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Bristol Medico-Chirurgical Journal, Bristol Medical Library, Bristol University.

British Journal of Children's Diseases (care of Messrs. Adlard & Son, Bartholomew Close, London. E.C.)

British Journal of Dermatology (care of The Secretary, 11, Harley Street, W.)

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Collected Studies from the Research Laboratory, New York. (The Librarian, Research Laboratory, foot of East Sixteenth Street, New York, U.S.A.)

Dublin Journal of Medical Science (care of Messrs. Fannen and Co., Grafton Street, Dublin)

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McGill University, The Librarian, Medical Library, Montreal, Canada

Medicinsk Revue (care of H. G. Dechloff, Leper Hospital, Pleiestiftelsen, Bergen).

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Reports of the Wellcome Research Laboratories (care of The Director, Khartoum, Egypt)

Revue de Médecine (Monsieur le Docteur Lepine, 30, Place Bellecour, Lyons)

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St. Bartholomew's Hospital Reports (care of Librarian, St. Bartholomew's Hospital, E.C.)

St. Luke's Hospital Medical and Surgical Reports (care of F. B. Howard, New York.

St. Thomas's Hospital Reports

Sanitary Commissioner with the Government of India.

Studies from the Pathological Laboratory, Exchange Department, University of California Library, Berkeley, Cal., U.S.A.

Studies from the Physiological Laboratory, Exchange Department, University of California Library, Berkeley, Cal., U.S.A.

The Carnegie Foundation for the Advancement of Teaching. (The Secretary, 576, Fifth Avenue, New York, U.S.A.)

The Medical Chronicle, Owens College, Manchester

The Medical Review, 66, Finsbury Pavement, E.C.

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Transactions of the Medical Society of London, 11, Chandos Street, Cavendish Square, W.

Transactions of the Royal Society of Medicine, 1, Wimpole Street, W.

Bulletins et Memoires de la Société Medicale des Hopitaux de Paris.

Upsala Läkareförenings Förhandlingar (per Prof. Hedenius, Bibliothèque de la Société des Médecins, Upsala, Suède)

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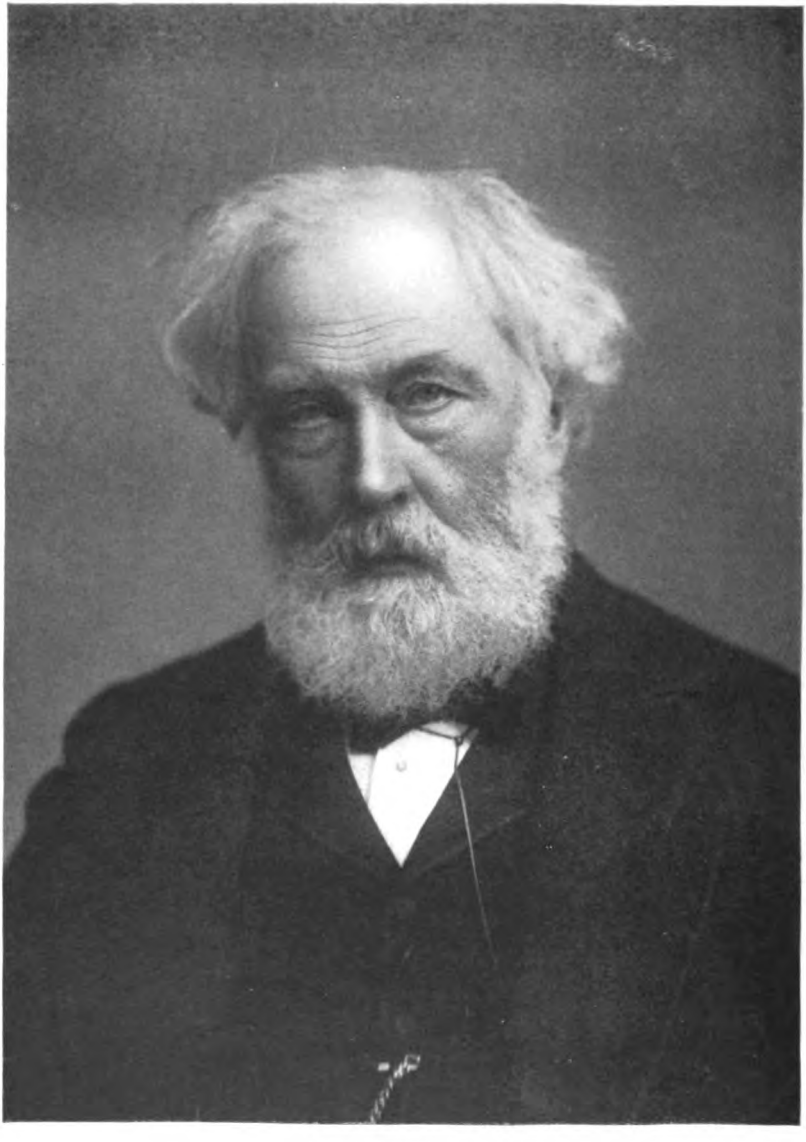


Samuel Wilks

Born 1824. *Died 1911.*

In Memoriam.

SAMUEL WILKS.

By

W. HALE WHITE.

Samuel Wilks was born on June 2nd, 1824, at Camberwell. He was the second son of Joseph Barber Wilks, cashier at the East India House, Leadenhall Street. Many of his ancestors had been in the East India House before him. Wilks himself was one of a large family. He had a brother who lived at Hampstead where he died shortly before Wilks. A sister married Sir Joshua Fitch, the educationalist; another sister, who survived him, became Mrs. Allport. An artist brother died young of consumption.

Wilks's education began in a dame's school, from which he went to a boys' school at Camberwell Green. In 1835, when he was eleven years old, he was sent to be taught by the Rev. Dr. Spyers at Wallop, between Andover and Salisbury. In 1836 Dr. Spyers was made head master of Aldenham. Wilks went with him and stayed at this school for three years. In 1839 he went for one year to University College School, and in 1840 he was apprenticed to the family doctor at Newington, Mr. Richard Prior, for it had been determined to make a general practitioner of him. He himself tells how "A sum of money was paid as usual, and soon I was able to make up medicines and pills for private patients in the surgery, also to vaccinate, bleed, and draw teeth with a very primitive instrument called 'a key.'

In the second year I was allowed to attend a course of anatomy given by Mr. Bransby Cooper at Guy's Hospital, which was only about a mile distant, and in the following year I attended the usual elementary courses, each course being separately paid for, the money being taken by Mr. Stocker, the apothecary. I afterwards had all my schedules properly filled up so as to pass the College of Surgeons and Apothecaries' Hall, as these diplomas were usually taken, although the one for the Apothecaries' Hall was the only one legally required. After a time I thought I would rather qualify for a physician and so took a medical degree. As the only one available was that of the London University, I found it was necessary to matriculate, and this threw me back, although my education had done me a good turn in enabling me easily to get up the Classics required, which I remember was one book of Homer's 'Iliad' and Sallust's 'Jugurthine War.'

"In 1847 I passed the College of Surgeons, the same year dressing for Mr. Aston Key. In 1848 I took the M.B. and in 1850 the M.D. [he took the Gold Medal]. In December, 1847, my master, Mr. Prior, died of pneumonia accompanying the epidemic of influenza then prevailing, so I endeavoured to keep the practice together by seeing all the patients and then to obtain as much as I could for it. Then I sold the horse, which had done me a good turn in going miles round the neighbourhood, and a man soon came who had heard of my wish, and I well remember his demeanour. He first felt the horse all down his legs, and then waived his cap in front of its eyes; after a few moments' thought he said, 'Will you take nineteen guineas for your lame and blind old 'oss?' This I readily did. I took his money and he walked off with the horse."

When Sir Samuel was a student Guy's Hospital consisted only of the present surgical building with the buildings either side of the front square and Clinical Wards and Petersham. Hunt's House, the present Out-Patient Department, the Medical School Buildings, the Nurses' Home, the Laundry, the Pathological block, the Dental block, and the College did not exist. Petersham Ward stood where now the Dental Buildings stand;

in place of Hunt's House was a row of small houses. The Treasurer's garden occupied the position of the block of warehouses at the junction of Maze Pond and St. Thomas's Street, which was quiet, for there was a gate at the entrance to it from the Borough High Street. Wilks used to tell that the alteration in hospitals in his life that struck him most was the increase of cleanliness, but even when he was a student, between 1842 and 1850, he was told that the wards were cleaner than they were formerly, and this was certainly true. Not long before his time they were dirty, squalid barracks, and it was Mr. Harrison who began the reformation; his chief reform was substituting iron for wooden bedsteads and so getting rid of bugs. This also lead to the dismissal of the bug-catcher, as his services were no longer needed. This official was paid £40 a year, which is the same salary as that now paid to a full physician.

When Wilks was a student, although things had improved, yet the wards were mean and comfortless, without flowers or easy chairs, but they were kept clean by continual scrubbing, and this was done by nurses who were not skilled and divided their time between nursing and scrubbing. The medical and surgical cases were all together, so that the physician or surgeon had often to go into several wards to see two or three patients, for it was thought that fever patients and those suffering from surgical diseases would be breathing a better atmosphere if neither were concentrated. The diet was poor and monotonous. Patients provided their own tea and were allowed to buy watercress and periwinkles of a man who went the round of the wards every afternoon. Poultices, which were spread upon tow were used in such numbers that the wards smelt strongly of them. There were no bed letters, but the diet and medicines ordered for each patient were written down in a book. The patients in the Clinical Wards were set apart for purposes of teaching, and just before Wilks entered as a student the Clinical Report Society was founded by the students with the object of reporting cases and so studying medicine in a more systematic manner. A secretary was appointed, and forms were

made for which a small subscription was demanded. The reporters met once a week to discuss the cases, and the Society was patronized by some of the Staff. Soon an obligatory system of the same kind took its place. It will give some notion of what medicine was in Wilks's youth to say that it was only at the time of his studentship that the microscope was beginning to be used, auscultation was in its infancy, and the differentiation between fluid in the chest and solid lung was considered very difficult.

While still a student, namely, in 1850, Wilks made his first appearance as an author. In the *Medical Times* of December 7th, 1850, there appears a review by him of a book on homœopathy. He was for the rest of his life interested in the subject, and I have often heard him talk about it. His arguments against homœopathy were not those of the usual kind about infinitesimal doses, for, as he says, the smell of flowers may produce symptoms although exhaled in infinitesimal doses. Wilks considers that the chief arguments against homœopathy are that the system has failed to make headway and, therefore, cannot have a scientific basis. Also that the law of healing "similia similibus curantur" had been before the world for some time and yet had not been accepted by any authoritative medical colleges, which would have been impossible if a new law of nature had really been discovered, and, lastly, the scientific way of treating disease is first to learn anatomy, physiology, and pathology, but the homœopathists take the treatment part first. His father had for a colleague at the East India Office John Stuart Mill, who read Wilks's review, but declined to give any opinion on the subject.

In 1851 Wilks became a member of the Royal College of Physicians; he was then clearly taking an active part in affairs at the hospital, for in a notice of the Physical Society for the years 1850-51, when Bright was Honorary President, we read that Wilks and Calaway were Honorary Secretaries. He wrote many papers for the "Guy's Hospital Reports." His first was a report of the Clinical Society (Guy's Hospital) from April, 1846, to March,

1847, Surgical Division, in the "Guy's Hospital Reports" for 1847, but his first medical publication in the Reports was published in 1853 (vol. VIII., Series II), and is headed, "Half-Yearly Report of all cases admitted into Guy's Hospital from the commencement of April to the end of September, 1853. Medical Report, by Samuel Wilks, M.D., London." To the end of his life he always strove to be accurate; therefore we are not surprised to read that Wilks, then 29 years old, speaking of the tables he had drawn up, said, "These have all been made solely with a view to scientific accuracy . . . A great many cases which the student has marked as cured in his book I have been obliged to return as relieved as being the more accurate and truthful expression." To contrast the work then with that of the present day, it is interesting to notice that this six months' report dealt with 969 medical in-patients, and the death-rate was 13·82 per cent. It extends to 47 pages, for Wilks describes many interesting cases in detail. It is clear from reading them that he was then, as always, particularly interested in diseases of the nervous system. In this same year, 1853, he was made physician to the Surrey Dispensary In 1854 he married Mrs. Prior, the widow of the practitioner with whom he had worked in his pupilage. She was the daughter of Henry Mockett, of Seaford, Sussex. He had no children, but, as many of us remember, Miss Prior, his stepdaughter, to whom he was much attached, kept house for him until her death a few years ago.

In 1856 he was elected to the Fellowship of the Royal College of Physicians, and he became Assistant Physician to Guy's Hospital. The Medical Staff then consisted of Consulting Physician, Richard Bright, M.D., F.R.S. Physicians, Thomas Addison, M.D.; G. H. Barlow, M.D.; H. M. Hughes, M.D.; Owen Rees, M.D., F.R.S. Assistant Physicians, W. W. Gull, M.D.; S. O. Habershon, M.D.; S. Wilks, M.D.

Wilks gave demonstrations on Morbid Anatomy, that is to say, he made post-mortem examinations daily at half-past two; he lectured on Pathology, and he was Curator of the Museum.

It was indeed fortunate for English medicine that he was given these posts, for it was he who was to show what a helpmate morbid anatomy is to the physician and that he could never really understand his clinical medicine unless he was familiar with morbid anatomy. Wilks was the first to make the profession really grasp the fundamental importance of the study of morbid anatomy, and therefore he may with truth be said to have laid the most important of the foundation stones upon which medicine rests. Many post-mortem examinations had, of course, been made before Wilks's time, but they had been made out of curiosity so that the physician or surgeon might find out if his diagnosis was correct. Wilks made them systematically, learning new facts from them, *e.g.*, that syphilis could affect internal organs. He thus raised morbid anatomy to the dignity of an essential branch of the science of medicine, and, further, he showed how invaluable post-mortem examinations are for teaching both students and staff.

The publication of his "Lectures on Pathological Anatomy delivered at Guy's Hospital during the Summer Sessions 1857—1858," in 1859 told the world the importance of this branch of study, and for the next twenty-five years almost all the energy of discovery in medicine was expended, and most fruitfully expended, in the study of morbid anatomy. This we owe to Wilks and to the labours of the Pathological Society of which he was an active member and ultimately president. In the preparation of a second edition published in 1875 he had the help of Walter Moxon, and in the preparation of the third edition, which he thoroughly revised and brought up to date, he was helped by Sir Cooper Perry. This was published in 1889. It is a wonderful book; over and over again it has happened that a morbid anatomist has thought he has discovered some new fact, but on reference to this book he finds that Wilks had observed it before him. The book is one of the classics of medicine, for Wilks had a genius for observation and he observed accurately. Naturally, therefore, he was a great admirer of Darwin, and ever since the writer knew him a portrait of Darwin hung in his study.

It is impossible to enumerate here all the observations he made in the dead-house, but undoubtedly by far the most important was that syphilis could affect internal viscera. This discovery was announced in 1863 in the "Guy's Hospital Reports," in a paper entitled "On the Syphilitic Affections of Internal Organs." I have already told how before Wilks's time post-mortem examinations were only made to see if the diagnosis was correct, so that when there was little doubt on this score no post-mortem examination was held. It happened that a man in a surgical ward died of a long illness due to syphilis; a large part of the cranium was carious, the scalp had partly sloughed away, the hair was destroyed, and there was a large cicatrix on the head. There were also other bones involved. In this case no post-mortem was wished for as the cause of death was evident, but Wilks says, "on my part the wish was quite the contrary, so the body was opened. I need hardly say that the interior of the body was a perfect revelation to me. Amongst other notable things was a gummatous mass in the liver, which I described as a fibrous material with some rays of the same substance around it. This I showed at the Pathological Society, and a drawing of it is in the 'Transactions.' This was the first specimen of the kind exhibited in London, and was received with considerable incredulity as to its nature by many of the members." This case was observed by Wilks some years before 1863, but it stimulated him to study visceral syphilis. At first his teaching that internal organs could be affected by syphilis was doubted, but the proposition was accepted in a few years, and Wilks's work on this subject led to his being elected a Fellow of the Royal Society in 1870.

When we think of the many effects syphilis is now known to produce, especially in the vessels and central nervous system, the immense value of Wilks's pioneer work is evident. He relates some interesting clinical applications of his discovery, for example, a young man had been discharged from the Post Office in Cape Colony on account of what was considered incurable disease of the liver. He had had syphilis, and Wilks came to the

conclusion that the lumps that could be felt on his liver were gummata; accordingly he prescribed iodide of potassium, and the lumps disappeared. As Wilks says, "he had been cured of his so-called cancer and again set up in life for the sum of two guineas, showing that the pecuniary rewards of the profession have nothing to do with the doctor's skill."

In two other papers in the "Guy's Hospital Reports" Wilks dealt with the subject of lardaceous disease. He showed for the first time that the spleen and kidney may be affected by it, and also that it was due either to suppuration or syphilis. His special interest in this last disease led to his being appointed a Member of the Commission on Venereal Diseases which was held at the Admiralty in 1864. The result of this was that it was made obligatory on all public women living in garrison towns to be examined once a fortnight. This measure caused such an outcry that in 1870 a Royal Commission was held on the subject and the examination of these women was abolished.

Wilks was always very proud of Guy's and very loyal to his predecessors, for by his writings he did much to call attention to their original work and to obtain for them their due recognition. For example, he made and published observations on disease of the suprarenal organs with the view of calling attention to the importance and value of Addison's discovery of the disease associated with his name; he also drew attention to his description of pernicious anæmia as a distinct disease, and to the fact that he (Addison) in 1836 gave as accurate and complete an account of the disease, which has at various times been known as caecitis, typhlitis, perityphlitis, or appendicitis, as has ever been written. Unfortunately even Wilks could not drive this properly into the minds of all doctors, many of whom believe that what we now call appendicitis is a discovery of the last few decades. He discovered Hodgkin's disease, but after doing so found that Hodgkin had many years before described some cases in the Medico-Chirurgical Transactions. I always have felt that Wilks's chivalry made him in this matter very generous to Hodgkin, for I think anyone reading Hodgkin's original paper

will doubt if Hodgkin really knew that he had discovered a new disease. Be that as it may, it was entirely owing to Wilks that the malady became known as Hodgkin's Disease. Wilks also wrote about Bright's disease, and this, too, served to direct attention to the greatness of Bright's discovery. He gained a prize at the Physical Society in 1845 for an essay on Bright's Disease. This was expanded and published in the "Guy's Hospital Reports" for 1853. It was very pleasant for him to read some years after in the article on Bright's Disease in Ziemssens Cyclopædia of Medicine, written by Bartels, "Samuel Wilks ("Guy's Hospital Reports," 1853) was the first to prove, and he did so in the clearest possible manner from the ample clinical and pathological materials at his command, that the condition of the kidney of which we are now talking, and which he described as 'the large white kidney,' ought not to be regarded as the precursory stage of that atrophic process which the German pathologist has proclaimed as the ultimate stage of every diffuse inflammation of the kidneys. That is the third stage of Morbus Brightii. In Germany the work of Samuel Wilks seems to have received little or no notice or recognition. At all events it exercised no influence upon the descriptions of the diffuse renal disease that appeared in our handbooks of clinical Medicine."

