist in this city who does a great deal of this kind of operative work, yet he has, I believe, very large tonsils in his own throat, and will not have them cut out. Another very eminent surgeon here has a hernia, but he prefers to wear a truss rather than submit to operation.

I venture to say that in nine-tenths of the cases of obstructed noses requiring operation you can do it just as well with Weir's forceps as with all the usual expensive paraphernalia; it is not at all necessary to have drills and trephines. With this little saw, devised by Curtis, you can remove easily many obstructions which are not easily managed with the forceps. After such operations there is much granular tissue formed which it is impossible to heal without strong applications and much patience. Eight-tenths of all cases of catarrh I believe are due to a thickened condition of the nasal mucous membrane, and usually this thickened condition can be sufficiently reduced by the proper use of certain acids, and also, when this is convenient, by the electro-cautery. Chromic acid properly used is probably the most efficient agent of this class, but if it cannot be used with a certain amount of deftness it is better to leave it alone. You cannot do any harm with glacial acetic acid, mono-chloracetic acid, and equal parts of carbolic acid and glycerin, so far as I know, for the ulcerations produced by these agents I have always seen heal readily and promptly. In the case of an adult, a crystal of chromic acid may be melted on a probe, or cotton may be moistened with a saturated solution of chromic acid and applied to the desired spot.

The instrument which I believe is most useful for the removal of adenoid growths in the naso-pharynx is one known as Gottstein's curette, which can be introduced into the naso-pharynx well against the septum and pulled down over these vegetations. It usually causes pretty sharp bleeding for a moment. The instrument does not seem to be capable of doing any harm, and it obviates the necessity of using an anæsthetic in a child. Professor Lefferts, of this city, now uses this instrument frequently, but before he employed it, he tells me, he used his finger-nail with a gag in the mouth. A series of sixty or seventy cases have recently been reported in which this instrument has been used without any accident. It is, of course, possible for some of this granular material to be drawn into the larynx, and, although I have never heard of such a case, it is a possibility which you should bear in mind. On the same principle, it is well to think what is advisable to do in case there should be any troublesome bleeding. Sponge or gauze

tampons with a string attached are the best for this purpose, the mouth being held open with an O'Dwyer mouth-gag.

The point of view of the general practitioner and that of the specialist will never be the same, and I have endeavored in this lecture to give you a certain broad appreciation of the subject. Remember that you have no right to operate upon any patient without having thoroughly explained the situation to him and the anticipated result from interference, otherwise you are not treating him fairly.

TREATMENT OF CHRONIC RHINITIS.

CLINICAL LECTURE DELIVERED AT THE VANDERBILT CLINIC.

BY G. M. LEFFERTS, M.D.,

Clinical Professor of Laryngoscopy and Diseases of the Throat, College of Physicians and Surgeons.

THE treatment of the various forms of chronic rhinitis is a subject upon which a great deal has been said and written. I am going to try to remember to-day that I am talking to men who will be general practitioners,—not specialists,—and who will not have at their command a multiplicity of apparatus, more or less costly, and that therefore I must be practical and deal with methods and instruments which are within the reach of every one, whether he be practising in the city or in the country.

How are you going to treat your cases of chronic rhinitis? First examine your patient and find out whether he really has nasal catarrhal trouble. Then determine what form of rhinitis it is. This will take you back to what I said last week,—namely, that there is more than one form, that some are curable and some are incurable, and that the day has gone by when you can class all catarrhal troubles of the nasal mucous membrane as "catarrh." You must recognize that there are at least three forms of catarrhal trouble. The third practical point is that in your conversation with your patient you must let him understand that you are dealing with a chronic affection, and that you cannot help him in as many days or hours as he has had the disease years. Tell your patients what their trouble is, what grade it is in, and how much you can do to help them, and, what is equally true and equally right, what you cannot do. If it be an atrophic or a fetid rhinitis, which will baffle your skill, let your patient understand that from the outset. In some cases you are bound to fail, simply because you cannot regenerate a mucous membrane which has become atrophied to such a degree that restoration is a physical impossibility.

The question whether constitutional treatment is indicated must be

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The question whether constitutional treatment is indicated must be

determined in each case. This point is too often overlooked. Take a patient with a strumous diathesis or a catarrhal diathesis. In each of these forms the constitutional conditions which underlie the local affair must be combated at the same time that you are treating the nasal condition. Cod-liver oil, the iodides, iron, a change of air and occupation, —all of these will suggest themselves to you in certain cases of catarrhal trouble. The majority of cases are treated by local measures alone; but never omit to ask yourself the question, Is there any underlying diathesis that should receive attention? The question will also arise, whether any of the drugs that have been recommended from time to time as having a beneficial therapeutic effect upon the mucous membrane of the respiratory tract in a diseased condition are really of great value. The three drugs that are commonly used are oil of cubeb, muriate of ammonium, and gum ammoniacum. Each of these is supposed to have its influence upon the respiratory mucous membrane, but you cannot cure your case of nasal catarrhal trouble by their use alone.

Absolute cleanliness is a very essential principle in the treatment of catarrhal affections of the nasal passages. This principle practically underlies all others. Ask yourselves, if you please, what benefit can be derived from the application of medicated sprays or powders to a mucous membrane that is protected and covered by a layer of thick, tenacious mucus, or encased in an armor of hard, inspissated crusts. The mucous membrane must be cleaned before your application is made, if it is to be of the slightest efficacy. On the other hand, the matter of cleanliness may be overdone, and much harm ensue. The real or fancied relief that every patient experiences after the use of the nasal douche leads him to employ it at constantly-decreasing intervals. This relief is only temporary. I have known patients to use a strong saline solution in this way six, eight, and even ten times a day. Now, the influence of treatment of this kind, or rather abuse of this kind, is sure to be harmful. The use of these strong saline solutions passed through the nose under considerable hydrostatic pressure is often the cause of the development of catarrhal inflammation. The frequency of your applications and the amount of fluid you use must be brought down to a minimum. When there is simply a mucous or a mucopurulent secretion covering the membrane, it can be easily removed by blowing the nose. No washing out is necessary in these cases. I have discontinued washing out the nasal passages in more than fifty per cent. of my cases, and, being relieved of an element of irritation, the results of treatment are better. In extreme hypertrophic rhinitis, where the mucous membrane bulges out and dams up the secretion in

the naso-pharynx, the patient cannot relieve himself, and some form of cleansing becomes a necessity. And much more does it become a necessity in atrophic rhinitis, where the hard crust blocks up the nasal passages. The douche, the spray, and the syringe will often fail to loosen these scabs, and they must be removed by instrumental means.

How are you going to cleanse the nasal passages? Never use the nasal douche; it is insufficient for the purpose for which it was constructed. It will not clean the upper part of the nasal passages, nor the naso-pharynx, nor the posterior pharyngeal wall. Dismiss from your minds the idea that any case of nasal catarrhal trouble was ever cured by the use of the nasal douche, or ever will be. There is nothing curative in the use of any of these cleansing or washing-out solutions. They are palliative, they are comforting to the patient, and they prepare the way for further remedial measures. The apparatus I prefer for washing out the nasal cavities is this "nasal spray apparatus." It is simply arranged to throw a very coarse spray in the right direction. The conical tip closes up one nostril completely; the fluid then enters one nasal passage and passes out by the other. Power is obtained by means of a double hand-ball tube. With it the nasal passages and upper pharynx may, except in rare instances, be thoroughly cleansed of secretions and crusts by the use of less than one ounce of fluid. Being a coarse spray, it washes up, loosens, and dislodges the secretions. There are cases, however, where no form of spray or wash will answer the purpose. These are the cases of atrophic or fetid rhinitis where the crusts are firmly attached, and where, as I have before said, instrumental interference is necessary.