The work to which I have up to now drawn attention was all done before 1866, and if Wilks had done nothing else he would have made himself famous, and, indeed, his life in this year was nearly cut short by an accident, as the following quotation from the *British Medical Journal* for August 25th, 1866, will show.— "NARROW ESCAPE.—On Tuesday week Dr. S. Wilks, who is staying at Scarborough, started alone along the sands to Filey. After walking about five miles, he found the projecting rocks hindered further progress. He attempted to retrace his steps, but the tide having risen considerably, he was soon hemmed in. He sought refuge in the cliffs. When the tide had receded it had become dark, so that he durst not come down, but was obliged to remain the whole night, and unfortunately

when daylight appeared the tide had again returned, and he was compelled to wait for hours. On Wednesday morning Mrs. Wilks and her son proceeded to Filey, and having no tidings of him, engaged the services of some fishermen, who, with the assistance of ropes, went over the cliffs and explored the rocks. After searching in vain for some time they found that Dr. Wilks, exhausted, had crawled over the rocks to the door of a cottage on the beach. The poor woman at once admitted him and administered a little brandy to him. His clothes, which were saturated to the neck, were immediately taken off, and he was put to bed, and messengers were afterwards despatched to Scarborough for a carriage, by which he was taken home to his hotel."

In 1867 he was elected full physician to Guy's Hospital, and when he took his share in the lectures on medicine he lectured on specific fevers, diseases of the chest, and diseases of the nervous system. The lectures on specific fevers and diseases of the chest were published in the *Guy's Hospital Gazette* for the years 1873 and 1874. They were so much appreciated by the students that they were reprinted in book form and sold by Mr. Williams at the Counting House. I have my copy by me as I write. I bought it as soon as I became a medical ward clerk, and it is well marked and thumbed, for I read it very carefully. The lectures are a plain straightforward statement of the facts then known without any padding, and as such were much appreciated by the students. Wilks, like most people educated at Guy's, believed that medicine should be learnt in the wards and that students should not place great reliance on lectures as a means of learning their profession. He begins by saying that "Lectures cannot afford you directly the knowledge which you require, but are only the instruments to be made use of in acquiring it." "What advantage is it to you to know the symptoms of pneumonia unless you are able to recognise these symptoms in a patient and draw from them the right inference as to the existence of the disease?"

Lecturing on Diseases of the Nervous System stimulated Wilks's interest in the subject. He had previously published a few papers on nervous disease, and after he lectured on these diseases, his writings were chiefly about them, and, in addition to strictly medical articles, we find him writing on temperaments: the Study of the Human Mind from a Physiological View; Notes from the History of my Parrot in reference to the nature of Language; On the Pupil in Emotional States; The Psychical Research Society; Sleep Walking and Hypnotism; and Ambidextry; indeed, after about 1870 his chief interest seemed to lie in the study of the disorders of the nervous system. His most original paper was one in the *Lancet* in 1872 on Alcoholic Paraplegia. Although he was wrong in attributing this disorder, which we now know as alcoholic peripheral neuritis, to changes in the spinal cord, to him is due the credit of discovering that there is a form of paraplegia due to alcohol. He recognised that it was commoner in women than men, and most of his patients were the wives of publicans. His lectures on Diseases of the Nervous System, published in 1878, second edition 1883, was very popular, not only because there was then no adequate work dealing only with diseases of the nervous system, but because of its intrinsic excellence. It is charming reading. It is about as far removed from a modern text-book on nervous diseases as it can well be, but it is far more interesting, although there is no long statement of the precise course of numerous strands of nerve fibres. Its interest lies in the descriptions of the disease, which are first-hand, and are not copied from books, and in its many illustrative cases, and in the fact that symptoms are mentioned as having occurred in particular patients. So many modern writers fail to make their books alive because they will not insert remarks about actual patients. Then, too, in Wilks's book there are illustrations from general literature that fix for ever the subject on the reader's mind. He is talking of sun-stroke and says, "There has been a very ancient and general belief in this noxious influence of heat in the case of children although the subject never seems to have been scientifically

studied I have always been impressed with this since my earliest childhood on account of a little brother of mine dying of head disease, the first symptom of which was severe headache after playing in the garden on a summer's day and from the distinct remembrance I have of my father likening the case to that of the Shunammite's son of whom we read that 'When the child was grown, it fell on a day that he went out to his father to the reapers. And he said unto his father, My head, my head. And he said to a lad, Carry him to his mother. And when he had taken him and brought him to his mother, he sat upon her knees till noon and then died.'" No book can be dull that thus introduces the most perfect story ever told.

Although now interested chiefly in diseases of the nervous system, Wilks never for a moment lost his interest in other medical subjects. In 1870 he published in the "Guy's Hospital Reports" an article on Arterial Pyæmia which we now call by the much less sensible name of malignant endocarditis. He was one of the first to make the existence of this disease known. Other examples of his power of original observation are his papers on the lines on the nails that follow severe illness, on linæ atrophicæ, and on verrucæ necrogenicæ.

During the time of which we have been chiefly writing, Wilks, like so many Guy's physicians, lived in St. Thomas's Street, but about 1869 he moved to 72, and shortly afterwards to 74, Grosvenor Street, where he remained until he retired from practice in 1901. He had been made full physician to the hospital in 1867. Seventy-four Grosvenor Street was a house well known to many of us. Perhaps the best remembered thing in it was the parrot who always lived in a front room immediately on the right as you went in. Behind this was Wilks's rather dark consulting room, lined with books, and beyond this again was a large dining-room. Upstairs there was a small room behind the drawing-room, and in this Wilks loved to gather a few friends for a talk and a smoke on Sunday evenings. In October, 1879, he was appointed Physician to the Duke and Duchess of Connaught.

Wilks was always to the front when there was any question of investigation about anything, especially anything related to medicine; therefore, we find he was one of those who helped to form the Association for the Advancement of Medicine by Research. This Society was founded on March 28th, 1882, at a meeting held at the Royal College of Physicians. The chief events that led to its formation were the passing of the present Vivisection Act (1876-77), the prosecution of Dr. Ferrier and and Dr. Gerald Yeo (1881), and a resolution passed at the International Medical Congress held in London in August, 1881. Preliminary meetings were held in Sir Andrew Clark's and Sir Risdon Bennett's houses. Sir James Paget was appointed Chairman, Sir Samuel Wilks Honorary Treasurer, and Dr. Pye-Smith Honorary Secretary. Sir William Jenner was President.

All the members of the Staff at Guy's Hospital are compelled to retire at the age of sixty. Wilks attained this in 1884, but he was specially requested by the Governors to retain his post for an extra year, for, owing to the death of Dr. Fagge in 1883 and Dr. Mahomed in 1884, there had been two vacancies recently, and the demonstrators and registrars from whom members of the Staff are usually chosen were young. He accordingly did not resign till 1885 when he was 61 years old. During the eighteen years of his full physiciancy he was in the front of all that was going on in the medical world. Thus, he delivered the Harveian Oration before the Royal College of Physicians in 1879. It was a rare honour for anyone to be called upon to do this at the age of fifty-five, and he delivered an address to the Pathological Section of the International Medical Congress held in London in 1881. He delivered the address in Medicine at the Annual Meeting of the British Medical Association held in Birmingham in 1872. He was President of the Section of Medicine at the meeting held in Cardiff in 1885; later on, in 1895, he was President of the Section of Pathology and Bacteriology at the meeting held in London. He was also President of the Pathological Society from 1881 to 1883; he was an original member of the Clinical Society and of

the Neurological Society, of which he was President in 1887, and his interest in diseases of the nervous system is shown by the fact that he was one of the original subscribers to "Brain." In 1884 the University of Edinburgh conferred upon him the honorary degree of LL.D. In 1885 he was made a member of the Senate of the University of London, and two years later he became a member of the General Medical Council. His holding of these offices was characteristic of his loyalty to the profession, for he hated meetings that had not to do with medicine directly, yet he was always willing to do the hard work in connection with the administrative side of professional work that has to be done by somebody.

During these eighteen years, too, he worked hard at Guy's Hospital, lecturing, visiting the wards, and doing all the routine work of a busy hospital physician. He was always much attached to the Physical Society, and was its Honorary President until his death, and for many years presided at the October meeting. He was last present at the meeting held in October, 1910, and here it may be added that for many years he was editor of the "Guy's Hospital Reports."

When in 1885 he retired from the active staff of Guy's he was made Consulting Physician; he used to say that the curious feature of his office was that he was never consulted, but he was often to be seen about the hospital frequenting especially the part in which he had worked earliest, namely, the post-mortem room. He was elected a Governor of the hospital. As far as I know the only other member of the Staff who has been so honoured during the last fifty years is Sir William Gull.

Wilks's career may be easily divided: the first period ended in 1866 when he became full physician at the age of 42. During this he had made himself famous by his medical writings, the most original of which announced the discovery of visceral syphilis. The second period extended from 1866 to 1885 when, at the age of 61, he retired from the post of full physician. His chief work during this period was in connection with diseases of the nervous system, and we now come to the third period from

1885 to 1901, when he retired from practice at the age of seventy-seven.

During this period of sixteen years he was, as usual, active; indeed, he never could be anything else; he was always talking, reading, or writing all through his life, but the chief event was that in 1896 he was elected President of the Royal College of Physicians. This he used to say was the greatest honour that had ever fallen to his lot. In 1893 Sir Russell Reynolds had been elected, defeating Wilks by three votes only among considerably over one hundred voters. Sir Russell announced in 1896 that he did not desire re-election, and many felt that Wilks ought to be elected, for he was by far the most eminent representative then alive of British Scientific Medicine. But the difficulty was to persuade him to stand. One of his colleagues called on him at four o'clock one Sunday afternoon, and argued and talked with him till ten o'clock the same evening, when Wilks cut short the discussion by saying he was going to bed. But he had not consented. This same colleague called again at five the next day. Wilks had told his servant to say not at home, but happily the servant forgot, and the argument was renewed. After about half an hour's conversation he yielded his consent, and was elected by a large majority. One of the President's duties is to make many after-dinner official speeches. Wilks was never tired of saying on these occasions that he was not fitted to be President, that such an office was not in his line, but that he had been driven into it against his better judgment by the excessive importunity and unreasonable pertinacity of one of his colleagues.

He was right in his own estimate of himself: he was not a remarkably good President, for he was not sufficiently business-like, but most certainly he was not a bad President, and the large majority of Fellows that made him President felt that by being President he honoured the College, which naturally wished that the most intellectual physician of the day should be at its head. Everyone admitted that Wilks held this position, and his portrait appeared in *Vanity Fair* over the title of "Philo-

sophical Pathology." He himself always regarded Sir Andrew Clark as the best President he had ever known; he used to say, "Before Clark became President if you asked a cabman to drive you to the College of Physicians he always said, 'Where is it?' but after Clark had been President a short time every cabman knew where it was."

In 1897 he was created a Baronet, and was made Physician-Extraordinary to Queen Victoria, and it was he who proposed the vote of thanks to the Prince of Wales, afterwards King Edward VII., for presiding at the Banquet, in aid of Guy's Hospital, at the Imperial Institute on Wednesday, June 10th, 1896. He made an admirable speech full of his well-known affection for Guy's. In 1897 he was entertained at dinner by his friends in order that they might congratulate him on his Baronetcy, and this dinner gave him more pleasure than anything else which took place in his life, for the numbers present were so great that the dinner had to be given in the Whitehall Rooms. As Mr. Cosmo Bonsor, the chairman, said, men came from far and wide to pay compliment to their most worthy and distinguished guest. Over forty letters of regret and apology for absence were received. The toast of proposing Sir Samuel's health was put in the hands of Dr. Pye-Smith, who truly said of him that, "all who knew him knew that he had never sought for fame, it had come to him unasked; he had never courted popularity, yet no one in his profession was more popular. He had made Solomon's choice of wisdom and long life; riches and honour had been added." Those who remember Wilks's speech in reply will recollect how admirable it was with its self depreciation, sadness, pride in being so beloved by Guy's men, and its quiet humour. He said there had only been three Samuels of any pretence—first, the Prophet; second, Dr. Johnson; and third, Mr. Weller, Junior.

Perhaps the love for him is best shown by the concluding paragraph in the *Gazette* about the dinner. "After Mr. Bonsor had responded, the majority of those present, many of

whom had to make long journeys that night, left the hall." After the dinner Wilks wrote to the *Guy's Hospital Gazette* a letter, published on page 500 (1897), in which he thanks those who came, and concludes thus, "Coming, as these gentlemen did, from all parts of England and Wales, and leaving their families and practices, they must have gratified their wish to greet me at great inconvenience to themselves. I regret that I could not shake hands with them all. Such a practical expression of friendship from nearly three hundred persons has deeply touched me, and is, indeed, a fact that I am scarcely yet able to realize. To all my friends then present I desire to express my most sincere thanks for this striking demonstration of devotion, friendship, and affection, and to assure them that they have made this occasion the most memorable day of my life." Old Guy's men as far off as Australia did not forget him, for he received an Illuminated Testimonial from all the old Guy's men—twenty in number—practising in New South Wales.

In 1892 there appeared a book that has given Guy's men so much pleasure, namely, the "Biographical History of Guy's Hospital, by Wilks and Bettany." He wrote the biographies of those of the Staff who had been personally known to him, but who had passed away at the time the book was written. Most readers of the "Reports" have read this book, some, like myself, have read and enjoyed it frequently, hence there is no need here to refer to it, except to remark that it is an illustration of the love of Wilks for Guy's, for it must have been a considerable labour for a busy man nearly seventy years old. He was an excellent biographer; you feel you know those of whom he writes; good points and less good points are all included.

The fourth and last period of his life dates from his retirement from practice in 1901 to his death. During the whole of this time he lived at 8, Prince Arthur's Road, Hampstead. For a large part of this time his step-grandson lived with him, but Wilks saw little of him for he was out at his work all day. But Wilks filled up his time with his passion for observation. A small sundial was put up, and at his window was a thermo-

meter arranged so that the outside temperature could be seen without opening the window. His favourite pictures and books went with him, as well as some of his furniture, so that the Hampstead house looked like that in Grosvenor Street on a small scale, the chief difference being that as it got plenty of sun it was more cheerful. He soon joined and quickly became President of the Hampstead Scientific Society. He was a most diligent attendant at its meetings, and it was greatly owing to his initiative that the observatory in connection with the Society was built. He continued his interest in all sorts of subjects; thus we find him in the last decade of his life writing on Keats, the orbit of the moon, vaccination in Italy, Farenheit's thermometer, the cared elm of Hampstead, death from lightning, hermaphroditism, and many other subjects.

He had always been fond of and a good judge of English literature; he had an excellent memory and was, therefore, able to light up his talk with appropriate allusions. He had a personal affection for many authors, and the photographs on the bedroom mantelpiece during his last illness were those of Wendell Holmes, Wordsworth, and Harriett Martineau. The activity of his mind was extraordinary, indeed, more extraordinary than I have ever known in anyone of his age, but I need produce no more evidence of this for it is well shown in the account of his life in Hampstead kindly given by Dr. Jessop and printed at the end of this article.

He made many friends at Hampstead; the inhabitants were proud of him, and his beautiful face and fine white hair were a familiar sight in the streets or on the Heath until he became too feeble to walk. The affectionate companionship of his neighbours, Mrs. and Miss Martelli next door on the one side of him, and Dr. and Mrs. Bashford on the other, helped to render life pleasant for him. His illnesses were rendered less irksome by the kindness of Dr. Jessop. His old friends in London used, too, to go and see him, and he was always very glad to see them. Until just before the end he talked as well as he ever did. He usually monopolised the conversation, indeed, some-

times there could hardly be said to be any conversation, but that was the better, for Wilks was a great talker. I remember calling on him one Sunday afternoon; although over eighty he was reading the *Lancet* without glasses and was smoking a cigar. After about half an hour, during which time the cigar had often gone out and been relit during his talking, coffee came in with muffins and crumpets. Wilks went on talking, and finally, when the muffins and crumpets were all cold, he ate them and drank the cold coffee, still smoking the cigar which by this time was in a deplorable condition. The time came for me to go, and as I went out at the door after saying good-bye, Wilks said, "You have told me no news, but I suppose you will say I have not given you a chance," which was perfectly true. Nevertheless, I had thoroughly enjoyed myself. If the callers were medical the talk was chiefly medical; especially he loved to talk about Guy's, for he had a greater veneration for Guy's Hospital and School than anyone I have known. It was very fine to notice his pride in his own school, and it inspired those who heard him talk to try and work for the School as Wilks himself had worked for it. If his visitors were not of his profession he always found something which interested them and about which he could talk; thus, I remember that at one time he was fond of talking about the burrowing bees on Hampstead Heath which he had been watching. At another time the conversation would be about the supposed unluckiness of the month of May for marriage.

His marvellous vitality was not limited to his mind, his body shared it, for during his life at Hampstead he had a severe attack of appendicitis, accompanied by a large abscess; this was operated upon. Soon after he suffered from an enlarged prostate; this was removed. In both instances his friend Mr. Symonds performed the operation. He also had a curious cerebral attack accompanied by unconsciousness which lasted two or three days, and finally, more than a year later, he had paraplegia which for months confined him to bed and prevented his moving his lower limbs, and led to his losing control of his

bladder. His spirits never deserted him; for example, on one occasion, not very long before his death, he sent for me because he did not feel at all well. He said, "Do you know why I sent for you? Because I knew you would not give me any medicine."

Two years before his death he began to write a book, that is to say, when he was eighty-five years old. His friend Sir Bryan Donkin helped him to see it through the press, and it was published a few months before his death. On the title-page he calls it "A Memoir," on the cover of the book it is called "Biographical Reminiscences of Sir Samuel Wilks." It can hardly be described, for there are few if any books with which it can be compared. It consists chiefly of an account of the history of medicine during Wilks's life as far as 1884 as seen in the articles published in the "Guy's Hospital Reports," but there are many other facts of interest, and Wilks gives a very short biography of himself. Was there ever another instance of a man beginning a book at the age of 85 and having determination enough to correct the proofs at the age of 87 as he lay in bed completely paraplegic.

He died on Wednesday, November 8th, 1911, and was cremated on November 11th. The respect in which he was held is shown by those who were present at his funeral. A list will be found in the *British Medical Journal* for November 18th, 1911.

In the book just mentioned Wilks says, "I quite discard the well-known dictum, *de mortuis nil nisi bonum*, which makes many biographies very pleasant reading to the friends, but to those who call themselves 'outsiders' must often, on perusal, be very like the biographies of medical men which we read in the weekly journals, when one feels like Goldsmith's 'Citizen of the World,' who declared how delightful it was to read the epitaphs in the English churchyard and see how those who had departed this world excelled in every private and public virtue." Nevertheless, I feel that it is impossible for me to convey a correct impression of Wilks, for he did excel in every private and public virtue, and, therefore, probably those who did not know him may think that what I say about him is like lapidary epitaphs, whilst in reality it falls short of the truth.

Wilks was more beloved by the profession than anyone I have ever met. As he himself said, to be honoured by one's own profession is the highest good fortune that can be attained by any man. What the public think of any doctor is of little matter. The affection of the profession was shown in many ways. I have already mentioned the enthusiasm with which he was greeted by the many who came to the dinner given to him when he received a Baronetcy; whenever he made a speech or presided at the Physical Society the reception given to him showed the depth of the affection for him. He himself used to call himself the doctors' doctor, for he was always being consulted by doctors about themselves, their wives, or their children, a sure sign of their esteem for him and their confidence in him. Perhaps nothing could better show this affection for him than the following skit by W. L. Braddon in October, 1885, the year of his retirement from the Active Staff of the Hospital. It is printed in the *Guy's Hospital Gazette*, November 18th, 1905. He was always affectionately known as "Sammy."