Various alkaline cleansing and disinfecting solutions may be used to wash out the nostrils. I use, with the nasal spray apparatus, either of the following:

℞ Acidi carbolici, ℈i;
Sodii boratis,
Sodii bicarbonatis, āā ʒi;
Glycerini,
Aquæ rosæ, āā ℥i;
Aquæ, ad Oi.

The quantity of carbolic acid in this solution is necessarily often varied to suit the susceptibility of different mucous membranes.

Or, better still,

℞ Sodii bicarbonatis,
Sodii boratis, āa ʒss;
Listerine, ℥i;
Aquæ, ad ℥iii.

The mucous membrane having been thoroughly cleansed, we come to the direct treatment of the catarrhal mucous membrane. Excluding for the present the use of caustics and of surgical measures, the treatment is based practically either upon the employment of various medicated fluids, used in spray by means of some form of atomizer, or upon the use of medicated powders, applied by means of insufflation. My own experience prejudices me strongly in favor of the spray. I believe the insufflation of medicated powder may be of some service in simple rhinitis; it is of no use in hypertrophic rhinitis, and it is contra-indicated in atrophic rhinitis. I now turn to the medicated spray. I believe that with a proper spray-tube and a sufficient amount of pressure no more perfect application can be made to the parts. Unless the treatment is done thoroughly and efficiently, systematically and persistently, it will fail. There is no royal road to the successful treatment of these cases. In making the application through the posterior nares, the patient should depress his tongue by means of a spatula, and the soft palate should be drawn forward with the palate-hook; if this is not done the soft palate will be drawn upward, approximating the pharyngeal wall, and the spray is useless. While your patient is breathing through the nose, the soft palate drops forward, and you can then grasp it. And now between the soft palate and the posterior pharyngeal wall the beak of your spray-tube is quickly introduced, and the spray thrown upward into the vault of the pharynx and forward through both nasal passages. I usually employ a force of between thirty and forty pounds, driving this atomized solution into all the little depressions and irregularities that exist in the nasal passages.

Various forms of apparatus are employed in making these applications of the spray. By far the most effective is the complete compressed air spray apparatus, like the one you see here. If you do not possess such an apparatus, you can employ one of the hand-ball atomizers, in which the propelling power is developed by the compression of india-rubber hand-balls. This is much less efficient, because less powerful, than the compressed-air spray. Moreover, both of the operator's hands are occupied with the working of the hand-ball spray, so that he cannot use the palate-hook, and I would therefore advise you, if you are obliged to use this form of instrument, to make the spray applications through the anterior nares.

The medicated solutions that I most commonly employ are the following, given in the order of their preference.

In any of these formulæ, "Listerine" may be substituted in part for the water, in the proportion of one part of the former to three of the latter.

1. Zinci iodidi, gr. v ad aq. ℥i;
2. Zinci sulpho-carbolatis, gr. v ad aq. ℥i;
3. Zinci sulphatis, gr. v ad aq. ℥i;
4. Ferri et ammonii sulphatis, gr. v ad aq. ℥i;
5. Ferri chloridi, gr. v ad aq. ℥i;
6. Acidi tannici, gr. v–xx;
7. Potassii chloratis, ℈i.

If the simple rhinitis has advanced far towards the hypertrophic stage, I begin at once with:

Iodini cryst., gr. iv;
Potassii iodidi, gr. x;
Zinci iodidi,
Zinci sulpho-carbolatis, āā ℈i;
Listerine, ℥i;
Aquæ, ad ℥iv.
Use as a spray.

The appropriate cases for medication as suggested above are those of simple catarrhal rhinitis, and certain cases of hypertrophic rhinitis where the hypertrophy is not excessive and where the tissue has not become completely organized. But suppose a case where there are large masses of organized hypertrophic tissue and, beneath this, enlargement of the erectile tissue. Such a case must be treated surgically, and you simply waste your time by employing any form of treatment I have so far spoken of. By surgically, I mean by the cautery or some form of caustic, which will succeed in reducing these hypertrophic masses. First apply a ten-per-cent. solution of cocaine, which will anæsthetize the parts and contract the mucous membrane tightly against the turbinated bone, giving you room to work. Then take a small probe, its end wrapped in absorbent cotton, and saturate this with one of the caustic agents at your command, such as nitric acid, glacial acetic acid, chromic acid, or silver nitrate; of these I always advise nitric acid. Under the guidance of the light from your head-mirror, you cauterize the turbinated bone at its point of greatest convexity, drawing the probe along in an antero-posterior direction. Squeeze out your probe, again dip it in the acid, and again cauterize throughout the whole length of the turbinated bone. Cauterize deeply, but do not smear the acid over the entire interior of the nasal passages. The action of your caustic, aside from destroying absolutely a line of hypertrophied mucous membrane, excites a hyperplastic inflammation of mucous and

submucous structures; as the product of this in time organizes and contracts, it obliterates in part the erectile tissue underlying the membrane and contracts the latter, a process that is aided by the shrinking of the strong cicatrix of the wound that you have made. Altogether the result will be that the redundant mucous membrane is drawn down into its proper place and remains there; the nasal passages are thus freed.

It may be that a condition will exist where the posterior extremity of the inferior turbinated bone is immensely hypertrophied, so that a tumor is formed in the posterior nares, blocking them up more or less completely. Cauterization will not remove such a growth. It can, however, be readily removed by means of the Jarvis snare. The nasal mucous membrane is first anæsthetized by cocaine. The snare is passed through the nasal passage until it slips over the hypertrophied mass, which is gradually cut away.

The treatment of atrophic rhinitis is a hopeless one. Your only indication is to keep the nasal passages perfectly clean and then lubricated by the use of vaseline, albolene, benzoinol, etc.

FOLLICULAR TONSILLITIS; INTUMESCENT RHINITIS—CAUTERIZATION; HYPERTROPHIC RHINITIS.

CLINICAL LECTURE DELIVERED AT RUSH MEDICAL COLLEGE, CHICAGO.

BY E. FLETCHER INGALS, A.M., M.D.,

Professor of Laryngology and Practice of Medicine, Rush Medical College; Professor of Diseases of the Throat and Chest, Northwestern University Woman's Medical School; Professor of Laryngology and Rhinology, Chicago Polyclinic, etc.

Case I.—This man asked me to look at his throat after he had already made a correct diagnosis of his disease. It is a case of acute follicular tonsillitis. The trouble began night before last with a general feeling of discomfort, followed after a few hours by aching and lameness in every part of his body, accompanied by a sensation of irritation in the left tonsil, which gradually became painful. The next morning after this onset his knees and other joints ached as though with an attack of acute rheumatism. To-day he is sweating freely and still complains of some lameness and headache, and speaks of a feeling of fulness or swelling in the throat, by which his voice is evidently modified. Upon deglutition he experiences pain, the same in amount whether he takes liquids or solids. Often patients complain more upon swallowing liquids than upon swallowing solids; in acute inflammation of the tonsils the reverse is usually true. Upon looking into this patient's throat, I find the tonsils moderately swollen and red; and upon each two or three small, white or yellowish-white patches, which have the appearance of being depressed, and are six or seven millimetres in diameter. The palate and uvula are slightly reddened and relaxed. The pharynx is not markedly so, but it contains an abnormal amount of mucus.

There are not so many of the follicles involved here as we usually find, and I think the depression of the yellowish patches, though characteristic, is somewhat greater than is generally observed. Not

submucous structures; as the product of this in time organizes and contracts, it obliterates in part the erectile tissue underlying the membrane and contracts the latter, a process that is aided by the shrinking of the strong cicatrix of the wound that you have made. Altogether the result will be that the redundant mucous membrane is drawn down into its proper place and remains there; the nasal passages are thus freed.

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BY E. FLETCHER INGALS, A.M., M.D.,

Professor of Laryngology and Practice of Medicine, Rush Medical College; Professor of Diseases of the Throat and Chest, Northwestern University Woman's Medical School; Professor of Laryngology and Rhinology, Chicago Polyclinic, etc.

Case I.—This man asked me to look at his throat after he had already made a correct diagnosis of his disease. It is a case of acute follicular tonsillitis. The trouble began night before last with a general feeling of discomfort, followed after a few hours by aching and lameness in every part of his body, accompanied by a sensation of irritation in the left tonsil, which gradually became painful. The next morning after this onset his knees and other joints ached as though with an attack of acute rheumatism. To-day he is sweating freely and still complains of some lameness and headache, and speaks of a feeling of fulness or swelling in the throat, by which his voice is evidently modified. Upon deglutition he experiences pain, the same in amount whether he takes liquids or solids. Often patients complain more upon swallowing liquids than upon swallowing solids; in acute inflammation of the tonsils the reverse is usually true. Upon looking into this patient's throat, I find the tonsils moderately swollen and red; and upon each two or three small, white or yellowish-white patches, which have the appearance of being depressed, and are six or seven millimetres in diameter. The palate and uvula are slightly reddened and relaxed. The pharynx is not markedly so, but it contains an abnormal amount of mucus.

There are not so many of the follicles involved here as we usually find, and I think the depression of the yellowish patches, though characteristic, is somewhat greater than is generally observed. Not

infrequently patients having an attack for the first time are very ill. In such cases, especially in children, the temperature not uncommonly reaches 103° to 105°; in later attacks it is not apt to be over 102° or 103°. The diagnosis ought, as a rule, to be made readily by simple inspection, but in some instances it is not easy. Occasionally a case beginning as a simple follicular tonsillitis will terminate in scarlatina. In such an event the redness is apt to be more marked upon the fauces, the posterior wall of the pharynx, and the palate, usually diffused, but sometimes in patches; while in tonsillitis redness and swelling are largely confined to the tonsils. If there be local pain in scarlatina, it is within the throat, and is not apt to be accompanied, as in tonsillitis, by stiffness at the angles of the jaws and more or less difficulty in opening the mouth. In the one disease there is the strawberry tongue and commonly the characteristic rash; in the other the tongue is coated with a yellowish fur, and there is seldom an accompanying eruption upon the skin. Some cases of tonsillitis are unquestionably of diphtheritic character. However, in diphtheria the tonsils are not so apt to be enlarged as in the other affection. There is usually no difficulty in opening the mouth, and the thick grayish-white membrane covers a larger area than the lacunar patches in tonsillitis, and may not be confined to the glands. The one or two large patches in diphtheria are distinctly raised above the surface; the usually numerous patches in tonsillitis are depressed, or, if raised, but slightly so. If forcibly detached, in the one case a raw, bleeding surface remains, not so in the other. The temperature in tonsillitis is much higher than in diphtheria, where it may be subnormal. There is apt to be enlargement of the neighboring lymphatic glands in the latter disease, not in the former.

There is, furthermore, in many cases of diphtheria a certain odor which cannot be described, but which I consider peculiar to the disease. I do not know that it is so recognized universally. Though in tonsillitis there is sometimes a very offensive breath, it has not the penetrating, sickening odor peculiar to diphtheria. In an attack of follicular tonsillitis a person is generally ill from four to eight days, after which he makes a rapid recovery without sequelæ. There are rare instances in which slight paralysis follows. Occasionally one recovers in forty-eight hours, or a little longer, without much medicine.

As to treatment. In the very beginning the bowels should be freely opened by a saline or mercurial cathartic. I think also that the recently-introduced antipyretics are valuable, and of these I consider phenacetin most effective in relieving discomfort. In this case I would order ten grains of phenacetin to be repeated each hour two or

three times; no more to be taken for several hours. Its sometimes depressing effects upon the heart may be guarded against by combining caffeine with it. Aconite, opium, and belladonna, in small, frequently-repeated doses, often act satisfactorily in cutting short the disease. The first should be given in half-minim doses of the tincture every fifteen minutes till the occurrence of free perspiration, afterwards once an hour for several doses, and subsequently with less frequency according to its effects. Tincture of opium in minim doses should be given every fifteen minutes till the cessation of pain in the throat, and subsequently at intervals of several hours as indicated; tincture of belladonna in a similar way in half-minim doses. If it is strongly suspected that the case is of rheumatic origin, the anti-rheumatic remedies are of prime importance. It is held by some that a large number of cases are of septic origin; probably some of these are truly diphtheritic in character. In such cases tonics are indicated, especially the tincture of the chloride of iron with strychnine or nux vomica.

As to local medication, I am able to afford most relief by the application of silver nitrate in a solution of sixty grains to the ounce, or of a solution of carbolic acid and tannic acid of each thirty grains, and morphine four grains, in water and glycerin, half an ounce of each. This will smart, but will give the patient great relief for from twelve to eighteen hours, and seems to go far towards checking inflammation. The former solution, if used in the very beginning, will sometimes abort the disease; it is not so efficient after the first forty-eight or seventy-two hours. The case before us has gone on past the worst point. It is already progressing towards recovery. I therefore shall advise simply the administration of tonics combined with a mildly sedative local application, as promising the best results. I shall recommend him to use a solution of about one and a half per cent. of carbolic acid as a gargle every hour, and internally tincture of iron as often as once in three or four hours, and quinine two grains about four times a day, with an occasional mild laxative.

Case II.—This man complains that his nose stops up when he is lying down. This has troubled him for a year, and it is much the worst on the left side. He complains of the dropping of mucus from the back part of the nose, or, as he expresses it, from the head; and also of a cough at night, which I have no doubt is due to the dropping of mucus into the throat. This increased secretion is due partly to increased activity of the mucous glands, partly to interference with evaporation by occlusion of the passages. When he lies upon his right side the right naris becomes closed, the left naris remaining open;

when he lies upon his left side the left is occluded and the right open. The difficulty which he experiences in breathing is characteristic of the condition which we find in his nose. Inspection with reflected light reveals the turbinated body of the left side so swollen as to half close the normal passage. That of the right side is not swollen so much.

This affection has existed for a year: we therefore expect very little benefit from local medication. In cases of this sort I know of no treatment which will justify a physician in taking a patient's time and money, except the use of the snare or of the cautery in some form. When you have no galvano-cautery, chromic acid may be employed, but it is far inferior to the former agent, both as regards the comfort of the patient and the time necessary to effect a cure. This case shall serve to illustrate the use of the galvano-cautery. The parts should be thoroughly anæsthetized by cocaine in four-per-cent. solution, applied carefully by pledgets of absorbent cotton wrapped on a probe. The cauterization is made by means of the knife-electrode, which, properly connected to a strong battery, is passed flatwise into the naris until it reaches the posterior portion of the turbinated body, when it is turned outward, so that the wire, which is to be heated, comes against the tissue. The current is then turned on and the instrument is drawn forward slowly, burning a groove down to the surface of the bone through the soft parts. If it does not cut rapidly and completely on the first passage it may be moved backward and forward over the same ground. As soon as the front end of the turbinated body is reached, the electrode is lifted from the wound and the current cut off.

The proper heating of the cautery is an important part of the operation. The current should be sufficient to produce a white heat in two seconds. The red heat does not burn sufficiently, and a heat beyond what is requisite produces bleeding by cutting too keenly.