Of all the boys that are so smart,
  There's none so smart as Sammy;
He is the darling of our Art,
  And Guy's his Alma Mammy.
No lady has a kinder hand,
  Or's gentler-like than Sammy;
His name is known from this here land
  To Seringapatamy!

Of all the days that's in the week,
  We dearly love this one day—
The day on which he comes to speak,
  And that is "Take-the-Bun" day!
Then, 'tis confessed, we're at our best,
  And all to talk with Sammy,—
He is the darling of our Art,
  And Guy's his Alma Mammy.

The hospital, and patients all,
They love the name of Sammy;
And who, when he's Examiner,
But would not be Examinee?
But now his long, long years are out
At Guy's, his Alma Mammy,
We shall regret, but ne'er forget
Our grand, and dear old Sammy!

Those who had been educated at Guy's were very proud of Wilks's scientific achievements, but it was not pride in them that led to deep affection, it was, I think, firstly, his honesty; he did his work, said what he thought, never courted popularity, never played to the gallery, never said or wrote anything which could possibly be thought to have for its object, however remote, an increase of his practice; indeed, he always maintained that medicine was a poor trade, but a glorious profession. Secondly, there was his detestation of all humbug; such detestation was usually expressed in scathing words. Thirdly, there was his intense loyalty to his profession and to Guy's; the first was shown by his ardent defence of vivisection and vaccination and of the encouragement he gave to all young men who wished to advance the knowledge of medicine, such left him determined to do all they could for the cause. The second, by his whole life; his affection for his hospital did not cease with his retirement from it, for he paid frequent visits to the hospital after he had left the Active Staff. Lastly, there was his beautiful and kindly face. He was so often photographed and painted that he used jokingly to call himself the professional beauty. Ever since I knew him he had silver white shaggy hair, and looked exactly the part of a grand old man.

As you got to know him more your veneration increased, for you came to see his intense affection—when he was well over 70 he slept for several nights in his day clothes on a sofa in his step-daughter's room when she was ill in case she should want anything in the night—and his courage. He once said, "There

was the inner life of a man, too, known only to himself—the family and pecuniary troubles, the physical difficulties, headaches, sleepless nights, and the like—he had had them all." He had more than his share, and the heroic courage with which he drowned them without a murmur of complaint by throwing himself into his work was astounding; it carried him through life to the end—was he not correcting proofs as he lay in bed completely paraplegic. He never spoke of his troubles or his own generous actions; those who knew him most intimately only discovered them by accident.

I have tried to show Wilks as a scientific man and investigator and to indicate his character. It only remains, therefore, to speak of him as a teacher and physician. He could, when he liked, place the ordinary facts of the common diseases before students in a systematic manner, and in a way that appealed to them; the proof of this is seen by the printing in the *Guy's Hospital Gazette* of his lectures on medicine and their subsequent publication by request of the students in book form, but this way of teaching was not congenial to him, and, therefore, in the wards he did not teach the details in that dull way that is sometimes necessary to help the less able students through their examinations; he was never a coach, but he was a great teacher. He did what it is essential for a first-rate teacher to do—he made the taught think for themselves. Some one or two points about the patient's illness would be taken and discussed or rather talked about by Wilks and always illuminated. From this it followed that his teaching, although not always appreciated by junior students, or by those whose heart was not in their work, was much sought by senior men fond of medicine. Many of them went round the wards with him, especially during his Sunday morning visits. These were reserved for senior men; the ward clerks were not expected to attend. At the time when I remember Wilks as a teacher he was at his best when discoursing on some case of nervous disease.

He had such a vast experience both in the post-mortem room and the wards that he arrived at a diagnosis with great rapidity,

and he was rarely wrong. He appeared to take in the case all at once, and he had to a remarkable degree the aptitude for assigning to each part its value. He resented being frequently accused of a scepticism about drugs: the fact was he was not a sceptic about them, no one could be louder in his praise of the proper use of iron, opium, mercury, quinine, and other drugs which are known to have definite actions for good, but what Wilks, with his scientific mind, set his face against was the giving of all sorts of drugs just because we must do something. Nothing could possibly be more unscientific than the old-fashioned prescription of many ingredients—it might do the patient more harm than good for all the prescriber really knew—but it soothed him into a belief that he was doing something for the patient, and thus it turned his attention away from the unfortunate circumstance that in reality he did not know what was wrong with the patient. Wilks simply had greater courage than most men, for when he did not know what was the matter with a patient he said so, and when he did not know what would do good he trusted to the defences of the body to heal the patient instead of giving all sorts of rubbish as many did because they felt they "must do something," as the phrase was. It requires considerable courage to do nothing when you do not know what to do.

Although, as I have already mentioned, Wilks was extremely popular with the profession (no one saw more sick members of it), yet he was never a fashionable physician. This is by no means to his discredit, his mind was so constantly full of the science of medicine that it could not turn easily to the various little details that contribute to the making of a large practice, and, further, if there was nothing much the matter with the patient Wilks could not take great interest in his case; but in a case of real illness he showed all the qualities of a great physician. Soon after I was qualified, a doctor asked me to stay in the house of a patient who was very ill. Wilks was called in; he quickly cleared up the doubt that had existed as to whether the patient was suffering from septicæmia or typhoid

fever, and gave it as his opinion that the man would die, which turned out to be correct. Nothing could have been better than the way in which he told the young wife who was expecting her confinement, encouraging her to bravery for the sake of the child, and helping her by his kindness, which I know she remembered for the rest of her life.

Sir Bryan Donkin, Dr. Edward Jessop, who looked after Wilks most devotedly during the years he lived at Hampstead, and Sir George Savage, were such close friends of his that I asked them to write a few words about him which I could append to my short account of his life, and I am grateful to them for having done so.

Sir Bryan Donkin says: My acquaintance with Sir Samuel Wilks began in 1871 when, as a student, I used to accompany the late Dr. Charlewood Turner on his visits on Sunday mornings to the wards of Guy's where Wilks was in the habit of giving clinical talks to a small number of men, including some who, like myself, came from other medical schools. From that time onwards I have regarded him as the most suggestive and stimulating teacher of the science and practice of medicine that I have known. About the year 1878 I became well acquainted with him personally, and for the last thirty years of his life was proud to know him as a friend.

He was a delightful travelling companion; and, at least to me, a visit to a picture gallery with him was a source of much greater pleasure and profit than similar visits in the company of specialist connoisseurs. His love of science and love of art illuminated and intensified each other. His sense of humour was no small part of his social charm—of this Guy's men need no reminiscences. I was always greatly struck by the enduring freshness and receptivity of his mind; to the last he was keenly interested in all new investigations and modes of thought. He never clung to an opinion because he had once held it strongly nor accepted a new one because it appeared fascinating.

In his later years he studied with much interest the controversies concerning evolution and heredity which have of late been rife among biologists. At that time I was also interested in these subjects and we often talked about them. Wilks agreed with Archdall Reid that neither the "biometrical" nor the Mendelian school was following the scientific method rightly, and said that each of them seemed to have only half lights on the meaning of Darwin's work and thought.

He also at this time talked about "mechanism," and vitalism, and the ultimate questions of metaphysics. He held that science had nothing to do with the question of why the universe came about or what is to become of it or why man was developed. He was, in fact, an agnostic in the full and serious sense of the word in which Huxley used it.

He was modest to a fault and would not have tolerated a Boswell. But, though he lacks a "*vates sacer*," "*illacrimabilis*" he will never be till all who knew him are gone.

Dr. Jessop writes: When Sir Samuel retired from practice and came to live at Hampstead he was not destined to have a peaceful and uneventful old age; on the contrary, the last ten years of his life formed a period of constant struggle between increasing physical infirmities and an unconquerable spirit; although, as always, physical infirmities at last won the battle his spirit remained undaunted to the end. Knowing very few people at first, he soon made a large circle of friends; having the power of attraction, and very wide interests combined with a decided personality, and being of a very sociable nature he quickly took a prominent position in the place. He was made President of the Scientific Society, a large and very much alive body. His versatility was shown by reading papers on various scientific subjects. He was a most regular attendant at the meetings, and always had original observations to make on the subjects discussed. Sir Samuel was indeed a man of very high mental calibre, with a strong scientific bent; no matter how dis-

tinguished an assembly of general scientists he might have been thrown into, his general knowledge of scientific matters was such that he could always have held his own.

In his own special domain of medicine he was, of course, in the very front rank of his time, if, indeed, not in a class by himself. If the excellent bibliography compiled by W. Wale (*Guy's Hospital Gazette*, November 25th, 1911) be referred to it will be apparent at once how wide was the range of subjects he wrote about, and if the papers themselves are read, it will at once be appreciated what an independent and original mind was brought to bear on whatever he wrote about, always interspersed with evidence of literary knowledge of quite a high order.

His chief characteristic was Investigation, his object being to get at the bed-rock of facts, and for this purpose his patience was inexhaustible. He did not possess a large library, but it was surprising the information he got out of what he had; his books were all well worn and accustomed to frequent use. It was with indomitable perseverance that he would pursue a search till he had impaled his fact. This was the same whether the information was to be gained from a book, a museum, or from some person or authority on the subject he was interested in. His last book, Biographical Reminiscences, was in great part written and the proof sheets wholly corrected whilst lying in bed completely paralysed from the waist downwards, with incontinence of urine, and paralysis of the bowel. Never was writing done under more adverse circumstances, but mental occupation was as the breath of life to him; this book finished, he rapidly failed.

His death-bed might form material for a picture, "The Ruling Passion Strong in Death"—propped up as well as his paralysed and attenuated form could be, three good-sized books of reference he had sent for to refer to the appearance of a nutmeg in its original form, lying on his bed, his feeble hands copying a drawing in one of the books. It was under these conditions his last hour of sensibility was spent; he became unconscious with his books around him. It was at best a poor victory for

Death; the mastery was not to be gained until the very seat of life itself in the brain was stricken—only by this means could his indomitable spirit be overcome.

Sir Samuel had an intense love of Nature in whose simplest products he could find interest and beauty. I have seen during illness his room with many beautiful flowers around, but nearest him he would choose to have a common hedgerow bunch, and with a botanical work and lens he would point out little peculiarities of growth, asking for any information you possessed. There would then be a looking up of doubtful items, and so on, *ad infinitum*, as long as it was possible to stay to discuss them.

His last journey to London was undertaken to visit the returned Arctic ship, the *Nimrod*, stationed near the Thames Embankment. His tottering limbs were assisted to let him make a thorough examination—he would be satisfied with nothing less. The proximity of the Cancer Research Laboratory was too attractive for him to miss, so he visited that the same day, showing the keenest interest in every method of research. His mind was too engrossed to observe the physical needs of the body, and it was a very exhausted man that returned home.

Perseverance was a great trait in his character, especially when it was a question of acquiring knowledge, his mind being fixed on some object, no trouble or physical inconvenience was too great to deter him from pursuing it. The same may be said when it was a question of getting his own way, for he always liked to have it, and, when necessary, would display considerable artfulness to obtain it, and if thwarted would show considerable impatience, but later on would always let you understand he recognised he was wrong if this was so. His nature was entirely free from the petty meanness of bearing resentment for even a strong difference of opinion from his own.

From the qualities he possessed it followed that he had a very considerable knowledge of general literature apart from being well abreast of recent science, and perhaps the department of literature he most loved was Poetry. His memory being excellent, he could generally make an apt poetic quotation; also his

lectures, such as the Harveian and the one delivered before the British Medical Association, were liberally interspersed with quotations. He had the greatest love and veneration for Shakspere; he could quote long extracts from the plays and sonnets. He was also very fond of Wordsworth. A poet with such an intense feeling for Nature would be sure to appeal to him as a kindred spirit. It was also very common to see the Bible near him, and consulted, too; he was very fond of and familiar with its contents. I am afraid he would have been a troublesome opponent to a bigoted person with a narrow religious creed. Though Sir Samuel in the ordinary sense of the term could not be called a religious man, he was essentially a good man, his actions all sprang from good motives, he was high principled, and of unswerving honesty of purpose, but the trend of his mind was such that it could hardly accept all the teaching commonly heard in the churches. However, he took great interest in theological questions and would always go to hear any original preacher whether in church or chapel. His writings were marked by a terse, not to say somewhat pugnacious, mode of expression which generally called forth some controversy; in fact, he often plunged into one, and he was absolutely fearless in expressing his opinion whether in speech or writing.

It is not within the scope of this contribution to discuss the impression his writings have made nor the influence his life has had on contemporary medicine, but remembering the extraordinary advances made within the last sixty years, it is remarkable how many of the conclusions he arrived at then are not obsolete now. This is because he wrote from personal experience and knowledge. An eminent authority on cancer, speaking of Sir Samuel, said to me, "Most of his ideas on cancer hold good to the present day, which can hardly be said of any other authority at that time." He was a great practical pathologist and wrote about what he himself saw in the post-mortem room, not about what other people had seen. Undoubtedly the post of pathologist he held at Guy's had a marked influence on his after life, for not only is it the best training a physician can have, but it was

one peculiarly suitable for developing his natural bent for investigation. It was probably due to him that the systematic examination of bodies was undertaken at Guy's, for previous to his time a special petition had to be signed by the medical attendant for a post-mortem, and this was only undertaken to verify or otherwise the diagnosis. If the cause of death was sufficiently evident there was considered to be no necessity for a post-mortem. It was not until an examination was made with the idea of gaining fresh knowledge or elaborating what already existed that the real study of pathology commenced, and it was largely due to Sir Samuel that this systematized investigation was initiated.

Undoubtedly the prominent feature of his life was his connection with Guy's Hospital; his affection and veneration for his old school was part of his being. Whether it would have been so of any other school it is impossible to decide, but he was so essentially a part of Guy's that one could not imagine him belonging to any other. It was as if he was a detached portion of the hospital, so closely was he identified with the place. Personally, whenever I think of Guy's, I always think of Sir Samuel Wilks as emblematic of the Spirit of that Hospital, as the ideal of a Guy's man, and I venture to think that Guy's men themselves would point to him as the ideal representative of the best traditions of their great School.

Sir Samuel lived a long life—87 years. He outlived most of his contemporaries, but he did not outlive his friends, for he had a great power of attraction and of inspiring affection. Whence came this power it is difficult to say. His manner was brusque and his words were not chosen with the idea of propitiation. He could be sarcastic and he could be angry, but he had a lovable nature and was a staunch and loyal friend. To quote his beloved Shakspere, "He was famous, sir, in his profession, and it was his great right to be so."

Sir George Savage writes: When asked to contribute to the biography of Sir Samuel Wilks, I felt, almost for the first time,

a very grave responsibility in relationship to my old friend and teacher; for I find that many tales and their memories have almost faded away. I will, however, recall the past as far as I can.

From 1862 till 1866 I was at Guy's Hospital, and from that time until Dr. Wilks resigned his position at the hospital I was frequently with him. In the earliest days he was Out-Patient Physician, and I remember even then, in the stuffy, crowded Out-Patient Department, his humour and quaint observations on men and women. Once, after ordering some simple tonic for a weakly, anæmic patient, he said he wished there was a machine like a piano on which he could play while seeing his out-patients, a machine which should supply suitable food and stimulant instead of physic. I also recall his smile as a chlorotic girl came into the room, when he said, "These women have an eye for colour: they always have green ribbons."

A small, emaciated boy was brought into the room, and Wilks at once said, "Prematurely aged; he sleeps with his grandmother." As it happened, he did. Wilks spoke of the supposed abstraction of health from the young by the old, and, as was so common with him, took the Old Testament to witness, and the example, King David.

But the most early associations of Wilks are, naturally, in the post-mortem room. He knew the histories of many of the cases which had to be examined, though he said ward-clerks ought to be present and give the physician's or the surgeon's diagnosis before the post-mortem was made. Yet he liked, from the material before him, to draw his own conclusions as to past symptoms. What struck one at once was the unbiassed way in which he looked at everything. He was not wedded to authority, but described what he saw. His pathology was that of the post-mortem room, not that of the book or authorities.

It is not for me here to enlarge on the many points of original observation made by him. He was often quaint in his expression. Thus, I remember him saying it was a pity that pathologists compared pathological material to pleasant foods, such

as damson cheese, prune juice sputum, or nutmeg liver. In the post-mortem room he did not confine himself to a simple description of the part seen, but often wandered far off into causation, symptoms, and treatment. He was, by all his colleagues, recognised as a fellow-worker, and not as a rival. When Hilton saw him in the operating theatre and asked his special mission, he replied that he had come to see what the surgeons were preparing for him; and when I remember the death-roll following operations I understand the remark.

He used, when he lived in St. Thomas's Street, to be not only a daily visitor to the wards, both surgical and medical, but it was his constant custom to come late in the evening, and, after seeing some patients, talk on and on in the corridor, in his peculiar way. Full of knowledge, and ever willing to communicate it, it was not always easy to follow his line of thought, for he often—in fact, generally—wandered in by-paths as he talked. His references to Scripture and to the poets were frequent, and often quaint. He was fond of quoting from the Books of Moses on sanitary and allied subjects.

I never attended any of his lectures on medicine, as he only became full Physician after I had left the hospital. Then there was a gap of several years when I was away from London. Yet, even then, I met him when I was in London. And I may make a confession. When I went up for my M.D. London Examination, Wilks was an examiner in medicine. I came up a few days before the examination and went round the wards with him, and noted carefully the topics he seemed most interested in. And, acting on this, I looked up carefully certain subjects, and, as a result, I managed to do very well.

Wilks always had a way of thinking aloud; I shall refer later to two instances of this. When I became Assistant-Physician to Bethlem Hospital I saw a good deal more of him. Soon after, I was appointed Lecturer on Insanity, and I was supported for that post strongly by Wilks, who, quaintly referring to a rival candidate, said that he had so many qualifications that he must be either a wonder or a fool. Anyway, I had the pleasure and

profit of visits by Wilks to Bethlem Hospital, and I wish I had kept notes of his many pregnant comments and remarks. He was always charmed with the honest straightforwardness of many maniacs, and he was pleased when a female patient, after asking his name, at once said, "Wilks, whelks, oysters, shell-fish," and he followed with interesting examples of association of ideas and of words.

I well remember his telling me that he was called into the country to see a patient who lived in a handsome house surrounded with every appearance of luxury. Wilks was told by the general practitioner that this man was suffering from melancholia with delusions as to sinfulness and poverty. Wilks recognised that the man was "sick unto death," and it was decided that he should be carefully nursed at home. On his death it was found that what he said was perfectly true, for he had been living extravagantly on trust money and had now come to the end of it. This case interested Wilks particularly, showing how possibly one might be mistaken if one trusted to the statement of the patient alone.

He criticised our classifications. For example, he said the most buoyant, bright, and active patients that he saw in the hospital were called general paralytics. He said, "This is prophetic, but prophecy is not usual in naming diseases."

All forms of nervous disease were of special interest to him; and when, in more recent times, syphilis and general paralysis were connected, he repeated his old dictum: how odd it was that for generations doctors considered syphilis, after affecting skin and bone, to stop always at the throat or rectum, not invading the body generally.

He was generally looked upon as a sceptic in therapeutics, but he had very strong faith in certain remedies. He was opposed to every form of what might be called generalised treatment on one principle. He was eclectic and empirical.