After a cauterization the patient should be seen in the course of four or five days, in order to make certain that there is no adhesion forming. Very often at this time it will be found that the large pellicle or exudation formed as a result of the burn, nearly an eighth of an inch in thickness, may be removed *en masse*. That will give the patient very great relief. It is usually well in this event to touch the part with a solution of silver nitrate about ten grains to the ounce. The patient need not be seen again for about ten days, when the other side of the nose can usually be cauterized; but the procedure can rarely be repeated within a week without starting severe inflammation, in which the side first cauterized is apt to participate; an interval of

two weeks or more should generally be allowed. For forty-eight hours after a cauterization the patient should wear a little cotton in the nostril when out of doors. For home use I give a powder containing four per cent. of cocaine, and direct the patient to use a small amount of it four or five times a day for four or five days, with a view to keeping down the swelling and preventing the formation of adhesions. Occasionally a patient does not need it; if the nose does not stop up, its use is unnecessary. In conjunction with the powder it is well to advise a spray containing half a grain of menthol, half a grain of carbolic acid, and three minims of the oil of cloves to the ounce of liquid albolene, for use two or three times a day. At the end of four or five days I give three parts of a similar powder, mixed with one of iodole, to be used twice a day if the nose stops up, once a day if it does not. This is the treatment also for hypertrophic rhinitis. In rhinitis intumescens, an example of which is now before us, a single cauterization on each side is sufficient in about one-fourth of the cases: not more than two will be needed in nine-tenths of the cases, and three or more in comparatively few. Be careful not to destroy any tissue unnecessarily. As soon as the nose is free the patient will be largely rid of that disagreeable sensation in the throat of falling mucus, which is so often the chief complaint.

INTUMESCENT RHINITIS.

CASE III.—This young man comes to us complaining of having been out of health for two or three years. His chief trouble is considerable stopping up of the nose, which for the past eight or ten months has been continuous. Before that time he had been subject to frequent colds in the head, and intermittent closure of the nares, particularly at night, with considerable discharge of mucus from the nostrils and dropping into the throat. The latter has been very annoying of late, and is accompanied by hawking and sometimes gagging and vomiting. He has also suffered from partial deafness, mostly of the right ear, which is worse at times and associated with much tinnitus aurium. A dull frontal headache has affected him almost continuously for the last six months, at times accompanied by a feeling of fulness at the root of the nose, and some weakness of the eyes, which "easily water." He says he sleeps with his mouth open, that his throat feels dry and irritable in the morning, and that he has frequent attacks of sore throat, hoarseness, and cough, especially in winter. At present he has no cough, but there is a good deal of pain in the chest, particularly on exertion. Examination of the thorax reveals nothing of a positive

nature to account for the pain. Upon inspection of the nose, I find the cavities are about four-fifths closed by enlargement of the inferior turbinated bodies; there is also an exostosis on the left side. The mucous membrane is abnormally red, and fine cobweb-like fibres of mucus stretch across the cavities from side to side, as if the two surfaces had been in contact and in separating had drawn out these films of viscid mucus. A view of the vault of the pharynx reveals abnormal redness of the mucous membrane except that covering the posterior ends of the turbinated bodies, which are so enlarged as to project far into the naso-pharynx. They are of a grayish hue, their surface somewhat nodular, and they almost completely occlude the posterior openings to the nasal cavities. There is also considerable viscid, muco-purulent secretion which extends down the posterior wall of the pharynx so as to be visible to direct inspection. The pharynx itself is dry and relaxed, and numerous prominent follicles and enlarged veins are apparent.

I have drawn out this man's history and symptoms somewhat at length because they are typical of this quite common affection, hypertrophic rhinitis, though all patients have not so large a congeries of symptoms. He complains of the pain at the lower portion of his chest on both sides, over the short ribs, hence along the attachment of the diaphragm. As I press upon the chest it does not aggravate the pain. The three tender points characteristic of intercostal neuralgia are absent. He has never had rheumatism, and has now no evidence of pleurodynia. If the pain were pleuritic, it would probably be confined to one side and be accompanied by peculiar physical signs, none of which are here present. Localized pain complained of as seeming to be in the lung itself is positively very rarely significant of pulmonary or pleural disease. My belief is that this pain is occasioned by the undue exertion thrown upon the diaphragm by the manner of breathing. The patient is obliged to make an extreme effort to draw in sufficient air to expand the lungs, owing to obstruction of the nares. The opening of these cavities would undoubtedly relieve the pain.

HYPERTROPHIC RHINITIS.

CASE IV.—The condition of this man's nose is chiefly distinguished from intumescent rhinitis, an example of which was just considered, in being continuously occluded. This patient has passed the stage of markedly intermittent swelling of the turbinated bodies. The soft parts have by repeated congestions become permanently thickened, and the bone itself is involved in hypertrophy. The bodies therefore no longer

contract to normal size under the influence of cocaine, nor yield easily to the pressure of the probe. The condition is not to be mistaken for a nasal polypus, which would be more elastic, and may be manipulated with the probe which can be passed between it and the outer wall of the nose, a thing not possible in the other affection. The posterior end of an inferior turbinated body might be mistaken for a polypus, but the latter is lighter in color and more translucent, and its surface is smooth, while that of the former is nodular and uneven. The treatment in this case is essentially the same as that recommended for intumescent rhinitis, except that redundant tissue must be removed. This may be accomplished by means of the galvano-cautery, by chemical caustics, or by scissors, burrs, trephines, or the écraseur. It is better to cauterize the soft tissues, as long as we can give the patient considerable relief by so doing, than to begin directly upon the bone itself.

Dermatology.

ON SOME CUTANEOUS DISEASES.

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL.

BY LOUIS A. DUHRING, M.D.,

Professor of Dermatology in the University of Pennsylvania.

CASE I.—ALOPECIA AREATA.

GENTLEMEN,—The first case I show you to-day is one of alopecia areata, occurring in a child five years of age. On the occiput she presents a well-defined smooth spot devoid of hair. The disease is a chronic one, and usually begins as one or more areas, which gradually enlarge and finally coalesce, or may remain stationary for a variable length of time. The skin soon shows signs of altered nutrition. After a time colorless down or normal hair may appear on the bald area. This drops out usually and grows in again, or it may remain, the scalp sooner or later returning to a normal condition. The diagnosis from ring-worm of the scalp, in most cases, is not difficult. The prognosis is usually good in young persons, though at times the disease yields only after a long period.

Arsenic is generally indicated, but often it fails. Locally, a lotion composed of carbolic acid f℥i and alcohol f℥vii is of benefit, and will be used in this case. In obstinate cases it is sometimes useful to blister with carbolic acid. After an epidermic eschar has formed, this must be discontinued for a week or longer, and then reapplied. Chrysarobin is of even greater value, but, unfortunately, is not safe, unless applied under the observation of the physician.

CASE II.—TUBERCULAR SYPHILODERM.

The lesions presented by this woman are of two years' standing, and are now in a stage of involution. This form of the disease usually appears a long time after the initial lesion, as a late manifestation. It often comes on insidiously. The lesions may terminate finally by absorption, leaving a pigmented spot, or, as in this case, by ulceration. No history of syphilis can be obtained from this patient,

which is often the case with the tubercular syphiloderm, especially in women. This eruption is not infrequently the first manifestation of syphilis presented upon the skin, sometimes several years after the initial lesion. The treatment which will probably be found of most benefit here is as follows:

℞ Hydrarg. iodid. rub., gr. ii;
Potass. iodid., ℥i;
Aquæ menth. pip., q. s. ad f℥vi.
M. et sig.—One teaspoonful with water t. i. d. after meals.

It is well to vary the vehicle from time to time, using elixir of calisaya or syrup of orange-peel as a substitute for peppermint water, since the same remedies must be used more or less continuously for a long time.

CASE III.—ULCERATING TUBERCULAR SYPHILODERM.

The next patient, a middle-aged woman, is also suffering with a tubercular syphiloderm. She presents a tubercular eruption upon her hands and wrists, together with numerous characteristic scars. The lesions have existed about a year. In places they are pustular, and they tend to sink in and destroy the cutaneous tissues. The skin in other places has become thickened and scaly. Some lesions present a tendency to the characteristic horseshoe form.

The constitutional treatment will be similar to that last ordered for the previous case. Locally, a plaster of mercury and salicylic acid will be applied and worn constantly. This is a better method than rubbing in an ointment. These plasters, made of various medicaments and strengths, will be found extremely useful in many chronic skin affections.

CASE IV.—IMPETIGO CONTAGIOSA.

This boy, aged ten, has been suffering with the disease about one week, and now presents numerous discrete pustules and vesico-pustules, pea-sized, superficial, and having the appearance of being stuck on, which, with the other symptoms, serves to characterize it from ecthyma. Drying up in a few days, the crusts drop off, leaving no trace. As a rule, the tendency of this disease is toward spontaneous cure, which may be materially aided by the following oiutment:

℞ Hydrarg. ammoniat., gr. xv;
Ung. zinci oxid., ℥i.—M.
Sig.—Apply thrice daily.

CASE V.—LUPUS ERYTHEMATOSUS.

This woman has been before us on several occasions, and you will recognize the disease as lupus erythematosus. There are several circumscribed, dark-red, chronically-inflamed patches, covered with greasy scales, on each side of the face. There is also a small patch on the ear, which is not an infrequent seat of this disease, though it usually affects the nose and cheeks. She has been using a salicylic acid paste on one side and sulphur ointment on the other. Both sides have improved, but the paste seems to have had the better effect. The sulphur ointment will be discontinued and a mercurial plaster substituted. These plasters are cut to fit the lesions, and should be worn constantly.

CASE VI.—ACNE.

The next patient presents a typical case of papular and pustular acne, affecting the nose, cheeks, and chin, and extending over a period of five years. The disease is inflammatory, usually chronic, and may appear in the form of papules, pustules, tubercles, or a mingling of these lesions, usually upon the face, but sometimes upon the back and chest, particularly in men. It is a common affection, and generally appears between the ages of fifteen and twenty-five, though it may last until the age of forty or fifty. As in this case, the lesions are often sluggish and indurated, when stimulating remedies are required, such as Vleminckx's solution. This must be diluted with from one to three parts of water, and the skin moistened for periods of ten or twenty minutes twice a day. After some days more or less exfoliation usually occurs, when the remedy may be discontinued and sulphur ointment substituted. It is usually necessary in the treatment of acne to change remedies at frequent intervals, as generally they lose their power in a short time. Then, too, different cases yield to different lines of treatment, some improving more rapidly under the application of a lotion, while others yield more readily to an ointment. Frequent bathing of the face with hot water, drying before remedial application is made, is useful in some cases. Of all remedies the one suited to the largest variety of cases is sulphur, though corrosive sublimate, white precipitate, and other mercurials are also useful. As a lotion the following formula will be found of value:

℞ Sulphur. præcip., ℥iss;
Acid. boric., f℥ss;
Ætheris, f℥i;
Aq. Cologniensis, f℥ss;
Alcohol., q. s. ad f℥vi.—M.

Sig.—Apply with the sediment as a wash twice daily.

CASE VII.—PSORIASIS.

This large and stout woman, of forty-five, presents an inflammatory disease of the skin, of eight years' standing, occupying the back of her hands and forearms, the palms being free. The lesions, which are somewhat modified by treatment, are superficial, confluent, scaly patches. The skin has become thickened and infiltrated. The disease usually disappears during the summer, but returns in winter. It is a chronic affection, some patches remaining indefinitely. It is an inflammation, produced by hyperplasia of the rete mucosum; is not contagious, and usually yields to treatment, though recurrence is the rule. Arsenic is of value, especially in the chronic form of the disease, though local remedies are those to be especially relied upon. Liquor potassæ and other alkalies are of service in some plethoric subjects, and this woman will be given ten drops of the former, three times a day, largely diluted with water, to be continued for a few weeks. The scales may be removed by frequent alkaline baths. An application of tar-ointment with salicylic acid, fifteen grains to the ounce, rubbed in from ten to twenty minutes twice daily, is useful. Chrysarobin, ammoniated mercury, and pyrogallic acid are also all valuable remedies.

CORNU UNGUALE.

CLINICAL LECTURE DELIVERED AT THE ST. LOUIS COLLEGE OF PHYSICIANS AND SURGEONS.

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GENTLEMEN,—Cutaneous horn, while not a rare affection, is sufficiently uncommon always to excite more or less interest. As a rule, you will find that it occurs most frequently about the face and the genitals of males. Foulerton has divided these growths into horns which are the result of the overgrowth of a toe-nail, those which are derived from atheromatous cyst-walls, and finally the papillary horns, which take their origin from warts or papillomata, this last category being by far the most frequent in occurrence. The horn of cutaneous origin is always keratodermic in origin and in form, as well as in structure. It is a true hypertrophy of the horny layer of the skin, and its growth seems to be almost indefinite so far as length is concerned, whereas it is always limited so far as the extent of its base is implicated.

In the case which I wish to present we have such a one as is unique, so far as I have been able to find after a careful examination of the literature of affections of the skin. It is one whose appearance does not seem to correspond at all either to the general description or the morphological elements of cutaneous horn. You will observe that the subject is a young man, a little over thirty years of age (Figs. 1 and 2), of mixed Mexican and American descent, who states that he has been affected with his present condition since birth. Upon an examination you will observe the following. Growing perpendicularly from each finger- and thumb-nail there is a horn whose base is the size of that of the nail, and whose length is about four inches. All these horns show a slight curvature towards the dorsum of the hand. They are firmly attached to the nails, and are movable only so far as the attach-

FIG. 2.—Cornu unguale, dorsal view.

FIG. 1.—Cornu unguale, view of palms.

ments of the nails will permit. There is no marked pain connected with ordinary movement, this being elicited only when the movement is so great as to affect the bed of the nail. The horns are not perfectly round in cross-section, but seem to be more or less ribbed, as we find in the case of cutaneous horns. One of these horns, the one on the second left finger, has been broken off, and the patient states that this was done accidentally, and he felt no particular pain or discomfort except at the time when this accident occurred. Upon examining the palms of the hands you will find that a curious condition exists. We have here a condition which simulates very closely tylosis, yet it is not tylosis, because these horny masses are congenital and not acquired. You will find that in the right hand, near the wrist, in the centre of the palm, there is a large horny mass, more or less fungous in external appearance, attached firmly to the skin, which is freely movable upon the connective tissue beneath. On the ulnar side, a little below this, there is a pea-sized excrescence, while the palmar surfaces of the thumb and little finger are involved by similar horny growths, which occur in a more or less linear shape, all the other fingers being also the seats of small masses of horny tissue. The left hand is similarly affected in the lower portion of the palm near the wrist, while the ulnar edge of the palm has three of these small, horny formations; the thumb and little finger are also involved on the palmar surfaces by linear masses of this same hypertrophy of the horny layer, the other fingers not being so extensively implicated, but still showing well-defined formations, which are circumscribed in character. The condition which you observe upon the hands exists also upon the feet, but as the subject derives his living from an exhibition of this peculiar deformity, and does not show his feet, he pares the horns off his toe-nails. They are continually growing, and grow comparatively rapidly. The soles of the feet are implicated in the same manner as the palms of the hands, particularly in that part of the anterior portion which is a little posterior to the digital clefts in the centre. This causes him a great deal of pain, on account of the pressure occasioned in locomotion and by the constant growth of these horny masses, aided as it is by the stimulation afforded through the pressure occasioned by standing and walking, which makes the tenderness in those localities still more accentuated. The heels suffer equally, and along the external borders of the soles there are numerous small horny masses disseminated in the same manner as that in which we find them upon the hands.