I may recall here an example of his experience and teaching. He said that Englishmen, as a rule, were never content unless their bowels were acting; in fact, that they seemed to consider

that with every revolution of the earth there ought to be a certain action of the bowels. He said that this never struck him so much as when, as a young man, he travelled with a Frenchman abroad, when he found that instead of the bowels normally acting once, in a Frenchman they seemed to act many times a day; and certainly, he thought, to their advantage, as he understood they never suffered from piles.

He had strong views about homœopathy and its followers. He was equally strong in his opposition to phrenology, while fully accepting the localisation of brain functions. I remember a student asking him whether the alkaline treatment for rheumatic fever was not rational. Wilks, for a time, made no reply; and then said to his clerks, "We will treat all our cases of rheumatic fever, for a time, with mineral acids, and you will see that the treatment on simple chemical lines is not to be accepted." He pointed out that there was no evidence that the acid was the cause of the fever, and not the consequence. Similarly, he attacked homœopathy because it pretended to treat like things with like things, and because belladonna produced a dry throat, it was useful for scarlet fever in which there was also a dry throat. He asked whether they wanted to treat a cause or one of a series of symptoms.

Wilks smiled in a pleasant way as fresh theories, not founded on pathology, appeared from time to time on the scene. He recognised the rise and fall of theories deduced only from symptoms or surroundings. In the wards he was not formal or pedagogic as a teacher. Much was to be learned from his methods and from his experience. I remember him saying that there is a class of industrious students who, note-book in hand, go round the wards with the physician and surgeon and write down the name of the patient and of the physician, then the disease, and opposite that the diet-list and the drugs. Such an one, reading the bed-card of a patient with pneumonia, saw that some brandy was ordered, also an ammonia mixture. In his mind at once was fixed, "Wilks, pneumonia, brandy, and ammonia." When, in later years, the student had become a

practitioner and sent for him in consultation in a case of pneumonia occurring in a full-blooded man for whom bleeding might have been good, he told Wilks he was treating pneumonia after his lines. This, of course, was a good example of Wilks's teaching. Such men have mental tables: one column for the disease, and one for the treatment, and possibly a third for the doctor's name. Such an one sent for him to see a congenital idiot, and he had given the child phosphorus in every form, with no beneficial result. Wilks merely said, "But you have not given him a convolution."

Tolerant of simple weakness, anything like sham or assumption produced a calm scorn which must have been gall and wormwood to the receiver. At Guy's he welcomed any man who had anything to teach. I shall always remember Dr. Laycock going round the wards with Wilks and laying down medical law very emphatically. Wilks was calm and interested, but afterwards was rather sarcastic on the self-satisfied Scotsman.

Among the most devoted outside followers of Wilks were H. G. Sutton and Hughlings Jackson of the London Hospital. In the early '60's they were constantly at Guy's; in the wards with Gull, and in the post-mortem room with Wilks, and irreverent students called them "Wilks's maggots." He early recognised their originality and their power, and valued them as friends all their lives.

I forget what started the idea, but once he said to me he would like to visit the Criminal Lunatic Asylum at Broadmoor. So one Sunday he and I spent the day—I think the week-end—with Dr. Nicolson. On the road down, I remember, he spoke of the benefit derived from old brandy after an influenzal attack. He said that ever since a severe attack of influenza he had been given to dram-drinking, and he found it beneficial. He certainly took sips of brandy on several occasions on the road down. He spoke then of the change in medical practice in relationship to treating fevers with alcohol. He believed very strongly that in nervous weakness or irritability following febrile diseases, alcohol might be of use, but he had no faith

in the large administration of alcohol. At Broadmoor he was introduced to criminal lunatics of all kinds, and was particularly interested in the young women who were there for infanticide. After the long day and many experiences, he said to Dr. Nicolson, "Is not the function of the physician to cure his patients? You say, 'Yes,' but do you honestly believe that you want to cure many of these people?" I regret, however, that I recall so little of what he said while in the Asylum or at Broadmoor.

Wilks delighted to have men of a younger generation about him, but he soon let bores know that they would not find in him a ready listener; he simply went on his own way, ignoring what they were saying.

When he was in Grosvenor Street, Sunday evenings were open for informal visits after about 8.30 p.m. Many times have I sat till near midnight in what one might call pleasantly mixed company: medical men, literary men, and an occasional artist or actor were to be met there. We used to go sometimes even when Wilks was out; and I remember his returning, on more than one occasion, and peeping into the drawing-room and, as usual, thinking aloud, and saying, "What a lot of them; what have they all come to-night for? There are not chairs enough for them, and I am sure there is not supper enough." There was chat and pleasant company. And after supper a few privileged persons were allowed to stop and smoke in his upper study. Such evenings left their effects on the younger men who were there, but little remains to be recorded, for the conversation was so general and ranged over every subject of the day.

Wilks, as I said, was not only constantly thinking aloud, but analysing himself and others in a quaint manner. I remember one foggy day him saying, "You know, the more highly educated you are, the less you talk." I asked for an explanation. He said, "Well, you see, there was a very thick fog, and I was coming up from the South of London, and when I got near the 'Elephant and Castle,' I had to get out and walk along the pavement, while my coachman kept practically in touch with

me. I then began to notice, as people passed me, that the most cultivated, the best dressed, in fact, looked at me and passed on. A class not so refined looked at me and turned round, perhaps for half a moment, to watch me and the carriage. But then, when it came to the lowest class, they stared, and one after another would say, 'Look at that old chap walking by his carriage!' They all thought it; it was only the lowest educated who expressed it." So in dealing with men you have to remember very often that the person who says most thinks least.

Without having any deep knowledge of the practical sciences, he was ever receptive, and willing to accept any fact which was established, but only such. Thus, he readily accepted the localisation of brain function, but was never wearying, as I have said, of attacking the phrenologist. In the same way, he followed with interest the work of the vivisectionist, and wrote at least one article exposing the fallacies of anti-vivisection.

He had an amusing way of saying that he thought he could do most things that he wanted to when he set his mind on doing them. Thus, once, he told me, he had to go down to a consultation near Rosherville. As there was no convenient train to take him back for two hours, he went into the Gardens, where he succeeded in knocking the pipe out of the mouth of Old Aunt Sally at the first shot. I do not, however, think he had any aptitude for games generally; I never heard him speak of sport as a thing he had ever taken part in. He loved to prowl.

I met him at Switzerland, and he thoroughly enjoyed the scenery, but I never succeeded in getting him to climb. He took a great interest in geology and antiquarian research, and up to the end he was an active member of the Hampstead Scientific Society, and wrote papers for it.

When appointed physician to the Duke of Connaught he said he feared he would not be at home at Court; and when he received his baronetcy he said he might have had the title earlier if he had been a William, for he noticed that titled members were mostly Williams or Williamses, and he could not remember a Samuel being titled in the profession before.

Wilks never did an overpowering practice, and yet it is rather surprising that this was so, for Guy's men were always willing to consult him about themselves. One day a Guy's man went to consult him. He happened to be the third who had consulted Wilks that morning. After carefully considering his case and advising him what to do, talking to himself he said, "What a large practice I should have if it were not for Guy's men!" This open speaking did not encourage practice. A tale is told of his going to the country to see a very loquacious, but hypochondriacal lady. After fully satisfying himself about her, he proceeded to examine the pictures on the walls, till the anxious-minded husband suggested that he had called him down to see his wife, and not his pictures.

Again, it is said that after examining a worn-out young man he muttered, "Age is relative; a worn-out old man at thirty." He saw one patient who suffered from an occasional fit of nocturnal epilepsy; these were always preceded by a throat aura. It was at the time that Hughlings Jackson was trying to stop fits by ligaturing the limb from which the aura started. Wilks, without explaining, sent the patient to get a throat spraying apparatus. The patient complained, when he reached home, of having been sent home "with a damned toy."

Many of Wilks's friends told him what he ought to do and what he ought not to do. As he said to me, "It is all very well, but if I followed their advice I should not be Wilks." This was eminently true, and no one would have liked the conventional instead of the true man.

I was struck that he was able to fill the chair of President of the Royal College of Physicians so well. No one was more severe in the obituary notices of Fellows. Anyone who had, by self-advertisement or pretence lowered the standard of the profession received no mercy.

Some years ago a few men—I believe Sir Bryan Donkin and Dr. Sturgess of the Westminster Hospital were the first—formed what was called "The Fifteen Club." This really consisted of fourteen men and Wilks. They were surgeons and physicians

who were, in thought and interest, admirers and followers of him. They represented all the London hospitals, and they met at the Café Verrey three times a year at dinner. Wilks was constant in his attendance, even to the last year of his life. All of us will treasure the memories of those evenings as among our very richest.

One of the most marked features of Wilks was his devotion to Guy's. It was an absorbing passion, and the last year or two of his life at Hampstead were occupied in writing a System of Medicine as seen from Guy's Hospital Reports. Others must refer to this part of his life, but his part in recognition of the discoveries of Bright's disease, Hodgkin's disease, and Addison's disease will always remain historical.

And now I have come to the conclusion of my notes, not only of my earliest teacher, but, to the end, my dearest friend.

CARCINOMA OF THE GALL-BLADDER ASSOCIATED WITH GALL-STONES.

By

J. FAWCETT and C. H. RIPPMANN.

The following paper is based on the records of Guy's Hospital of the cases of carcinoma of the gall-bladder during the forty years 1867—1906, inclusive. The abstracts of the cases form an appendix to this paper.

Carcinoma of the gall-bladder is a subject on which many dogmatic statements have been made as regards its origin and diagnosis, and we propose to draw attention to some points in connection with these in the first part of this paper.

With regard to the incidence and frequency of gall-stones, our statistics correspond, in the main, to those of previous observers. Out of 592 cases, in which gall-stones were found at the post-mortem examination, there were 48 cases of carcinoma of the gall-bladder accompanied by gall-stones, a percentage of 12·3.

Rolleston, in his treatise on "Diseases of the Liver, Gall-Bladder and Bile Ducts," states that primary carcinoma of the gall-bladder occurs in from 4 to 14 per cent. of all cases of cholelithiasis.

Of the total number of cases of carcinoma of the gall-bladder in the Guy's Hospital Records, gall-stones were found in 87 per cent., the figures given by different authors varying from 70 to 95 per cent. As regards sex, in the 48 cases there were 38 females and 10 males, or nearly four to one, a figure which corresponds to those of preceding observers. The ages of the patients in the series are given in the accompanying table, from

which it is seen that 38 of the 48 cases occurred between 41 and 70 years of age.

TABLE A.—AGE TABLE.

21—30 years	2 cases.
31—40 „	3 „
41—50 „	14 „
51—60 „	13 „
61—70 „	11 „
71—80 „	2 „
Age not stated	3 „
	48 cases.

The duration of life after the appearance of the first symptoms of illness is given in the following table; 34 cases, or 71 per cent. lived six months only, while a further 10 cases succumbed within twelve months, or 44 cases in all, and, therefore, over 90 per cent. of the total number.

TABLE B.—DURATION OF LIFE AFTER APPEARANCE OF THE FIRST SYMPTOMS.

Less than 1 month	4 cases.
2 months	7 „
3 „	7 „
4 „	6 „
5 „	3 „
6 „	7 „
7 „	2 „
8 „	1 „
9 „	1 „
10 „	2 „
11 „	0 „
12 „	4 „
13 „	1 „
15 „	1 „
17 „	1 „
Doubtful	1 „

On turning from the statistics to other features of our cases, the one which struck us most was the difficulty experienced in arriving at a diagnosis. As far as we are able to determine from the reports, a diagnosis of carcinoma of the gall-bladder was not made in any one of the 48 cases, a statement which, incredible though it may sound, yet, in reality, represents very fairly our present position in regard to diagnosis at the bedside of malignant disease of the gall-bladder.

When we compare this result with the statements made in some of the articles in text-books, and in monographs, we cannot help but question them, even if it were only for the credit of our physicians during the forty years which these cases cover. Thus, it is stated "frequently there is a history of gall-stone colic," "if gall-stones be present, there will be the usual antecedent history of colic," "in some instances the clinical history has pointed to the view that the jaundice was originally due to an ordinary attack of biliary colic."

On the other hand, Rolleston, in his book on "Diseases of the Liver," seems to us to take a more correct view when he says that often there is no history of gall-stone colic in fatal cases of carcinoma of the gall-bladder, and he quotes Kehr to the effect that clinical evidence of cholelithiasis is wanting in the majority of the patients.

Which of these views is the true one, and does our analysis help us to decide? To try to answer this question we have investigated some of the chief symptoms and signs recorded in the 48 cases, the details of which will now be referred to.

I. *Abdominal pain* (*vide* Table C).—Pain is noted as having been experienced for a longer or shorter period in 33 of the 48 cases, or 68·7 per cent. In only six of them could the pain be described as paroxysmal in type, and in not more than two of them was it in any way suggestive of "biliary colic." These two cases are Nos. 10 and 47. In Case No. 10, where death took place at the age of 42 years, the history states that the patient had suffered from jaundice attributed to "gall-stone" when she was 25 years of age, and that the year before admission she again became jaundiced, and had attacks of abdominal pain over a period of seven weeks. In Case No. 47 the symptoms occurred at intervals for four years, and eleven gall-stones were removed about two years before her death. In the other four cases, Nos. 13, 30, 36, 40, the pain commenced two, five, three, and eight months respectively before admission, that is to say, during the period in which, in all probability, the "new growth" was developing. The pain,

although paroxysmal, was of an indefinite type. In case No. 13, it was described as consisting of "two months' colic in epigastrium." In case No. 30, as "pain at intervals in the right side, for several years, varying in duration from a few days to six or seven weeks." In case No. 36, as "an agonising pain on several occasions, one attack lasting for half an hour." In Case No. 40, as consisting of "violent pain in the stomach, more severe for six weeks preceding admission to hospital."

Although it is a well-known fact that the pain occasioned by gall-stones is by no means always of the nature of a typical "biliary colic," yet in this group of cases it is less frequently so than usual. If the character of the pain is considered apart from the other signs or symptoms of the disease, there is little typical about it; in the four cases just alluded to, the attacks of pain were of such a nature as are often seen in association with malignant disease of the stomach, of the large intestine, and other abdominal viscera.

In the remaining 27 of the 33 cases the character of the pain was too indefinite to allow of any diagnostic importance being attached to it. In eleven of these cases pain was felt in the right side, and is described as "pain" without any qualifying adjective descriptive of its nature.

Now let us turn to the period of time before admission during which pain was experienced. In two cases only, Nos. 10 and 47, was the period of such a length as to have undoubtedly preceded that which marked the commencement of the "new growth." The duration of the pain in 31 cases is stated in the following table :—

15 months	before admission...	1 case.
12 "	" ...	1 "
11 "	" ...	1 "
9 "	" ...	4 cases.
8 "	" ...	1 case.
5 "	" ..	3 cases.
4 "	" ...	3 "
3 "	" ...	4 "
2 "	" .	11 "
Under 1 month	" ...	2 "

In the remaining 15 of the 48 cases in this series pain is not referred to in the reports.

The conclusions arrived at as the result of the investigation of the symptom of pain are as follows:—

1. That so-called "biliary," or "gall-stone colic," is seldom present in cases of carcinoma of the gall-bladder associated with gall-stones.

2. Consequently, that if attacks of biliary colic have taken place, their occurrence is in favour of the attacks being due to gall-stones unaccompanied by malignant disease of the gall-bladder.

3. As a corollary to the above statements it follows that surgical interference in cases of gall-stones should not be advised on the ground that carcinoma of the gall-bladder is likely to ensue, for in the majority of cases of carcinoma of the gall-bladder the pain does not partake of the character of a biliary colic. When pain is experienced in cases of carcinoma of the gall-bladder accompanied by gall-stones, it is atypical, and generally coincides in its appearance with, and is probably dependent upon, the development and progress of the "new growth."

In making these statements we do not, for a moment, suggest withholding surgical interference in cases of "colic" thought to be due to gall-stones, but to draw attention to the fact that if attacks of typical biliary colic have occurred there is a well-grounded hope of the absence of carcinoma of the gall-bladder, and that in consequence the prognosis is much better. We are, therefore, justified in holding out this prospect to our patients, without tempering our judgment and prognosis by the fear at the back of our minds that surgical exploration may reveal a neoplasm of the gall-bladder as well as calculi.

4. That although the diagnosis of carcinoma of the gall-bladder is, apart from surgical intervention, likely to remain uncertain, yet one of the reasons for failure to arrive at a probable diagnosis in the past is the misleading statements already referred to; for example, that "the usual antecedent history of cholelithiasis is to be looked for," whereas the truth is that such a history exists in a very small proportion of cases.

TABLE C.—Pain.

Case No.	Name.	Duration of Symptoms before Admission.	Duration of Life after Admissi n.	Character of Pain.
3	Esther S. ...	15 months in all	Only a few days in ward.	A "drawing sensation." Unable to lie on left side. Catching pain on exertion for 6 months.
4	Annie L. ...	5 months	—	Good deal of pain, increasing in severity and preventing sleep. Preferred to lie on left side.
5	Jane R. ...	9 months	8 months ...	Now and then a little pain in right hypochondrium.
6	Amelia S. ...	3—4 months	—	Pain in right hypochondrium.
7	Sarah W. ...	4 months	About 2 weeks...	Severe pain in right hip, spreading to right side of abdomen.
10	Eliza J., æt. 42	At 25 years of age had jaundice attributed to a "gall-stone." A year before admission again jaundiced, and had attacks of abdominal pain for 7 weeks.	—	Colic.
12	Mary Ann M. ...	3—5 months	—	At first pain on coughing, and more or less continuous pain in hypochondriac region for about 3 months.
13	William W. ...	2 months' "colic" in epigastrium.	- -	Paroxysmal, epigastric pain.
14	Maria R. ...	2 months	—	Pain in right side of abdomen.
16	Mary H. ...	3 weeks	1 month ...	General abdominal pain and pain in back.

TABLE C.—PAIN (*continued*).

Case No.	Name.	Duration of Symptoms before Admission.	Duration of Life after Admission.	Character of Pain.
17	Esther G. ...	9 months	1 month ...	Pain in right side.
21	Susan B. ...	2 months	—	Abdominal pain.
22	Sally C. ...	3 months	3 weeks ...	Aches and pains in abdomen. Pain worse in right side for 6 weeks.
27	Harriet M. ...	12 months before admission pain in region of gall-bladder for a day or two. 4 months before death a similar attack with jaundice.	3 weeks ...	Colicky attack.
28	William K. ...	2 months	8 days	Severe epigastric pain.
29	Susannah A. ...	2 months	A few days ...	Pain on pressure in hepatic region.
30	Sarah F. ...	5 months	14 days	Pain in right side for several years (?6—14) at intervals of a few days to 6—7 weeks.
31	Mary B. ...	9 months	1 month ...	Pain in right hypochondrium.
32	Catharine B. ...	11 months	1 month ...	General abdominal pain.
33	Mary Anne A....	7 weeks	3 weeks ...	Pain in back and between shoulders.
34	T. N. (a female)	2 months	—	Gnawing pain in the left side. Later attacks of pain two or three times a week.

TABLE C.—PAIN (*continued*).