This unique condition is one of rare interest, not only on account of the fact that it is the only one in existence, but also because it seems

not to share one particularly well-known element of all keratodermic growths,—that is, of having occurred as the result of some external irritation. In his case the growth is entirely congenital. He was born with this peculiarity. And the peculiarity further lies in the fact that the hypertrophy of the nail—which this certainly is—is not of the form in which we usually find it. As you are aware, nails when they hypertrophy generally do so in their length; and when they hypertrophy in their thickness there is a certain limit to the amount of growth which takes place. The thickness may be as much as a quarter of an inch, but it stops there, and the nails are then ribbed, or have longitudinal or transverse furrows, or there may be one thick longitudinal rib, such as you frequently see as the result of accident. But in this case the growth seems to have taken place according to the disposition of the laminæ of the nails, just as you observe in cutaneous horn occurring upon the skin, where the hypertrophy takes place by horizontal accretions,—that is, in the same direction in which the layers of the horny tissues are disposed. These horns, furthermore, resemble ordinary cutaneous horn in their morphology in this respect, that they are ribbed longitudinally, and the examination of a portion shows that there is horizontal striation as well,—that is, there is a double striation, a condition which is commonly observed in cutaneous horn. The horny hypertrophy occurring on the palms and soles is not due to any friction, nor to any external stimulation or irritation, for the subject has never worked, and has carefully avoided all such means as would irritate his palms and soles, except so far as he could not avoid walking. He has found out one thing, however, and that is that irritation will rapidly increase the growth of the horny tissue in those places where it already exists, and there is a marked tendency in the other portions to the formation of tylotic patches, showing that there is an inherent susceptibility to a proliferation of the elements of the horny layer of the skin.

This deformity does not particularly incommode the subject in the ordinary duties and functions to which his hands are supposed to respond. He can pick up pins and needles; he rolls his own cigarettes; he puts his hand in his pocket and takes out whatever articles he may wish to have; and in general it is only through some unforeseen accident that any injury is likely to happen to him, and the attachment of the bases of these horns to the distal phalanges of the fingers is so firm that, as you see, one of the horns was broken off before the nail was torn away from its bed. A close examination fails to show any indication of a nail as we generally understand it. We can

see that the lower surface is attached in a normal manner to its bed, but there is no border of a free nature such as we observe in the normal nail. It seems rather to be round or oval in shape, and the growth is upward instead of being in the direction of the distal extremity of the fingers. So far as can be judged, these horns are very light. While the tissue is hard and resisting, there does not seem to be any marked density occurring in connection with it. A prominent feature in which it differs from ordinary horny growths is the fact that we do not have that little raised border around the growth which we usually observe in cutaneous horn. If there be any particular form to be observed, it is that the external surface arising from the nail is rather curved, with a tendency to be conical for a certain distance, while the entire growth itself is somewhat smaller at its distal than at its proximal extremity.

There is no irritative process to be observed about the bases of these growths, such as we are accustomed to see in any considerable cutaneous horn, and the conclusion to be derived from this is that there is no tendency for it to assume an epitheliomatous process, which, as you know, is the normal termination of cutaneous horn. It may be argued that the patient is yet too young for any malignant process to set in; but, on the other hand, the length of time—over thirty years—during which these growths have existed would certainly have some influence in producing, if not a malignant process, at least an ulcerative and destructive one, were there any tendency for such a condition to arise. Even in the horny growths of the soles of the feet and upon the nails of the toes, although there is subjective friction and irritation, and all those causes which would naturally predispose to ulceration, we find that there is merely pain, and that there is no evidence of any irritation of an inflammatory nature; in fact, it is a question in my mind whether a great deal of this pain is not purely neurotic in character, for the distribution of the hypertrophy of the palms and soles is very suggestive of a neurotic element for its origin, and we know that there are cases of horny growths of papillomata of a warty nature in which a distinct neurotic element can be traced as an etiological factor. In addition to this, it has been observed that the prolonged administration of arsenic is sometimes the cause of horny growths in the hands. Tylosis of the hands due to the prolonged administration of arsenic is not a rare occurrence, any more than are the objective symptoms of herpes zoster and other herpetic affections, and, as we know, the prolonged administration of arsenic has an influence upon the nerves, particularly those which are concerned in trophо-

neurotic changes acting upon the cutaneous surface, and especially upon the horny layer of the epidermis.

The tendency of these growths, as manifested in the present instance, is to grow indefinitely, and after a certain time a condition will be observed such as we see in this case,—viz., a change in the direction of growth, this direction being in a spiral, and then continuing in this manner for an indefinite period of time. I have had occasion to see this same condition occur in a mule. There was an hypertrophy of the hoofs, which, as you know, correspond to the human nails, and this hypertrophy had been permitted to go on. It was of such an extent that the hoofs grew to the length of seven or eight feet, the general form being that of a spiral, after having grown in a straight direction for probably eight or nine inches. This is the only other analogous condition which I have ever had occasion to observe. The present condition will continue indefinitely, as I have stated, unless the growth be cut, and thus limited in extent.

So far as treatment is concerned, there is but one thing to do, and that is to prevent any further growth. The patient is averse to having this done, from the fact that he makes his living by exhibiting his deformity. But in case such a step should be necessary or desirable, it will be well to bear in mind that the mere cutting of the growths will not accomplish the purpose. It is absolutely necessary to destroy all those elements which participate in the proliferation of the horny cells which go to produce the growth, and in order to do this it is absolutely necessary to take the nail and separate it from its bed, seeing also that no particle is left in contact with the matrix; and, further, to use the electro-cautery and destroy the entire nail-bed and matrix thoroughly. If but one small particle be left remaining, it will form the focus for a new development and the starting-point of a new growth.

So far as the tylosis of the horny formation which occurs upon the palms and soles is concerned, this may be relieved by removing the greater portion by means of the knife, followed by the energetic use of keratolytic agents. But this will not be sufficient. The tendency of this hypertrophy and horny layer to remain must also be overcome, and, as it seems to be of neural origin in this case, probably the best method would be the use of general nerve tonics, such as are especially directed to the sympathetic system, and to the trophoneurotic elements of that system. In that way, by bringing about a better condition of the nerves, and giving greater tone to the so-called trophic nerves, the proneness to trophic disturbance which is manifested in this case by this peculiar horny growth will be partially, if not entirely, overcome, and ordinary irritation, such as is encountered in the daily vocations of life, will produce no more than the common callus which nature throws around as a protective against these irritating influences.

So far as a prognosis is concerned, there need be very little fear entertained as to the ultimate results in this case. As I have stated already, the horns may continue growing indefinitely, unless they are prevented from doing so by being cut off. The horny growths in the palms and soles would seem, from present indications, to have a tendency to take on the following form: the first, middle, and ring fingers will very probably be affected in the same manner as the thumb and little finger,—that is, there will be a linear distribution of the horny masses, and this may not be limited to the fingers, but may also encroach upon the palm, and it would not be surprising to see the entire palms and the entire soles affected by this growth unless there be some means taken to prevent it. I have already indicated the method which should be employed. If it were necessary for the individual to engage in manual labor, he would suffer in proportion to the amount of horny growth present, just as he now suffers rather acutely from the disseminated growth which occurs upon the soles. However, even under such circumstances, even with the involvement of the entire palms and soles, the trouble would be amenable to rational treatment, for it must not be forgotten that this growth involves only the horny layer of the epidermis, and, although there may be some slight implication of the cutaneous layer of the true skin, it is only a compensatory hyperplasia for the better protection of the layers underlying the hypertrophy which is present. As it is now, a peculiar feature is present in the case. The pulps of the fingers are not affected. There is normal sensation present in them, as well as sensibility, and the touch is rather delicate, as is shown by the fact that the individual can pick up the finest needle, can thread a needle, and can seize very small particles of various materials placed upon a surface limited in extent. He can distinguish between smoothness and roughness. In fact, he is quite dexterous, in spite of the deformity which exists. Sensibility of the nail-bed is not so great as we observe it normally, and this is probably due to the fact that it has been subject to more or less irritation.

One other point to which I wish to direct particular attention in connection with the appearance of these horns is this: the nail-fold, as you will observe, is somewhat dimly marked, and it has grown over the surface of the horn for a considerable distance, a condition which

is not uncommon in those who do not take proper care of their nails. You will find that there is a tendency in a great many to the formation of pterygium of the nails, as it is called. The nail-fold becomes firmly attached to that portion of the nail which overlies the lunula, and is gradually dragged along by the nail itself during its slow growth. But there is a limit even to this. You will find that after a certain length has been attained the pterygium does not become any greater. Even in this case, where there is so enormous an hypertrophy of the nail-tissue, the nail-fold is comparatively small in proportion to the amount of surface involved. The fact of finding this fold is a very important one in the recognition of the trouble, for it is proof positive that the affection is hypertrophy of the nail, as one of the most marked morphological characteristics of the nail still remains, and upon examination it will be found that, although there is also an extension of membrane from the palmar surface, by feeling carefully the free border of the nail can be indistinctly made out,—another proof of the fact that there exists an hypertrophy of the nail, occurring in an aberrant manner, it is true, but still an hypertrophy of the nail. It is for this reason that I have called the disease cornu unguale, or nail-horn, because it is a true horn springing from the nail, due to hypertrophy of the nail, and composed of the same morphological elements as the nail, and not having its origin, as ordinary cutaneous horn has, in papilloma, or wart.

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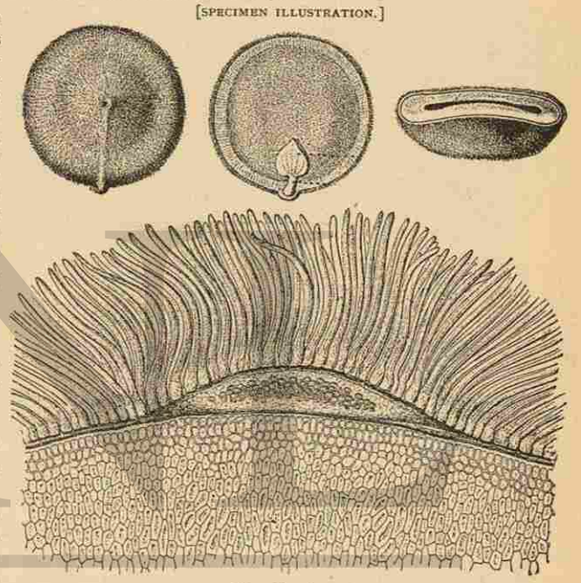

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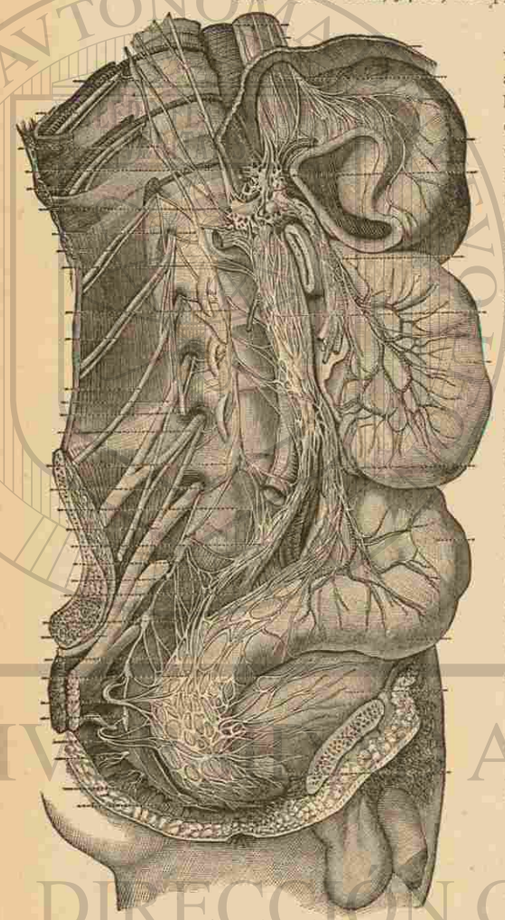

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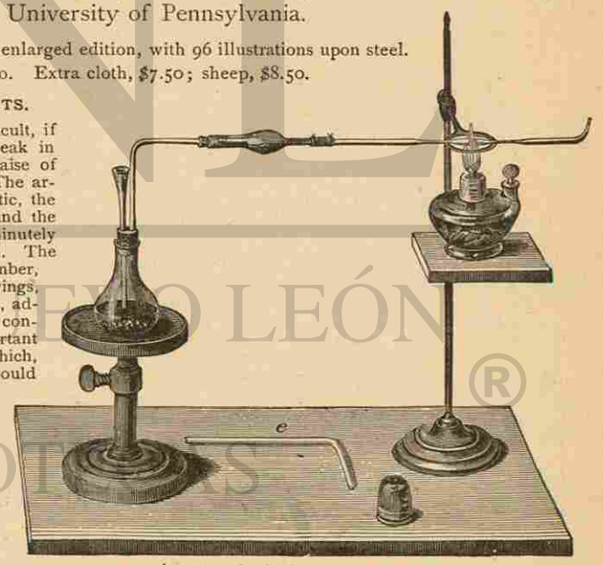

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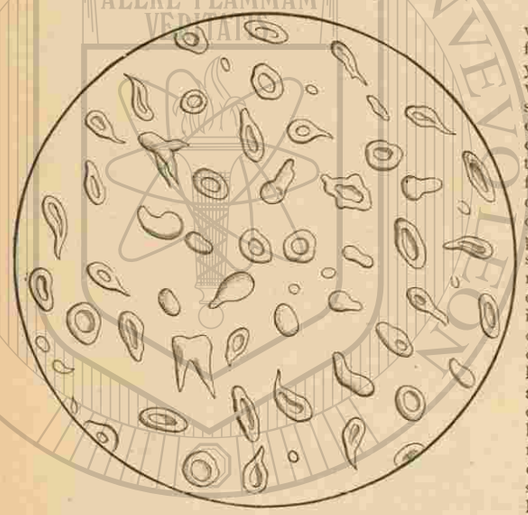

Blood in pernicious anæmia.
[Illustration from chapter on "Diseases of the Blood."]