Case No.	Name.	Duration of Symptoms before Admission.	Duration of Life after Admission.	Character of Pain.
35	Ellen D. ...	14 days	—	Pain in left side. Symptoms of chronic intestinal obstruction.
36	Eliza J. ...	3 months	1 month ...	Agonising pain on several occasions, one attack lasting for half an hour.
37	William W. ...	3 months	3 months ...	Slight abdominal pain.
38	Martha C. ...	3 months	3 months ...	Abdominal pain.
39	Richard C. ...	2 months	1 month ...	Aching pain and tenderness in right side.
40	Harriet, P. ...	8 months	2 months ...	Violent pains in stomach 8 months before admission. More severe pain for 6 weeks.
41	Henrietta F. ...	9 months	3 months ...	Pain in middle of back and nausea; also pain in left and right sides.
42	Lavinia P. ...	4 months	1 month ...	Attack of indigestion, after which inability to sit up without pain.
45	Josephine B. ...	2 months	1 month ...	Severe pain in left lumbar region 2 months before admission.
46	John L. ...	7 weeks	2 weeks ...	Abdominal pain.
47	Jane A. ...	4 years (probably)	2 months ...	Pain in abdomen. 11 gall-stones removed in 1903.
48	Emily M. ...	2 months	2 days	Abdominal pain and vomiting attacks for a week.

II. We now proceed to consider another symptom, *jaundice*.

Jaundice existed, at some period of the illness, in 29 of the 48 cases, or 60 per cent. The degree of jaundice is not referred to in most of them. The following conditions were found at autopsy in these cases:—

1. Secondary deposits in portal glands, 3 cases.
2. Secondary deposits in the liver, or enlargement of the liver, 18 cases.
3. Gall-stones in common bile duct and hepatic ducts, 12 cases.
4. Common bile duct narrowed or obstructed by new growth, 7 cases.
5. Hepatic ducts dilated, 11 cases.
6. Common bile ducts dilated, 7 cases.

In 22 of the 29 cases the ducts of the liver and the gall-bladder attracted the special attention of the demonstrator. In 12 cases calculi were present either in the hepatic ducts or in the common bile ducts, and in nine of these the ducts are described as dilated, four of them showing an inflamed condition with pus in the ducts or in the gall-bladder. In four of these twelve cases the common bile duct, or hepatic ducts, were involved in an extension of the new growth. In five cases the common bile duct was invaded or obstructed by the new growth. In the remaining five cases the hepatic ducts are described as dilated, and, in addition, a suppurative cholangitis, with or without pus in the gall-bladder, existed in four of them.

Thus, a mechanical factor appears to be one of the chief causes of the jaundice which is due, either to obstruction of ducts by "new growth," to inflammation of, with exudation into, the biliary channels, or to the presence of calculi in conjunction with one or both of the previously mentioned conditions. In the remaining 7 of the 29 cases, where the ducts are not described as involved, six cases presented secondary deposits in the liver; one, abscesses in the liver; and in one case, in which secondary deposits were present, the gall-bladder contained pus.

III. *Tumour* (*vide* Table D).—In an earlier part of this paper attention was directed to the fact that a diagnosis of carcinoma of the gall-bladder had not been made in any of the 48 cases referred to. Consequently the statements in text-books to the effect that "a tumour may be felt in more than half the cases," or "a characteristic tumour can be detected in the region of the gall-bladder," sound improbable, and when we compare the signs in our 48 cases with the condition found at the autopsy the difficulty experienced in diagnosis becomes intelligible, while text-book statements like those just referred to convey a false impression to our minds.

(*a*) A tumour was felt during life in 26 of the 48 cases. In 10 of the 26 cases the swelling was thought to be an enlarged liver, and this was proved correct at the autopsy. Of the remaining 16 cases, there were 5, namely, Nos. 13, 14, 27, 31, 41, in which the description of the tumour felt during life might have suggested the presence of an enlarged gall-bladder, but even this number, 5 out of 48, is a good deal less than "half the cases," in which it has been stated that a characteristic tumour is found. The swelling is described as follows in the five cases referred to :—

Case 13.—There was a projection over the gall-bladder region, and the liver was enlarged.

Case 14.—The gall-bladder was felt to be full of gall-stones, and the liver was enlarged.

Case 27.—A globular mass was felt in the region of the gall-bladder.

Case 31.—An oval swelling beneath the costal margin, and a nodule on the edge of the liver.

Case 41.—A rounded mass was felt at the outer margin of the right rectus muscle.

On turning to the condition found at the autopsy, it is clear that what was felt in these cases, with the exception of No. 14, was the new growth surrounding the gall-bladder, and extending into the liver.

Thus, in No. 13 the gall-bladder is described as embedded in a mass of cancer, its wall being much thickened, and its cavity about the size of a hazel nut. In No. 27 the wall of the gall-bladder was infiltrated with growth extending into the liver. In No. 31 the wall of the gall-bladder was much thickened by new growth, extending for a short distance into the liver. In No. 41 a mass of soft growth, 8 inches in diameter, was found replacing the gall-bladder.

In the remaining 11 of this group of 26 cases, the tumour (*vide* Table D) did not exhibit sufficiently distinctive characters to indicate the gall-bladder as the primary site; it is portrayed in a vague way which expressed probably the feeling of doubt in the mind of the physician as to its origin. For example, it is described in the reports as "a swelling in the right hypochondriac and iliac regions," "a tumour in the right hypochondrium, reaching nearly to the iliac fossa," "a tumour in the right lumbar region," and so on.

(*b*) Now let us proceed to the description of the pathological conditions ultimately found, and see if any further explanation can be discovered of the failure to arrive at a diagnosis.

In 19 of the 48 cases the gall-bladder is described as contracted, or shrunken up, under the liver, its wall being infiltrated with growth. It was, therefore, impossible to detect a "characteristic tumour" in this group.

In 6 cases the report states that the wall of the gall-bladder was infiltrated or thickened by the growth, which extended into the liver. It seems that the swelling felt in five of these cases was either the enlarged liver, or secondary deposits, or a mass of new growth in the gall-bladder region which consisted of an extension of the growth into the liver. The impossibility of determining, clinically, the origin of a mass of this kind, with any approach to certainty, is obvious; it could only be guess-work, inasmuch as an antecedent history, suggestive of biliary colic or other symptoms pointing to disease of the gall-bladder, is rarely present.

In 8 cases (Nos. 12, 17, 18, 35, 36, 38, 40, 48) the gall-bladder is described as being infiltrated, or thickened, by growth.

In the clinical records of six of these cases (Nos. 18, 35, 36, 38, 40, 48) there is no reference to the existence of a tumour of any kind, while in No. 12 there was said to be a lump in the right side of the abdomen, and "the liver was enlarged and tender," and in No. 17 "the liver was enlarged." Thus, in 33 of the 48 cases, or 68·7 per cent., there was no "characteristic tumour."

In the remaining 15 of the 48 cases, the conditions recorded in Table D were found. In eleven of them the gall-bladder was involved in a mass of growth of considerable size, for example, as large as a fist, or a cricket ball, or larger. In this group of cases it would seem that a "characteristic tumour" might have been felt during life, but when reference is made to the clinical records, it is seen that in only six of them (Nos. 23, 32, 34, 39, 44, 46) was a tumour felt, and in two (Nos. 44 and 46) there was nothing whatever to point to the gall-bladder as the original site of the mischief.

The following is the description of the tumour in these cases:

Case 23.—A tumour in the right hypochondrium, reaching nearly to the iliac fossa.

Case 32.—A tumour in the right hypochondrium.

Case 34.—A tumour in the right side of the abdomen, extending four and a quarter inches below the costal margin.

Case 39.—A large hard mass in the right hypochondrium, extending into the lumbar and iliac regions.

Case 44.—A tumour in the right lumbar region.

Case 46.—A diffuse swelling below the right costal margin, which was incised and pus evacuated.

In this group of six cases, the tumour, if associated with jaundice or other signs and symptoms suggestive of interference with the biliary canals, might have led to a probable diagnosis of disease of the gall-bladder, provided that involvement of other organs could be excluded. On the other hand, the clinical records of our cases prove that such a differentiation is exceedingly difficult, and that the opportunity of arriving at a definite diagnosis of carcinoma of the gall-bladder is rarely afforded to us.

TABLE D.—TUMOUR.

Case No.	CONDITION DURING LIFE.	CONDITION AT AUTOPSY.
1	Swelling in hypochondriac and iliac regions	Gall-bladder contracted. Full of gall-stones and surrounded by growth, which extended deeply into the liver. There was a large abscess in right side of abdomen, communicating with duodenum, but shut off from general peritoneal cavity.
2	A small lump in right loin, increasing in size.	Gall-bladder embedded in mass of growth, and so shrunken as hardly to admit little finger.
3	A hard mass in right side of abdomen felt 6 months before admission.	Gall-bladder embedded in growth, the cavity being the size of a walnut. Liver greatly enlarged.
4	——	Large tough fibrous mass, including a gall-stone, occupying the position of gall-bladder. Liver enlarged.
5	Liver increased in size under observation, and nodular masses felt in it.	Gall-bladder formed a white shrunken mass, from which the liver was invaded.
6	——	Gall-bladder much contracted.
7	Liver large and irregular.	Liver much enlarged by secondary deposits, particularly in neighbourhood of gall-bladder. Pancreas and duodenum infiltrated by growth.
8	——	Gall-bladder contracted; immediately around it a large mass of growth extending into the liver.
9	——	Gall-bladder contracted around gall-stones.
10	——	Wall of gall-bladder infiltrated by growth, which extended into the liver.
11	——	Gall-bladder contracted and full of gall-stones, continuous with patch of new growth extending into liver.
12	Lump in right side of abdomen. Liver enlarged and tender.	Wall of gall-bladder infiltrated to thickness of half an inch by new growth.
13	Projection over gall-bladder region, and liver enlarged.	Gall-bladder embedded in a mass of cancer, its walls being much thickened, and its cavity about size of a hazel nut.
14	Gall-bladder filled with gall-stones and palpable. Liver enlarged.	Gall-bladder dilated and contained 20–30 gall-stones, its walls being infiltrated by growth, which spread into the substance of the liver.
15	——	Wall of gall-bladder replaced by new growth.
16	——	The growth of the gall-bladder formed a large mass.

TABLE D.—TUMOUR (*continued*).

Case No.	CONDITION DURING LIFE.	CONDITION AT AUTOPSY.
17	Liver enlarged (98 oz.).	Wall of gall-bladder a quarter of an inch in thickness. Contents 2 dr. of creamy pus and four gall-stones one-third of an inch in diameter.
18	——	Wall of gall-bladder a quarter of an inch in thickness.
19	——	Gall-bladder compared to a scarred fibrous sac, with a nodule in wall extending into liver.
20	Liver enlarged.	Gall-bladder the size of a pigeon's egg, with growth around neck extending into liver.
21	Increase in area of hepatic dulness.	Gall-bladder region occupied by mass of growth the size of an orange.
22	Large hard mass on right side between costal margin and anterior superior spine of ilium.	Wall of gall-bladder infiltrated by growth extending into liver.
23	Tumour in right hypochondrium reaching nearly to iliac fossa.	Gall-bladder surrounded by mass of growth as large as a fist.
24	Liver enlarged and hard.	A tumour of gall-bladder as large as a cricket ball invading liver.
25	——	Gall-bladder contracted, its distal half invaded by growth half an inch in thickness.
26	——	A mass of growth two and a half inches in diameter extending into liver.
27	A globular mass felt in region of gall-bladder.	Wall of gall-bladder infiltrated with growth extending into liver.
28	Liver with hard edge and two nodules in it.	Gall-bladder the size of an egg infiltrated with growth.
29	Hard nodular enlargement of the liver.	A tumour of gall-bladder as large as a cricket ball, the growth extending into the liver.
30	——	Gall-bladder shrunken and infiltrated with growth, which extended into the liver and head of pancreas.
31	An oval swelling beneath the costal margin, and a nodule on the edge of the liver.	Wall of the gall-bladder much thickened by new growth, extending for a short distance into the liver.
32	A tumour in the right hypochondrium.	A tumour of the gall-bladder the size of a cricket ball projecting from the liver.
33	Liver much enlarged, and tender mass above umbilicus.	Gall-bladder thickened and firmly contracted round a gall-stone measuring 1 by 1½ in.

TABLE D.—Tumour (*continued*).

Case No.	CONDITION DURING LIFE.	CONDITION AT AUTOPSY.
34	A tumour on right side of abdomen extending 4½ in. below costal margin.	Gall-bladder much distended, with thickened wall projecting from under the liver.
35	——	Gall-bladder infiltrated by growth.
36	——	Gall-bladder thickened.
37	Liver enlarged and an irregular mass felt in it.	Gall-bladder contracted, with growth extending from it into liver.
38	——	Wall of gall-bladder thick and infiltrated with growth.
39	A large hard mass in right hypochondrium extending into umbilical and right iliac region.	Mass of soft growth 8 in. in diameter was found replacing the gall-bladder.
40	——	Gall-bladder thickened and infiltrated by growth.
41	A rounded mass at outer margin of right rectus muscle.	Gall-bladder infiltrated with growth shrunken up under liver.
42	Liver enlarged.	Wall of gall-bladder infiltrated by growth invading the liver. Liver much enlarged by secondary deposits.
43	——	Mass of growth around gall-bladder.
44	A tumour in right lumbar region.	Gall-bladder infiltrated with growth formed a mass 9 in. in length in the under surface of the liver. A large secondary deposit in the right lobe of the liver.
45	——	Gall-bladder, its wall thickened with growth, contracted up under liver.
46	A diffuse swelling below right costal margin, which was incised and pus evacuated. The wall was stated to be infiltrated by growth.	An abscess cavity containing gall-stones, the growth extending into the liver. Secondary deposits in liver.
47	——	——
48	——	Wall of gall-bladder infiltrated by growth. The cavity contained 1½ oz. of pus. The liver was much enlarged.

Therefore, after an analysis of the cases we arrived at the conclusion that a "characteristic tumour" in cases of carcinoma of the gall-bladder is exceptional, the explanation being that in many cases the gall-bladder, infiltrated by growth, is contracted up beneath the liver, and consequently cannot be felt. In other cases, where a tumour is palpable, it consists of a mass in which the gall-bladder is embedded, and which extends into the liver; or it is a large swelling with boundaries so ill-defined as to render it impossible to locate the original site of the disease from the information gained by the examination of the tumour alone.

At the best, diagnosis at the bedside is only possible in a small percentage of cases, and, therefore, surgical exploration is the only method left to us of securing definite information of the site of origin of the tumour and is the method which should be adopted where a reasonable prospect exists of the presence of a new growth likely to prove amenable to treatment.

IV. Gall-Stones in their relation to the Development of Carcinoma of the Gall-Bladder.

In a paper read by Dr. J. P. Candler before the Royal Society of Medicine (Pathological Section) on January 17th, 1911, it was upheld that the frequency of the association between gall-stones and carcinoma of the gall-bladder had been over-estimated. The total number of autopsies investigated was 2,228. Gall-stones were found in 315, a percentage of 14·13, and there were only two cases of carcinoma of the gall-bladder, both of which were associated with gall-stones.

Dr. Hale White concludes that about 20 per cent. of those who have gall-stones ultimately suffer from malignant disease of the biliary passages; Dr. Rolleston, 4 to 14 per cent.

Dr. Candler does not think that these figures can be taken to represent the incidence among the general population, as they are based upon cases seeking hospital advice on account of signs and symptoms due to the cancer, and, therefore, the true proportionate relation between cases of cancer and gall-stones is

upset. He points out that if the figures above related were correct, there ought to have been as many as 40 to 60 cases of cancer of the gall-bladder in the Claybury statistics. His conclusion from the evidence obtained from autopsies at the Claybury Asylum is "that the incidence of primary carcinoma of the gall-bladder and bile passages in their relation to gall-stones among the general population is by no means so great as hospital statistics would lead us to think, and, consequently, there is no sufficient justification for assigning to gall-stones so important a rôle in the production of primary malignant disease of the gall-bladder."

This conclusion seems to us a fair representation of the facts, and helps to explain some of the curious features to which attention has already been drawn.

We have referred to the absence of typical attacks of biliary colic in these cases, to the fact that when pain was experienced it was of an indefinite type, and in many cases of no longer duration than would correspond with the development of the new growth. If a stone is a factor in originating malignant disease, it is, at any rate, a feature of interest that colicky pain of the type commonly associated with biliary calculi is so infrequently experienced.

Again, such a statement as the following, that when growth succeeds on calculi the symptoms due to the latter often pass into abeyance, is not borne out by our cases. The right explanation, to our mind, is that brought forward by Dr. Candler, namely, that hospital statistics have led to an exaggerated estimate of the proportion of cases of gall-stones which are ultimately followed by carcinoma. If it is correct, that a well-formed stratified calculus can be formed in six months, then there would be ample time for the formation of stones during the progress of development of the new growth, the inflammation of the gall-bladder, and disintegration of the cells and glands in its wall providing the deposits of cholesterin.

The presence of cholecystitis, an essential to the formation of gall-stones, is seen at many of the autopsies in these cases. Also,

the "new growth" often causes obstruction, and thereby favours stagnation of the bile and the possibility of infection by micro-organisms with a consequent cholecystitis.

The fact that gall-stones occur less frequently in cases of carcinoma of the bile ducts than in those of carcinoma of the gall-bladder is, to our view, no evidence in favour of the carcinoma being dependent upon the presence of the gall-stones, for, as previous observers have shown, in the bile ducts there is no direct interference with the lining membrane and glands of the gall-bladder whence the cholesterin is derived.

We may similarly dispose of the statement that secondary growths of the gall-bladder are not accompanied by the formation of gall-stones, inasmuch as they have not the same relation to the mucous lining as a primary growth, the outer layers only being involved, and in consequence they are not attended with the destructive changes which occur in cases of primary growths of the gall-bladder. Again, the duration of life after a secondary deposit has taken place is, in most cases, too short to allow of the formation of a gall-stone.

Thus, we feel justified in emphasising the point made earlier in this paper, that a diagnosis of gall-stones does not call for operation on account of the danger of development of carcinoma, but rather because of the complications likely to ensue from infection and inflammation of the gall-bladder and of its connecting ducts.

V. In addition to the points already dealt with in this paper there are one or two groups of cases of special clinical interest.

Group I.

The symptoms and physical signs were those due to the effect of the disease upon the body generally, cases which may be described as "latent." The cases in this group fall under two headings :—

(*A*) Those in which the symptoms were dependent upon a progressive cachexia without any localising signs. This "latent"

group is well recognised in relation to malignant disease in other organs, perhaps particularly in the case of the stomach, although it is sufficiently common in similar affections of the pancreas, intestines, and other viscera.

Case 11.—Mark G., æt. 62, was admitted for what is described as bilious vomiting of two months' duration. He rapidly became thinner, and on the day of his death, which took place three weeks after admission, he vomited a considerable amount of blood. *Autopsy*. The gall-bladder was contracted and full of gall-stones, closely packed, causing projections on the surface. The wall was much thickened, and from its upper surface a circular patch of new growth spread out into the liver. The wall of the gall-bladder was quite inseparable from this mass of growth. There were two enlarged glands in the portal fissure, and a number of small nodules in the omentum and mesentery.

Case 16.—May H., æt. 34, was admitted with œdema of the legs of six weeks' duration. She had been confined four months before admission. She was much emaciated, vomiting was frequent, the veins of the abdominal wall were distended. The vomiting persisted, and the patient, becoming gradually weaker, died three weeks after admission. *Autopsy*. The growth of the gall-bladder formed a large, dense, white mass; its interior contained a number of gall-stones. The wall of the growth was half an inch in thickness at the neck of the gall-bladder; it had invaded the substance of the liver for about a quarter of an inch, and was also beginning to infiltrate the pancreas. There were secondary deposits in the pleura and heart; the abdominal aorta was embedded in a mass of growth. The inferior vena cava was compressed, and the common iliac veins occupied by ante-mortem thrombus. The duodenum was narrowed by adhesions in the region of the gall-bladder.

Case 25.—Rebecca H., æt. 43, was admitted for wasting of two months' duration. She became gradually weaker and died a month later. *Autopsy*. The gall-bladder was found to be small and to be contracted in its proximal portion around three faceted stones; the distal half was infiltrated by growth, its wall being

half an inch thick and its lumen a mere slit. The gall-bladder was adherent to the transverse colon. There were many small secondary deposits in the peritoneum, on the surface, and a few in the substance of the liver, and also in the glands alongside the aorta. The liver was small, weighing only 30 ounces. The coils of intestine were more or less matted together.

(*B*) Those in which the symptoms and signs were due to secondary deposits of new growth. The signs of the primary disease, if present, were overlooked. Cases of ascites—

Case 6.—Amelia S., æt. 43, was admitted for ascites of doubtful origin. Death took place suddenly three and a half weeks later. *Autopsy*. The gall-bladder was much contracted, contained one small stone, and was adherent to the duodenum; its coats were thickened with whitish growth, and Dr. Fagge writes, "I have no doubt that this had been the primary lesion." There was a large cancerous subperitoneal mass in the pouch between the uterus and the rectum with secondary deposits also in the omentum and peritoneum elsewhere, and also in the pleuræ.

Case 8.—Sarah S., æt. 58, was admitted for ascites of five months' duration and vomiting. She had been ailing for a year. Death occurred ten days later. *Autopsy*. The gall-bladder was contracted, its surface woolly and ulcerated, and immediately around it was a large mass of growth extending into the liver in all directions. The common bile duct was distended to a size greater than that of a little finger with a number of small gall-stones compressed into one mass. There were numerous secondary deposits in the peritoneum with many adhesions between the stomach and the intestines.

Case 15.—Susan M., æt. 55, was admitted for vomiting, ascites, and extreme emaciation. Paracentesis abdominis was performed. Death took place 14 days after admission. *Autopsy*. The wall of the gall-bladder was formed of new growth, and its semiloculated interior contained several gall-stones. There were secondary deposits of growth in the peritoneum, the omentum, and mesentery.

Case 30.—Sarah F., æt. 63, was admitted for swelling of the legs and abdomen. Ten days later she developed diphtheria, to which she succumbed. *Autopsy*. The gall-bladder was found to be shrunken and its wall infiltrated with growth which extended into the liver and to the head of the pancreas. Its cavity was full of small stones. There were secondary deposits in the liver and in the glands in the portal fissure.

Group II.

The cases succumbed to some independent disease which may have rendered the recognition of the malignant disease difficult or impossible, or, on the other hand, the cancer was not sufficiently developed to produce symptoms and signs of its presence.

Case 9.—Mary B., æt. 62, was admitted with a femoral hernia which was operated upon; death occurred eight days later. *Autopsy*. The gall-bladder was found to be contracted around a collection of hard faceted gall-stones ; its neck was greatly indurated and thickened by an opaque white gristly growth, which histologically is stated to be "a typical carcinoma." The common bile duct was dilated throughout its length.

Case 19.—Fred. K., æt. 77, was admitted in a semi-comatose state, following a paralysis of the left arm of sudden onset, which had taken place a week previously. *Autopsy*. The gall-bladder was a scarred fibrous sac containing a number of stones (over 200) of various sizes. In the wall was a nodule of growth the size of a marble extending into the liver. Histologically it was regarded as a carcinoma. There were secondary deposits in the glands of the mesentery and in the visceral peritoneum.

Case 26.—Mary K., æt. 56, was admitted with an ovarian tumour, and when the abdomen was opened some peritonitis in an early stage was found. *Autopsy*. A mass of white growth measuring two and a half inches transversely and extending backwards about four inches into the liver substance was found occupying the site of the gall-bladder, which itself was packed

with small faceted stones. There were numerous secondary deposits in the liver. The common bile duct appeared healthy, and was not dilated.

Group III.

Cases of intestinal obstruction (Nos. 18, 35, and 45 may also be included in Group I. (*B*)).

Case 18.—Sarah S., æt. 59, was admitted for chronic intestinal obstruction of nine weeks' duration, the diagnosis being ? carcinoma recti. Lumbar colotomy was performed. *Autopsy.* The gall-bladder was greatly thickened, its walls a quarter of an inch thick, its cavity filled with scores of small faceted gall-stones. The omentum was thickened, and in it and in the mesentery were many masses of firm white fibroid material thought to be of "malignant" type. The mesenteric glands were large, hard, and white.

Case 35.—Ellen D., æt. 70, was admitted with chronic intestinal obstruction. For five months she had suffered from ingravescent constipation, and fourteen days before admission the obstruction became more marked, with vomiting for four days. Anastomosis was established between the small intestine and the transverse colon. Death ensued five days after admission. *Autopsy.* Secondary deposits of growth were found in the peritoneum, a stricture of the gut having formed as the result of a local peritonitis. The gall-bladder was infiltrated by growth, and contained about 26 calculi.

Case 40.—Harriet P., æt. 52, was admitted with chronic intestinal obstruction of eight months' duration; she was somewhat jaundiced and wasted. A growth was found involving the hepatic flexure; a lateral anastomosis was performed, and death ensued five days later. *Autopsy.* The gall-bladder was found to be much thickened and infiltrated by growth which had extended into and constricted and kinked the transverse colon. The liver was also invaded. In the common bile duct were three or four calculi, one of which was fixed in the duct at its lower end.

There were some smaller stones in the hepatic ducts, which were everywhere dilated. The glands in the portal fissure contained secondary deposits, and the portal vein was thrombosed.

Case 45.—Josephine B., æt. 49, was admitted for a tumour in both iliac regions, wasting, and recent symptoms of intestinal obstruction. A mass of secondary growth was found constricting the colon and sigmoid flexure. The gall-bladder was contracted up under the liver and its wall thickened by growth. *Autopsy.* The gall-bladder was found to be contracted up under the right lobe of the liver, and its wall was thickened by growth. Its interior contained a large oval stone with a number of small ones. In the adjacent part of the liver was a deposit having the diameter of a half-crown piece. The glands alongside the abdominal aorta were enlarged by secondary deposits, and extending out from these into the pelvis was a layer of growth to which the colon and sigmoid flexure were not only adherent, but narrowed by the extension of the neoplastic tissue around them.

APPENDIX OF CASES.

Case 1. — *Carcinoma of the gall-bladder. An abdominal abscess communicating with the duodenum.* — Julia C., æt. 46, was admitted in November, 1866, under the care of Dr. Habershon, and died two months later. In July, 1866, she had noticed a swelling in the right hypochondriac and iliac regions. She had occasional attacks of vomiting, and had become very thin.

Autopsy.—The gall-bladder was contracted, full of gall-stones, and surrounded by "growth" which extended deeply into the liver. The hepatic ducts were dilated to the size of a little finger and were filled with thick grumous bile. Calculi were present in the hepatic and in the cystic ducts. The portal glands were enlarged. There was a large abscess in the right side of the abdomen communicating with the duodenum, but shut off from the general peritoneal cavity. *See Insp.*,* 1867, No. 9.

* Insp. refers to the records of Post-mortem Inspections kept in the Curator's Room in the Museum.

CASE 2. — *Carcinoma of the gall-bladder. Secondary deposits in the liver, lungs, and pleuræ.*—Frances H., æt. 65, was admitted in 1873 under Dr. Pye-Smith. At Christmas, 1872, she had noticed a small lump in the right lumbar region which continued to grow. When admitted she was much wasted and deeply jaundiced. Death ensued about seven months after the time when the tumour was first noticed. Previous to this the patient had always enjoyed good health.

Autopsy.—The gall-bladder was embedded in a mass of growth. It was so shrunken as hardly to admit the little finger into its interior, and it contained many very small stones. The hepatic ducts were much dilated. There were secondary deposits in the liver, the pleuræ, the lungs, the pelvic peritoneum, and the omentum. *See Insp.*, 1873, No. 302.

CASE 3. — *Carcinoma of the gall-bladder. Secondary deposits in the liver. Gall-stones.*—Esther S., æt. 46, was admitted under Dr. Moxon in November, 1873. Since her last confinement, fifteen months before, she had been unable to lie upon her left side on account of a "drawing sensation" in the opposite shoulder. She also complained of a "catching pain" on exertion. Six months since a hard swelling was detected in the right side of the abdomen, and a month before admission the skin became yellow. When admitted she was deeply jaundiced; occasionally she suffered from great pain which was increased by taking food; she left the hospital after a month, and did not return until August, 1874, when the tumour was found to have greatly increased in size, and there was general anasarca. Death ensued a few days later.

Autopsy.—The gall-bladder was found to be embedded in "growth," which in this region was more fibrous and older than that in the liver. The cavity of the bladder, in size about equal to a walnut, contained a dozen small faceted stones. The liver was enormously enlarged by masses of growth. *See Insp.*, 1874, No. 310.

CASE 4. — *Carcinoma of the gall-bladder. Gall-stones.* — Annie L., æt. 57, was admitted in 1875 under Dr. Pye-Smith. She had been in good health until five months previously when she began to feel pain in the right side. She was wasted and had become increasingly yellow for three weeks. Death was preceded by a drowsy condition, which gradually deepened.

Autopsy.—A large, tough, fibrous mass occupied the situation of the gall-bladder, which itself was not recognisable as to its cavity, except where a gall-stone was enclosed and surrounded by a thick, almost purulent mucus. The liver was large and the hepatic ducts were dilated. The growth was described by Dr. Goodhart as possessing the histological character of a polyhedral-celled carcinoma of "acinous" type. *See Insp.*, 1875, No. 205.

CASE 5. — *Carcinoma of the gall-bladder. Secondary deposits in the liver. Gall-stones.*—Jane R., æt. 63, was admitted under Dr. Fagge

in August, 1876. In November of the previous year she had had "a bilious attack," and afterwards "inflammation of the lungs." Jaundice appeared in March, 1876; she had slight pain in the right hypochondrium. On admission she was much emaciated, and the skin was of a light yellow colour. The liver increased in size under observation, and nodular masses were felt in it. Death occurred eight months after admission to hospital.

Autopsy. — The gall-bladder formed a white, shrunken mass with a cancerous growth extending from it into the substance of the liver; it contained about six faceted stones. The cystic duct was puckered and narrowed; the hepatic ducts in the liver were greatly dilated. A gall-stone about the size of a nut was contained in the common bile duct below the strictured portion. The liver contained numerous secondary deposits of growth, as did also the glands in the portal fissure. *See Insp.*, 1876, No. 449.

CASE 6.—*Carcinoma of the gall-bladder. Secondary deposits in the peritoneum and pleuræ. Gall-stones.* — Amelia S., æt. 43, was admitted under Dr. Galabin with ascites of three to four months' duration and of doubtful origin. Death took place quite suddenly two and a half weeks later.

Autopsy.—The gall-bladder was much contracted, and contained one small stone, and was adherent to the duodenum; its coats were thickened with whitish growth, and Dr. Fagge writes: "I have no doubt that this had been the primary lesion." There was a large cancerous subperitoneal mass in the pouch between the uterus and the rectum, with secondary deposits also in the omentum and peritoneum elsewhere, and also in the pleuræ. *See Insp.*, 1877, No. 254.

CASE 7. — *Carcinoma of the gall-bladder. Suppuration. Gall-stones.* —Sarah W., æt. 46, was admitted under Dr. Moxon with jaundice of five months' duration. The patient had had severe pain in her right hip four months previously; this gradually extended upwards to the abdomen, and for three weeks she had had swelling of the legs. On admission the liver was found to be large and irregular, and her chief symptom was extreme abdominal pain.

Autopsy.—The liver was much enlarged by secondary deposits, and "in several parts the nodules had all run into one and rendered a large tract of the liver cancerous; this was particularly so in the neighbourhood of the gall-bladder," says Dr. Goodhart. The gall-bladder was full of thick pus, its duct as well as the common bile-duct running through a mass of cancer; three or four faceted stones were present in the gall-bladder. The pancreas and the duodenum were infiltrated by a mass of growth which had invaded them from the neighbourhood. Histologically, Dr. Goodhart describes the cells of the growth as of an epithelial type, and says he thinks that the growth was oldest in the region of the gall-bladder. *See Insp.*, 1878, No. 14.

CASE 8. — *Carcinoma of the gall-bladder. Gall-stones.* — Sarah E., æt. 58, was admitted under Dr. Hicks, and died ten days later. She had been ailing for a year; five months previous to admission the abdomen began to swell, and she suffered from nausea and vomiting. On admission there was considerable ascites; there was frequent vomiting.

Autopsy. — The gall-bladder was contracted, its surface woolly and ulcerated, and immediately around it was a large mass of growth extending into the liver in all directions. The common bile duct was distended to a size greater than that of a little finger with a number of small gall-stones compressed into one mass. There were numerous secondary deposits in the peritoneum, with many adhesions between the stomach and the intestines. *See Insp.*, 1878, No. 109.

CASE 9.—*Carcinoma of the gall-bladder. Gall-stones. Femoral hernia.* —Mary B., æt. 62, was admitted under Mr. Davies-Colley with a femoral hernia, which was operated upon. Death took place eight days later.

Autopsy.—The gall-bladder was found to be contracted around a collection of hard, faceted gall-stones; its neck was greatly indurated and thickened by an opaque, white, gristly growth which, histologically, is stated to be "a typical carcinoma." The common bile duct was dilated throughout its length. *See Insp.*, 1879, No. 204.

CASE 10. — *Carcinoma of the gall-bladder. Gall-stones. Colic.* — Eliza J., æt. 42, was under the care of Drs. Habershon and Wilks. At 14 years of age she suffered from "spasms" in the abdomen, and at 25 years of age had jaundice, which was attributed to a gall-stone. In 1878 she had another "attack of gall-stones," which lasted seven weeks and then passed off suddenly. For some months previous to admission she had noticed that her abdomen was becoming larger.

Autopsy.—The gall-bladder contained a large number of small faceted stones; it was irregularly sacculated, and its walls were infiltrated by a growth which extended into the adjacent liver substance. The common bile duct was dilated, and contained two or three faceted stones. *See Insp.*, 1879, No. 467.

CASE 11. — *Carcinoma of the gall-bladder. Gall-stones.* — Mark G., æt. 62, was admitted under Dr. Pye-Smith for bilious vomiting of two months' duration. He rapidly became thinner, vomited frequently, and on the day of his death, which took place three weeks after admission, he vomited a considerable quantity of blood.

Autopsy.—The gall-bladder was contracted and full of gall-stones closely packed, causing projections on the surface. The wall was much thickened, and from its upper surface a circular patch of "new growth" spread out into the liver. The wall of the gall-bladder was quite inseparable from the mass of growth. There were two enlarged glands in the portal fissure, and a number of small nodules in the omentum and mesentery. *See Insp.*, 1881, No. 244.

CASE 12. — *Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver, lungs, etc.* — Mary A. M., æt. 74, was admitted under Dr. Pavy for a "lump" in the right side, accompanied by pain, which she had first noticed about three months before. She was jaundiced and in much pain. The liver was enlarged and tender and there was an irregular swelling on the right side below the costal margin.

Autopsy.—The wall of the gall-bladder was infiltrated by "malignant growth," and was about half an inch in thickness; this was undergoing softening in one part. The gall-bladder and the dilated cystic duct contained 240 cubical faceted stones. There were secondary metastatic deposits of growth in the liver, the lungs, and in the bronchial and mesenteric glands. *See Insp.*, 1882, No. 191.

CASE 13.—*Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver and peritoneum.*—William W., æt. 49, was admitted under Dr. Pavy with jaundice of a week's duration. He had had an attack of colic eight weeks previously. The liver was enlarged, and there was a projection over the region of the gall-bladder. The pain diminished in intensity; the jaundice increased.

Autopsy.—The gall-bladder was embedded in a mass of cancer, its wall being much thickened and its cavity about the size of a hazel nut. It contained two very small gall-stones about the size of millet seeds. A probe was passed with difficulty along the duct which was surrounded by cancerous tissue. The liver was matted to the under surface of the diaphragm by cancerous tissue, and there were many secondary deposits in its substance. The peritoneum presented a number of secondary nodules. *See Insp.*, 1882, No. 394.

CASE 14.—*Carcinoma of the gall-bladder and duct. Gall-stones.*—Maria R., æt. 64, was admitted under Dr. Pavy with intense jaundice of six weeks' duration and pain in the right side of the abdomen. The gall-bladder, filled with gall-stones, was palpable. The liver was enlarged.

Autopsy.—The wall of the gall-bladder was infiltrated by growth, which had spread from it into the substance of the liver; the sac was dilated, and contained about 20 to 30 faceted gall-stones. There was a large calculus in the cystic duct, the size and shape of a mulberry, causing complete obstruction, and around it was a deposit of growth spreading out along each hepatic duct. *See Insp.*, 1883, No. 189.

CASE 15.—*Carcinoma of the gall-bladder. Gall-stones.*—Susan M., æt. 55, was admitted under Dr. Pye-Smith for vomiting and ascites. She was "tapped." Emaciation was extreme, and the patient only lived fourteen days after admission.

Autopsy.—The wall of the gall-bladder was formed of new growth, and its semiloculated interior contained several gall-stones. There were secondary deposits of growth in the peritoneum, the omentum, and the mesentery. *See Insp.*, 1883, No. 211.

CASE 16. — *Carcinoma of the gall-bladder. Gall-stones.* — Mary H., æt. 34, was admitted, on July 26th, under Dr. Moxon with œdema of the legs. She had been confined four months previously, and the legs had been swollen afterwards for six weeks. Three weeks before admission she had an attack of severe abdominal pain. August 1: Vomiting was frequent; the veins on the abdominal walls were distended; the patient was much emaciated. August 16: The vomiting had continued and increased in severity. The patient gradually became weaker and died three weeks after admission.

Autopsy. — The "growth" of the gall-bladder formed a large, dense, white mass. Its interior contained a number of gall-stones. The wall of the growth was half an inch in thickness at the neck of the gall-bladder; it had invaded the substance of the liver for about a quarter of an inch and was also beginning to infiltrate the substance of the pancreas. There were secondary deposits in the pleura and heart; the abdominal aorta was embedded in a mass of growth; the inferior vena cava was compressed, and the common iliac veins occupied by ante-mortem thrombus. The duodenum was narrowed by adhesions in the region of the gall-bladder. *See Insp.*, 1884, No. 281.

CASE 17. — *Carcinoma of the gall-bladder. Gall-stones. Colic.* — Esther G., æt. 50, was admitted, on August 5th, under Dr. Pavy with pain in the right side of nine months' duration. She had been in the London Hospital six months before with jaundice, and an operation was then proposed to her. On admission she had ascites as well as jaundice. There was considerable œdema of the legs. The edge of the liver was felt below the level of the umbilicus. Vomiting was a prominent symptom, and also melæna. Death took place four weeks later.