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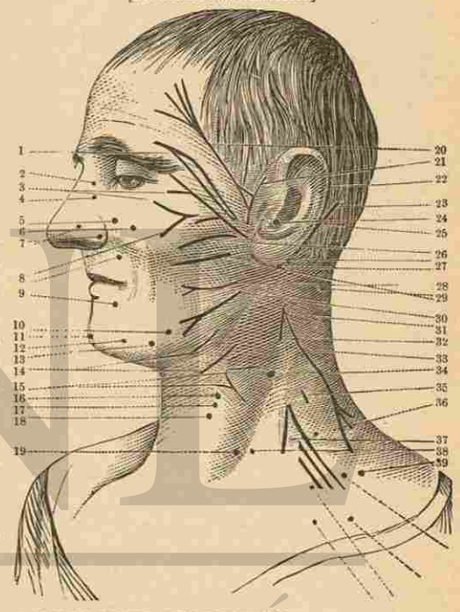

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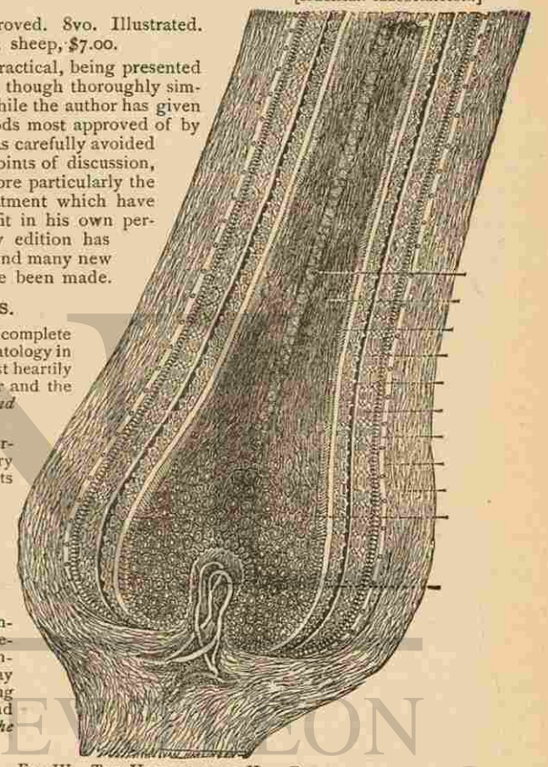

Fig. IV.—The Hair and the Hair-Follicle.

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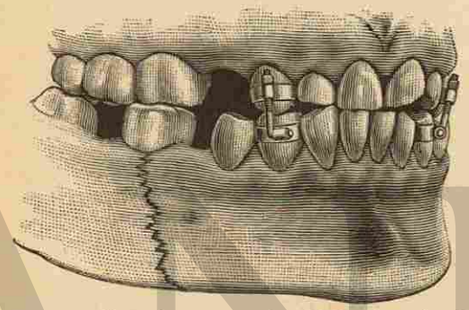

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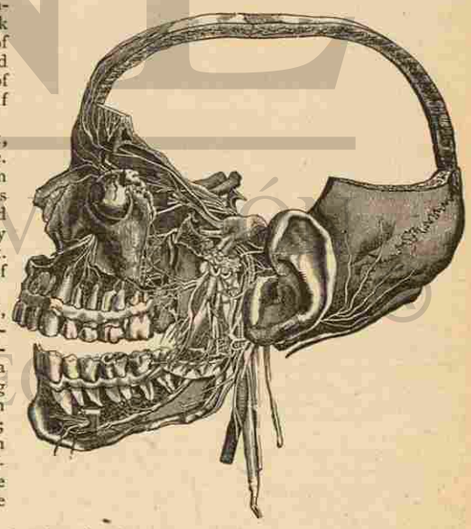

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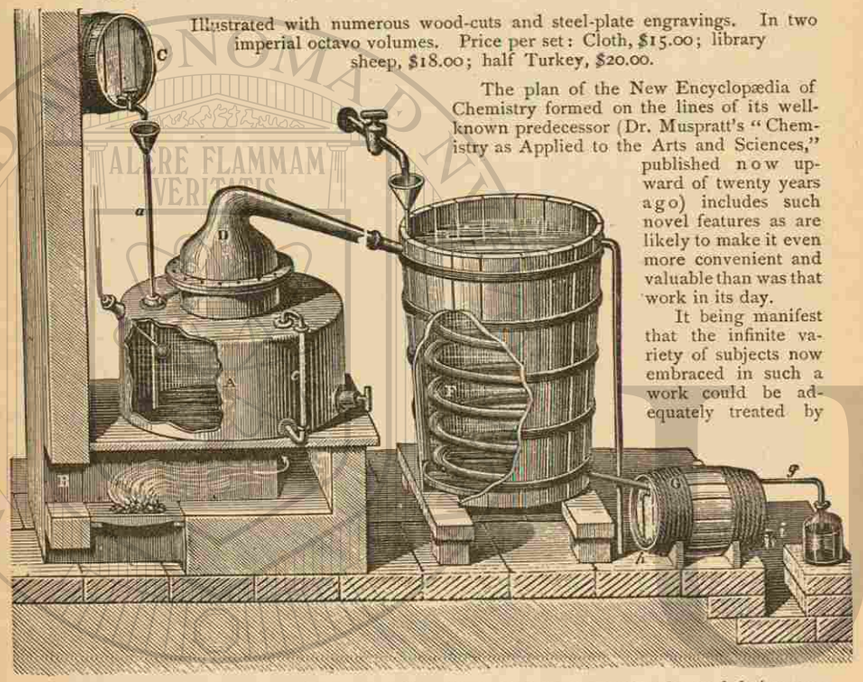

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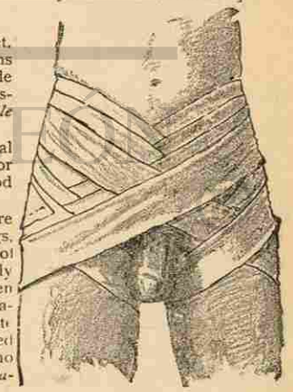

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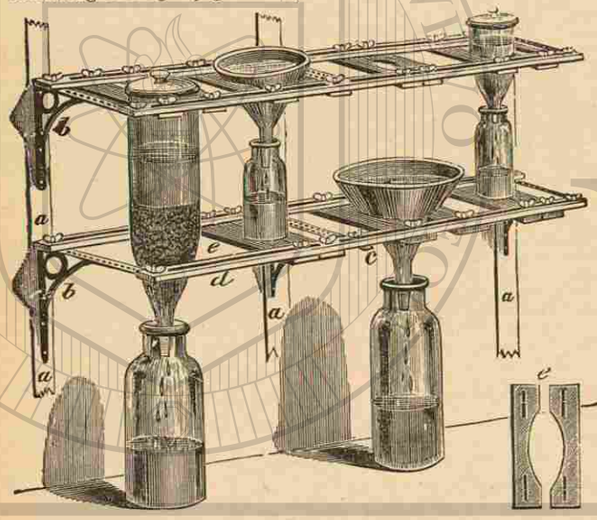

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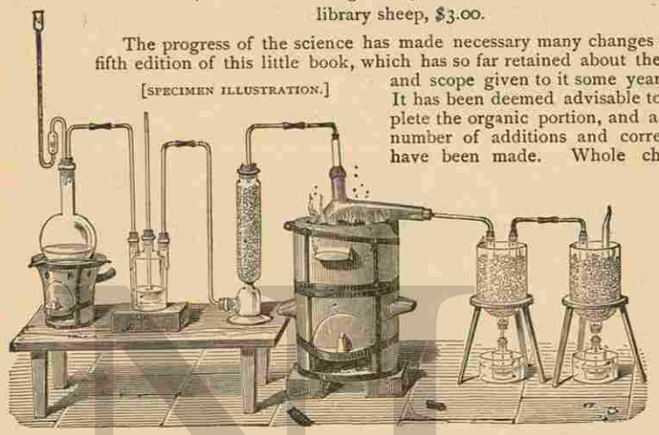

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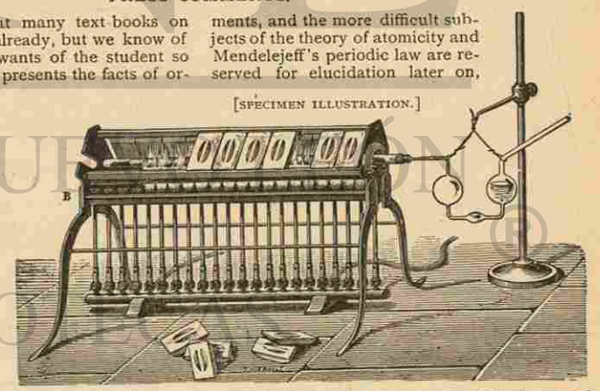

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