Autopsy.—The gall-bladder was greatly thickened, and of cartilaginous consistence, its wall being a quarter of an inch thick. It contained about two drachms of "creamy pus" and four gall-stones about one-third of an inch in diameter. The inflammatory thickening had quite shut off the gall-bladder from the common duct. The liver weighed 98 ounces, was smooth, and contained no secondary deposits, and the ducts were greatly dilated. The glands in the portal fissure were not enlarged. *See Insp.*, 1886, No. 300.

CASE 18. — *Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the peritoneum.*—Sarah S., æt. 59, was admitted under Mr. Bryant for chronic intestinal obstruction of nine weeks' duration for which lumbar colotomy was performed. The patient died two days afterwards.

Autopsy.—The gall-bladder was greatly thickened, its walls a quarter of an inch thick, its cavity filled with "scores of small faceted gall-stones." The omentum was greatly thickened, and in it and the mesentery were many masses of firm white fibroid material thought to be of malignant type. The mesenteric glands were large, hard, and white. *See Insp.*, 1887, No. 129.

CASE 19.—*Carcinoma of the gall-bladder. Gall-stones.*—Fred. K., æt. 77, was admitted under Dr. Pye-Smith in a semi-comatose state following paralysis of his left arm of sudden onset a week before.

Autopsy.—The gall-bladder was a scarred fibrous sac containing a number of stones, over 200, of various sizes. In its wall was a nodule of growth, the size of a marble, extending into the liver. Histological examination showed the tumour to be composed of a reticulated stroma with large deeply staining cells, and it was regarded as a carcinoma. There were secondary deposits in the glands of the mesentery and in the visceral peritoneum. *See Insp.*, 1889, No. 281.

CASE 20.—*Carcinoma of the gall-bladder. Gall-stones. Pus in the gall-bladder. The duodenum constricted.*—George A., æt. 58, was admitted under Dr. Hale White with jaundice and an enlarged liver. There was no history of colic. The wasting continued; the jaundice became more intense, and the patient died four months later.

Autopsy.—The gall-bladder was distended to the size of a pigeon's egg. It contained three faceted calculi, each of the size of a hazel nut. A deposit of growth around its neck extended for about half an inch into the liver. The fibroid growth extended along behind the duodenum obstructing both the cystic and hepatic ducts, and also narrowing the duodenum. Histologically it presented the structure of a columnar-celled carcinoma with abundant fibrous stroma. There were several secondary deposits in the mesentery. The duodenum was constricted at a distance of two inches from the pylorus, so that its lumen would only admit the middle finger with difficulty. *See Insp.*, 1889, No. 366.

CASE 21.—*Carcinoma of the gall-bladder. Gall-stones. Ascending suppurative cholangitis* (*? pylephlebitis*).—Susan B., æt. 40, was admitted under Dr. Taylor, on August 31st, with abdominal pain and jaundice. The pain commenced two months previously, vomiting began a fortnight, and jaundice ten days before admission. On admission the abdomen was somewhat distended, and there was some increase of the area of hepatic dulness, with fulness and tenderness in the epigastrium and opposite the ninth costal cartilage. The patient became gradually weaker, vomiting was frequent, the temperature was irregular and usually raised.

Autopsy.—The gall-bladder region was occupied by a mass of growth of firm consistence, the size of an orange. Its inner surface was irregular, in diameter about an inch and a half, and it contained numerous irregular gall-stones and a thick reddish material similar to that in the liver. The growth invaded the adjacent liver tissue and the left branch of the portal vein. The liver was considerably enlarged, its tissue was soft, and on section a thick reddish material escaped from many places; it looked like pus and broken down blood clot, and it was doubtful if it came from the portal vein or from the hepatic ducts. There were several small cavities filled with the same grumous pus in the liver in places. *See Insp.*, 1890, No. 373.

CASE 22.—***Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver.***—Sally C., æt. 65, was admitted, on August 10th, under Dr. Pavy with jaundice and pain in the right side. For the previous two years she had complained of loss of appetite, and for three months before admission she felt ill and suffered with "aches and pains" in the abdomen. Six weeks before admission the pain was worse on the right side, and two weeks before jaundice appeared. On admission a large hard mass was felt on the right side extending halfway between the costal margin and the anterior superior spine of the ilium. Death took place about three weeks later, patient being in a state of cholæmia.

Autopsy.—The wall of the gall-bladder was found to be infiltrated with growth which invaded the adjoining part of the liver. There were several gall-stones, two of large size, in its interior. Secondary deposits were present in the liver and on the under surface of the diaphragm. Histologically the growth proved to be a spheroidal-celled carcinoma. *See Insp.*, 1892, No. 303.

CASE 23.—***Carcinoma of the gall-bladder. Gall-stones. Empyema of gall-bladder.***—Fanny P., æt. 37, was admitted, on September 14th, under Mr. Lane with a tumour in the right hypochondrium reaching nearly to the iliac fossa. She had suffered from occasional attacks of jaundice. An exploratory laparotomy was performed, and secondary deposits were found in the liver.

Autopsy.—The gall-bladder was surrounded by a mass of growth as large as a fist; its interior contained pus and numerous faceted gall-stones. The growth had fungated through into the duodenum, and there were secondary deposits in the liver and in the mesenteric glands. *See Insp.*, 1893, No. 411.

CASE 24.—***Carcinoma of the gall-bladder. Gall-stones. Abscesses in the liver.***—William H., æt. 64, was admitted under Dr. Goodhart, on January 4th, with jaundice which had appeared in October, 1894. In November, 1894, Dr. Goodhart "felt lumps in the liver which have since cleared up." He had suffered a good deal with vomiting, and had wasted considerably. The liver was hard and enlarged. The temperature was raised in the evening, generally to about 101°—102°. Later the patient became drowsy, the feet began to swell, and death took place on January 28th. The condition was thought to point to a primary growth in the stomach.

Autopsy.—In the region of the gall-bladder was found a tumour as large as a cricket ball, adherent to and invading the liver. This tumour was the wall of the gall-bladder, and was composed of a soft, breaking-down growth with some semi-purulent contents. In the common duct were three gall-stones. The liver was of a dark green colour and adherent to the diaphragm. There was an abscess between the liver and the diaphragm holding about two ounces of greenish pus, and there were many abscesses in the liver. One of the larger branches of the right

pulmonary artery was filled with ante-mortem thrombus. Histologically the growth is reported to be a short columnar-celled carcinoma of scirrhous type. *See Insp.*, 1895, No. 30.

CASE 25.—*Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the peritoneum, glands, etc.*—Rebecca H., æt. 43, was admitted, on June 14th, under Dr. Taylor for wasting of two months' duration. She became gradually weaker, and died about a month after admission.

Autopsy.—The gall-bladder was found to be small and to be contracted in its proximal portion around three faceted stones; the distal half was infiltrated by growth, its wall being half an inch thick and its lumen a mere slit. The gall-bladder was adherent to the transverse colon. There were many small secondary deposits in the peritoneum, and on the surface, and a few in the substance of the liver, and also in the glands alongside the aorta. The liver was small, weighing only 30 ounces. The coils of intestine were more or less matted together. Histologically the growth of the gall-bladder is described as a scirrhous glandular carcinoma, the cells varying in shape, but for the most part elongated. *See Insp.*, 1895, No. 253.

CASE 26.—*Carcinoma of the gall-bladder. Gall-stones.*—Mary R., æt. 56, was admitted under Mr. Dunn with an ovarian tumour. When the abdomen was opened some early peritonitis was found to be present. The cyst burst during the operation; peritonitis supervened, and the patient died on March 26th. There was no history of any biliary colic.

Autopsy.—A mass of white growth measuring two and a half inches transversely and extending backwards about four inches into the liver substance was found occupying the site of the gall-bladder, which itself was packed with small faceted stones. There were numerous secondary deposits in the liver. Histologically the growth is described as "a spheroidal-celled carcinoma, some of the alveoli being lined with cylindrical epithelium, much stroma; alveoli medium sized; rest of liver fatty." The common bile duct appeared healthy, and was not dilated. *See Insp.*, 1896, No. 101.

CASE 27.—*Carcinoma of the gall-bladder. Gall-stones. Exploratory laparotomy.*—Harriet M., æt. 57, was admitted under Mr. Howse, on May 6th. A year previously she had had an attack of shooting pain of short duration in the region of the liver. In January, 1896, she had a similar attack followed, on this occasion, by jaundice which had remained. The patient stated that she had lost two to three stone in weight. On admission she was deeply jaundiced. A globular mass was felt in the region of the gall-bladder. When the abdomen was explored, on May 19th, a large, hard, cancerous mass was found with secondary deposits in the liver and elsewhere. Death ensued ten days later.

Autopsy.—The gall-bladder wall was found to be thickened, ulcerated, and infiltrated with carcinomatous deposit. The growth extended into the liver, and in the centre of the mass were a number of gall-stones. The growth was adherent to, but did not obstruct, the duodenum, and there were secondary deposits in the liver and along the lesser curvature of the stomach. *See Insp.*, 1896, No. 197.

CASE 28.—*Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver, etc. Colic.*—William K., æt. 55, was admitted, on September 2nd, under Dr. Taylor with jaundice. Two months previously he had been taken ill while at work and vomited for about six hours. He had severe epigastric pain, followed the next day by jaundice and clay-coloured stools. On admission he was deeply jaundiced, and the hard edge of the liver could be felt with two hard nodules in its substance. He became drowsy and more jaundiced, and died in a semi-comatose state eight days after admission.

Autopsy.—A large hard mass of growth was found connected with the gall-bladder; it was about the size of an egg, and in its interior were a number of minute stones and thick mucus. The cystic duct was dilated and full of calculi, and so also was the common bile duct. The growth invaded the adjacent liver tissue, and there were many secondary deposits in the liver and elsewhere. *See Insp.*, 1896, No. 376.

CASE 29.—*Carcinoma of the gall-bladder. Gall-stones.*—Susannah A., æt. 65, was admitted, on February 18th, under Dr. Washbourn for abdominal pain. Her illness commenced at Christmas, 1896, with pain in the right hypochondriac region. There was a hard nodular enlargement of the liver. The patient was much wasted and died a few days later.

Autopsy.—The gall-bladder was found to be about as large as a cricket ball, and on section it contained two stones about the size of large peas with an innumerable collection of black rounded grains. The growth invaded the liver by continuity, and there were secondary deposits in the liver, the peritoneum, and in the adhesions between the liver and the diaphragm. *See Insp.*, 1897, No. 78.

CASE 30.—*Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver and glands.*—Sarah F., æt. 63, was admitted under Dr. Goodhart, on October 6th, with swelling of the legs and abdomen and pain in the right side which she is said to have had for fourteen years. The patient remained in much the same condition until October 16th, when she developed diphtheria and died a few days later.

Autopsy.—The gall-bladder was found to be shrunken, and its wall infiltrated with growth which extended into the liver and to the head of the pancreas. Its cavity was full of small stones. There were secondary deposits in the liver and in the glands in the portal fissure. *See Insp.*, 1897, No. 414.

CASE 31. — ***Carcinoma of the gall-bladder. Gall-stones. Cholecysto-duodenal fistula.*** — Mary B., æt. 39, was admitted under Dr. Taylor for abdominal pain which she first noticed in March, 1897. In June a swelling appeared beneath the ribs on the right side, and the patient became jaundiced. Three weeks before admission she had diarrhœa and melæna. At eighteen years of age the patient had suffered with gastric pains and vomited coffee-ground material, and at twenty-five years of age had had an attack of hæmatemesis. On admission a swelling was detected in the epigastrium and a small nodule on the edge of the liver. On December 8th, 1907, patient had some rigors, and on December 20th she was jaundiced. On December 29th the abdomen was explored and a malignant growth found in the region of the gall-bladder.

Autopsy.—The wall of the gall-bladder was found to be much thickened by a new growth which had spread to a small degree into the adjacent liver tissue. There were a few cholesterin calculi in the interior of the gall-bladder, and many in the hepatic, cystic, and common bile ducts. In its lower part the growth involved the duodenum, and there was a fistulous opening between them. The stomach was greatly dilated, the first part of the duodenum was firmly adherent to the gall-bladder, causing a sharp kink, apparently one of the main factors in the dilatation of the stomach. There was also an old ulcer of the duodenum one and a half inches below the pylorus. Histologically the growth is stated to be a cylindrical epithelioma. *See Insp.*, 1898, No. 8.

CASE 32. — ***Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver and glands. Exploratory laparotomy.***—Catharine B., æt. 44, was admitted under Mr. Lane for a tumour in the right hypochondrium which had been noticed a month previously, and had rapidly increased in size. Three days before admission she became jaundiced. Exploratory laparotomy proved the mass to be a malignant growth of the gall-bladder.

Autopsy.—A large mass of growth, the size of a cricket ball, projected from the liver in the region of the gall-bladder, the wall of which was quite replaced by the growth. There were several small dark grey faceted stones in the gall-bladder and in the ducts. The growth had extended along the cystic and the common bile ducts, and had occluded the pancreatic duct so that the pancreas itself had undergone cystic degeneration. There were many large secondary deposits in the liver and in the abdominal glands. Histologically the growth is said to have the structure of a cylindrical-celled carcinoma. *See Insp.*, 1898, No. 116.

CASE 33.—***Carcinoma of the gall-bladder. Gall-stones.***—Mary Ann A., æt. 59, was admitted under Dr. Hale White with jaundice, abdominal pain, and vomiting. Seven weeks previously the jaundice appeared, she lost her appetite, and experienced considerable pain at the lower part of the back with vomiting after food. The liver was much enlarged, and a

tender mass was felt two and a half inches above the umbilicus. The patient became gradually weaker, dying three weeks after admission.

Autopsy.—The gall-bladder was found to be thickened and firmly contracted around a large gall-stone measuring one inch by one and a half inches. The common bile duct contained many white faceted calculi. The commencement of the common bile duct, the cystic duct, and the neck of the gall-bladder were thickened and the lumen narrowed by a growth which histologically is described as being a glandular carcinoma. Dr. Bryant says, "the primary seat of the growth was the cystic duct," but it is not clear that this was the case from the description of the autopsy. It might quite well have been the gall-bladder, of which no microscopical section appears to have been made. There were secondary deposits in the liver and in the glands. *See Insp.*, 1898, No. 205.

CASE 34.—*Carcinoma of the gall-bladder. Gall-stones. Exploratory laparotomy. Secondary deposits in the liver. Empyema of the gall-bladder.*—T. N., a female æt. 49, was admitted, on July 27th, under Dr. Pitt with abdominal pain and jaundice. For about the past two or three months she had suffered from attacks of pain in the left side two or three times a week. Later a doctor examined her and found a swelling on the right side of the abdomen. The jaundice appeared in July. On admission a hard, deeply-seated tumour was felt at the lower edge of the liver which itself reached four and a quarter inches below the costal margin. An exploratory laparotomy was performed and the tumour found to be malignant.

Autopsy.—The gall-bladder was much distended, projecting out from under the liver. The wall was much thickened, and in its interior was a very large egg-shaped calculus with several smaller ones, and also a thick, yellowish, muco-purulent fluid. The cystic duct and the upper half of the common duct were blocked by calculi, and they, with the ducts in the liver, were greatly dilated. In the liver were many secondary deposits, the largest, about two inches in diameter, being in the immediate neighbourhood of the gall-bladder as if it had invaded the viscus from that region. Histologically the growth presented the structure of a columnar-celled carcinoma. *See Insp.*, 1899, No. 280.

CASE 35.—*Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the peritoneum. Chronic intestinal obstruction. Laparotomy.*—Ellen D., æt. 70, was admitted under Dr. Taylor, on March 26th, for chronic intestinal obstruction. She had suffered with difficulty in getting the bowels relieved since the previous October; fourteen days before admission the obstruction became more marked, and vomiting commenced four days previously. The abdomen was opened and a "short circuit" arranged between the small intestine and the transverse colon. Death took place five days after admission.

Autopsy.—Secondary deposits of growth were found in the peritoneum, a stricture of the gut having formed as the result of a local peritonitis. The gall-bladder was infiltrated by growth and contained about twenty-six calculi. On histological examination the growth was stated to be a carcinoma. *See Insp.*, 1900, No. 126.

CASE 36.—*Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver. Empyema of the gall-bladder. Suppurative cholangitis. Abscess in the liver.*—Eliza I., æt. 57, was admitted under Dr. Pitt, on April 27th, with jaundice and loss of flesh. Early in this year she became jaundiced, and passed a gall-stone, and afterwards she passed several calculi, and the jaundice remained. On admission she complained of pain over the region of the gall-bladder. The urine was albuminous, and contained casts and epithelial cells. On May 27th she was nearly suffocated as the result of "swallowing her teeth," which became impacted in the larynx. Shortly after this she became very noisy and weak. She died about five weeks after admission.

Autopsy.—The gall-bladder "was found to be much thickened by what appeared to be a malignant growth." It was tightly packed with faceted calculi of various sizes, and there was a quantity of greenish pus present. In the liver there were several small abscesses and also nodules of growth in both lobes. The hepatic ducts were dilated and filled with pus. There was a fistulous opening between the commencement of the common bile duct and the duodenum, "no doubt caused by a stone ulcerating through." *See Insp.*, 1901, No. 176.

CASE 37.—*Carcinoma of the gall-bladder. Gall-stones in the common bile duct.*—William W., æt. 68, was admitted under Dr. Hale White, on October 31st, 1901, with jaundice, abdominal pain, and vomiting. The symptoms first appeared in July when he began to feel ill and to lose his appetite. He became jaundiced in August, but he had no pain. On admission he was in a drowsy condition, and the jaundice persisted. The liver was enlarged, and an irregular mass was felt in it. Later, hæmorrhages appeared on the hands, the feet, and the back, and he gradually became weaker and died on January 17th, 1902.

Autopsy.—The gall-bladder was found contracted up into a firm, fibrous mass with growth extending from it directly into the liver. The inner surface was irregular and trabeculated. Two centimetres above the opening of the common bile duct into the duodenum a gall-stone was impacted, and above it were two or three smaller stones. The common bile duct was much dilated down to the place at which the stone was impacted. On histological examination the growth was found to be a short columnar or cubical-celled carcinoma with a very fibrous matrix. The ducts in the liver were dilated and filled with greenish mucoid fluid. *See Insp.*, 1902, No. 33.

CASE 38. — *Carcinoma of the gall-bladder. Gall-stones.* — Martha C., æt. (?), was admitted under Mr. Lucas, on March 17th, with constant pain in the abdomen which had commenced in December, 1901, and in March, 1902, was accompanied by sickness. On admission she was in great pain and was frequently sick. The abdomen was opened and a growth was found and partly removed. In May the sickness and also the pain returned, and death took place in June.

Autopsy.—The wall of the gall-bladder was found to be very thick and to be infiltrated with growth. The sac was full of large faceted calculi. There were secondary deposits of growth in neighbouring lymph glands. On histological examination the growth was found to be a columnar-celled carcinoma. There was a general suppurative peritonitis, a portion of the gut having given way at the place where the growth had been partially removed. *See Insp.*, 1902, No. 242.

CASE 39. — *Carcinoma of the gall-bladder. Gall-stones. Cholecysto-duodenal fistula.* — Richard C., æt. 66, was admitted, on July 8th, under Dr. Hale White for pain in the right side of the abdomen of two months' duration. On admission a large, hard mass was felt in the right hypochondrium extending into the umbilical and right iliac regions. The patient suffered much from vomiting; he became very emaciated and had considerable pain. Death took place a month after admission.

Autopsy.—A mass of very soft necrotic growth, eight inches in diameter, was found entirely replacing the gall-bladder, and in its interior were nine faceted calculi. There was a fistulous opening between the growth and the duodenum, and the growth extended into the liver by continuity. On histological examination the growth was reported to be a spheroidal-celled carcinoma. *See Insp.*, 1902, No. 347.

CASE 40. — *Carcinoma of the gall-bladder. Gall-stones in the common bile duct. Chronic intestinal obstruction. Cholecysto-colic fistula.*—Harriet P., æt. 52, was admitted under Dr. Perry, on July 23rd, for abdominal pain and vomiting which had commenced in November, 1901, and had been more severe for the past six weeks. The abdomen was opened on August 5th and a growth found to be obstructing the hepatic flexure of the colon. Death took place on September 16th.

Autopsy.—The gall-bladder was much thickened and infiltrated by growth which had extended into, constricted, and kinked the transverse colon. The liver was also invaded. In the common bile duct were three or four calculi, one of which was fixed in the duct, at its lower end. There were some smaller stones in the hepatic ducts which were everywhere dilated. The glands in the portal fissure contained secondary deposits, and the portal vein was thrombosed. Histologically the growth presented the structure of a spheroidal-celled carcinoma. *See Insp.*, 1902, No. 417.

CASE 41. — ***Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver and peritoneum. Constriction of the duodenum. Exploratory laparotomy.***—Henrietta F., æt. 50, was admitted under Dr. Taylor, on July 10th, with jaundice of three weeks' duration. She had been in ill-health since October, 1902, with pain in the shoulder and flanks and later with attacks of vomiting. On admission a rounded mass was felt at the outer margin of the right rectus muscle. While she was in hospital the attacks of vomiting and the abdominal pain were frequent. The jaundice became more marked and the pain continuous. On August 12th an exploratory operation was performed, and the mass was found to be malignant. The patient gradually became weaker and died on October 27th.

Autopsy.—The wall of the gall-bladder was found to be two or three times the usual thickness, and it was irregularly infiltrated with growth. It had shrunken up under the liver and in so doing had pulled up the pyloric extremity of the stomach and the duodenum. In its interior were nineteen pigment and cholesterin stones. There were secondary deposits in the liver, the peritoneum, and the kidney. The duodenum just beyond the pylorus was constricted by adhesions. On histological examination the growth was found to be a spheroidal-celled carcinoma. *See Insp.*, 1903, No. 406.

CASE 42. — ***Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver and glands. Operation.***—Lavinia P., æt. 51, was admitted under Mr. Lucas, on March 7th, with jaundice and a tumour in the umbilical region. In November, 1903, the patient had had what is described as a "severe attack of indigestion" with vomiting after food. For the next few weeks she was fed on peptonised milk and the sickness ceased. A tumour was first noticed in the epigastrium about this time and jaundice appeared. The patient rapidly lost weight. On admission, in addition to the features above recorded, the liver edge was felt one and a half inches below the costal margin. Laparotomy was performed and the edges of the gall-bladder were stitched to the margins of the abdominal incision. Some 280 calculi were removed, and during the next three weeks about 40 more. The jaundice was not relieved by the operation, and the patient died a month after admission in a cholæmic state.

Autopsy.—The wall of the gall-bladder was found to be infiltrated by growth, and there was a recent acute cholecystitis. The common bile duct was dilated, and just above the duodenal orifice was a small mass of soft white growth which apparently blocked the duct. Above it were two or three small stones; the inner surface of the duct was ulcerated. On histological examination the growth was found to be a carcinoma. The liver was much enlarged by secondary deposits, and in the region of the gall-bladder it was directly invaded by the primary growth of that sac. There were secondary deposits in the glands along the aorta. The body was deeply jaundiced, and there was a recent acute peritonitis in the upper part of the abdomen. *See Insp.*, 1904, No. 184.

CASE 43. — ***Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the peritoneum, pleuræ, liver, and glands.***—Anne A., æt. 59. was admitted under Mr. Targett, on November 21st, for a tumour in the abdomen first noticed in July, 1904, and also for vomiting which had become severe about a week previously. The abdominal cavity was opened and a quantity of fluid escaped. (There are no further notes of the case in the report.) Death took place a week after admission.

Autopsy.—The main "mass of growth was in direct contact with the gall-bladder and appeared to follow the course of the bile passages." There were two calculi in the bladder which itself was somewhat dilated. The liver contained a large number of secondary deposits; there were small nodules scattered over both visceral and parietal peritoneum and over both pleuræ; the glands alongside the aorta were enlarged by growth. Histologically the tumour proved to be a columnar-celled carcinoma. *See Insp.*, 1904, No. 545.

CASE 44. — ***Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver and pleura. Pleural effusion.***—John C. (age not stated), was admitted under Mr. Golding-Bird, on September 5th, with a tumour in the right lumbar region which was first detected in the previous January. There was marked loss of weight, said to be five stone. An exploratory operation was performed and a large mass of growth was found involving the gall-bladder. Death occurred 10 days after admission.

Autopsy.—The wall of the gall-bladder was found to be much thickened and on the under side of the liver was a mass of growth nine inches in length with gall-stones in its centre. Histologically the structure of the growth was found to be a spheroidal-celled carcinoma. There was a large deposit of growth in the right lobe of the liver, and the pancreas was involved in an extension of the primary growth. The surface of the pleuræ was studded with secondary deposits, and there were sixty ounces of blood-stained fluid in the left pleural cavity. *See Insp.*, 1904, No. 445.

CASE 45. — ***Carcinoma of the gall-bladder. Gall-stones. Secondary deposits in the liver, glands, and peritoneum. Chronic intestinal obstruction. Laparotomy.***—Josephine B., æt. 49, was admitted under Mr. Golding-Bird for a tumour in the left iliac region and wasting. In November, 1904, she had had a severe attack of pain in the left side and back, and lately symptoms of obstruction of the bowels developed. On February 2nd laparotomy was performed, and the colon and sigmoid flexure were found to be tightly bound down in the pelvis and left lumbar region.

Autopsy.—The gall-bladder was found to be contracted up under the right lobe of the liver and its wall was thickened by growth; its interior contained a large oval stone with a number of small ones. Histologically the structure of the growth was a cubical-celled carcinoma. In the adjacent part of the liver was a deposit having the diameter of

a half-crown piece. The glands alongside the abdominal aorta were enlarged by secondary deposits and extending out from these into the pelvis was a layer of growth. The colon and sigmoid flexure were not only adherent to this, but were narrowed by the extension of the neoplastic tissue around them. *See Insp.*, 1905, No. 64.

CASE 46.—*Carcinoma of the gall-bladder. Gall-stones. Empyema of the gall-bladder. Suppurative cholangitis. Secondary deposits in the liver and glands.*—John L., æt. 49, was admitted under Mr. Symonds with the history that he had been suffering from abdominal pain and attacks of shivering for seven weeks and that latterly he had developed jaundice. On admission the patient had a temperature of 104·6°. There was a tumour projecting below the right costal margin; this was incised and pus and gall-stones were set free; the wall of the gall-bladder was found embedded in growth. The patient died on February 16th.

Autopsy.—All the tissues were seen to be jaundiced. An abscess cavity extended for a distance of about five inches from the abdominal wound into the substance of the liver. The gall-bladder could not be recognised, but there were gall-stones in the abscess cavity. There were secondary deposits of growth in the liver and in the glands in the portal fissure. Suppurative cholangitis was present. No microscopical examination of the growth is reported. *See Insp.*, 1905, No. 84.

CASE 47.—*Carcinoma of the gall-bladder. Gall-stones. Operation.*—Jane A., æt. 56, was admitted under Mr. Symonds with the history that she had been jaundiced from 1901 to March, 1903, when a gall-stone was removed from the gall-bladder. The symptoms recurred in April, 1904, and in January, 1905, Mr. Symonds removed the gall-bladder with some "growth" around it; a part of the bile duct was also resected. The patient gradually became weaker and died on March 1st.

Autopsy.—A soft growth was found in the common bile duct. The duct was dilated, as were also the ducts in the liver. The growth on histological examination was found to have the structure of a spheroidal-celled carcinoma. *See Insp.*, 1905, No. 116.

CASE 48.—*Carcinoma of the gall-bladder. Gall-stones in the common bile duct. Secondary deposits in the liver and glands. Empyema of the gall-bladder. Ascending suppurative cholangitis.*—Emily M., æt. 48, was admitted, on June 8th, under Dr. Fawcett for intense and increasing jaundice of a week's duration. She had been well until April when she had "a bilious attack." With the onset of the jaundice she had several attacks of abdominal pain and vomiting. The jaundice increased and was accompanied by pyrexia, and the patient wasted rapidly. The liver was enlarged upwards to the level of the third right intercostal space. Death occurred two days after admission.

Autopsy.—The gall-bladder was found to contain about one and a half ounces of creamy pus. Its wall was infiltrated throughout with growth, and the cystic duct was blocked by growth, as was also the common bile duct, and in the lumen of the latter were two tiny calculi bathed in thick pus. The liver was much enlarged, having many small nodules of growth scattered through it, and its ducts were dilated and filled with pus. The gastro-hepatic omentum was infiltrated and thickened by growth, and many of the lumbar glands contained secondary deposits. Histologically the growth was a cylindrical-celled carcinoma. *See Insp.*, 1905, No. 286.

NOTES FROM THE EAR AND THROAT DEPARTMENT.

ENUCLEATION OF THE TONSIL.

By

W. M. MOLLISON AND A. M. ZAMORA.

FIFTY-SIX cases of enucleation of the tonsil during the year 1912 were collected for investigation mainly with a view to deciding whether this operation gave good results in cases of otorrhœa, but while this was the main point of interest, other points presented themselves in the course of the investigation. Of the 56 cases collected, only 35 came up for re-examination in response to requests sent by post.

The technique of the operation was the same in all cases, and may be described briefly as follows: The tonsil is grasped with Tilley's forceps and the mucous membrane cut through with blunt-nosed scissors at that point where it passes on to the tonsil from the anterior pillar of the fauces. It is now possible to push the anterior pillar from the tonsil, enter the peritonsillar space and shell the tonsil out from its bed either with scissors or with the finger. When the tonsil is attached only by its inferior prolongation, a blunt Mackenzie's guillotine is slipped over the forceps and this inferior prolongation divided. The patient lies on his back with the head turned slightly to the right, and the operator stands on the right side. The right tonsil is enucleated first, in order that the blood shall not obscure the view during the removal of the second tonsil.

It is not proposed to discuss the relative merits of the various methods now employed to remove the tonsils. Doubtless the efficiency of removal by the blunt guillotine increases materially with practice, but however efficient recently-devised guillotines may be, there will always be some tonsils which will defy removal by these means, and it is these cases which require an enucleation operation. Though it is possible that some of the cases considered could have been done by the blunt guillotine method, the great majority were chosen on account of their difficulty.

The complications arising from the operation during the whole series of cases amounted to one case of reactionary hæmorrhage and one of rather severe pharyngitis. The operation is one which needs considerable practice before the best anatomical results are obtained consistently, and it was noticed in this respect that there was definite improvement with experience. The left lower pole is the part most frequently missed, and it is of great importance to make the removal complete.

Appended is a tabulated list of cases.

With regard to the effect of enucleation on otorrhœa, 21 cases gave the following figures:—Cure, 7; Improvement, 3; No Change, 11.

The percentage of complete cures, then, is not great, but if it be taken into account that the class of patient is highly unfavourable, and that any aural discharge is frequently neglected, it may be said that the results are not unpromising.

A comparison with the series of cases of operation on the mastoid antrum, radical and conservative, for the relief of otorrhœa collected from the same source by Mr. V. Glendining* shows the percentage of cures to be higher in these mastoid operations.

Mastoid Operations (42 Cases): Cures, 23; Improvement, 8; No change, 11. Percentage of cures, 54·8.

Enucleation (21 Cases): Cures, 7; Improvement, 3; No Change, 11. Percentage of cures, 30·4.

* "Guy's Hospital Reports," vol. lxvi.

Name.	Date of Operation.	Operation.	Previous Symptoms.	Present Condition.	Condition of Throat.	Remarks.
Rose F. ...	Mar., 1911	Enucleation	Constant sore throats...	Entirely relieved ...	Slight remains left lower pole and right upper pole	—
Arthur S. ...	Sept., 1911	Enucleation	Left otorrhœa, occasional Sore throats Deafness	Worse in every way	Slight left lower pole remains	Voice "rougher" after operation.
George H. ...	Sept., 1911	Enucleation + Adenoids	Double otorrhœa, slight Sore throats ... ... Deafness	Cured Cured Cured General health greatly improved	Clear	Voice markedly improved— "louder and clearer"
Frank N. ...	Dec., 1911	Enucleation	Left otorrhœa ... ... Lymphadenitis .. Dyspnœa ... ... Deafness	Cleared up at once Improved Disappeared Improved	Right posterior pillar scarred	Definite improvement in voice from "rough" to "clear"
Lily P. ...	Dec., 1911	Enucleation	Sore throats Deafness Vertigo and tinnitus	No change at all. Now has cervical adenitis, and has had one attack of quinsy since operation	Left lower pole remains (slight)	Pharyngitis after operation. Voice "goes" when throat is inflamed
John S. ...	—	Enucleation	Chronic otorrhœa ...	*In statu quo*	—	Radical subsequently
Frances P....	—	Enucleation	Tuberculous glands in neck. Glands previously operated upon	Developed phthisis	—	—

Name.	Date of Operation.	Operation.	Previous Symptoms.	Present Condition.	Condition of Throat.	Remarks.
Nurse E. ...	Feb., 1912	Enucleation	Sore throats and quinsy	Occasional trouble on left side	Left side slight remains	—
			Chronic catarrhal deafness	Better		
			History of rheumatic gout with swelling of a finger one month before operation	No sign of rheumatism now		
Ada B. ...	Feb., 1912	Enucleation	Double otorrhœa for years	Right side, constant otorrhœa. Left side dry on examination	Left side slight remains	—
			Sore throats	Cured		
Elsie G. ...	April, 1912	Enucleation	Sore throats	Cured	—	—
			Deafness...	Same as before		
			Colds	Lessened		
Winifred H.	Mar., 1912	Enucleation	Right-sided otorrhœa of long standing. Left side used to discharge, but had become dry	Right side ceased shortly after operation	—	—
			Sore throats	Cured		
			Deafness...	Worse		
Alfred S. ...	April, 1912	Enucleat. L Guillotine R	Otorrhœa (long standing)	Unaffected	Right side remains. Scarring of left anterior pillar. No functional effects	—
			Sore throats	Cured		

Name.	Date of Operation.	Operation.	Previous Symptoms.	Present Condition.	Condition of Throat.	Remarks.
Nurse B. ...	—	Enucleation	Sore throats ... ... Neurosis	Improved. Throat still gets sore, but there is no septic matter	—	Patient also had moveable kidney. (Wrote)
Roy S. ...	April, 1912	Enucleation	Sore throats ... ... Pale and ailing ... Right-sided total unilateral deafness (probably congenital)	Cured Now fat and well Unaffected	Left lower pole remains	—
Maud H. ...	Feb., 1912	Enucleation	Left otorrhœa persisting after radical operation	Unaffected by Enucleation	—	Subsequent second operation on ear
William G...	Jan., 1912	Enucleation	Frequent sore throats	Cured	Small portion of left remains. Subsequently removed under gas	—
John B. ...	Jan., 1912	Enucleation	Chronic otorrhœa ... Chronic tonsillitis ... Deafness	Stopped almost directly after operation Cured Cured	Left lower pole remains	—
Percy P. ...	May, 1912	Enucleation	Otorrhœa ... ... Deafness	Cured Nearly well	—	Wrote
Herbert H.	May, 1912	Enucleation	Otorrhœa (years) ... Deafness	Cured Unaffected	—	—

Name.	Date of Operation.	Operation.	Previous Symptoms.	Present Condition.	Condition of Throat.	Remarks.
Daisy H. ...	Sept., 1912	Enucleation + Removal of aural polyp., left side	Left otorrhœa (years) ... Sore throats Polypi removed two or three times from left ear	Perforation only just moist, though she has had no treatment for it. Sore throats better	Left anterior pillar scarred. Right side small remains	—
Jack G. ...	Sept., 1912	Enucleation + Inferior turbinectomy	Otorrhœa 6 years ...	Left ear still moist. Throat well	—	—
Ernest O. ...	Nov., 1912	Enucleation + Adenoids	Otorrhœa ... ... Sore throats Deafness	Unaffected Improved. General health improved	—	Wrote
Edith B. ...	July, 1912	Enucleation + Adenoids	Otorrhœa persisting after double radical	Unaffected Throat well	—	—
May B. ...	Aug., 1912	Enucleation	Frequent sore throats ... Otosclerosis Deafness	Cured Improved	—	—
Clara H. ...	Sept., 1912	?	Chronic otorrhœa ...	Unaffected	Remains (extensive) on both sides	Probably enucleation was not performed
Herbert S....	Sept., 1912	Enucleation	Frequent sore throats .. Nasal obstruction ...	Cured Still had nasal catarrh	—	—

Name.	Date of Operation.	Operation.	Previous Symptoms.	Present Condition.	Condition of Throat.	Remarks.
William V.	Sept., 1912	Enucleation	Sore throats ... ... Rhinorrhœa ... ... Bad general health ...	Cured Unaffected Much improved	—	Septal deflection present. ? Accounting for rhinorrhœa
Nellie C. ...	Dec., 1912	Enucleation	Left-sided chronic otorrhœa	Unaffected	—	Subsequent radical mastoid operation. Granulations in antrum of greenish colour
William G...	Dec., 1912	Enucleation	Frequent sore throats... Hoarseness ...	Cured Cured	—	—
Charles W.	Sept., 1912	Enucleation	Left chronic otorrhœa, on account of which he was refused as a teacher by the Education Office	Cured Later accepted	—	—
Nurse S. ...	July, 1912	Enucleation	Sore throats	Cured	Left lower pole remains. Removed later under gas	—
Richard C....	Oct., 1912	Enucleation	Chronic otorrhœa for years	Unaffected	—	—
Matilda G....	May, 1912	Enucleation	Chronic otorrhœa ...	Unaffected	—	—

If, however, it is taken into account that the cases of mastoid operations were examined only a short time after discharge from the hospital, and if allowance is made for the large number of late recurrences which develop in these cases, it will be seen that the comparison is not unfavourable.

It is the object of this paper to emphasise the importance of enucleation of the tonsil as a curative measure for otorrhœa, and to point out that in view of the relative severity of the two operations and the difficulty of after-treatment of the mastoid cavity in hospital patients, the tonsil operation should be performed in every case where there is the slightest suspicion of the infection coming from the pharynx, before having recourse to the radical mastoid operation.

When the operation was performed for sore throats or hoarseness the results were uniformly good. Out of 21 cases only 2 bad results are recorded, and in both of these the enucleation was incomplete. There is no doubt that in certain cases the operation has a marked effect on the voice, but the class of patient treated was not favourable for this investigation, though the small amount of evidence points to a beneficial effect.

FOUR CASES OF ACUTE SUPPURATION OF THE FRONTAL SINUS OR ETHMOIDAL CELLS WHICH CAUSED SUPPURATION IN THE ORBIT

By

W. M. MOLLISON.

The cases are of some interest in that they presented themselves in the first place on account of symptoms affecting the eye; two presented sinuses in the upper eyelid, one was a case of orbital cellulitis, and one presented an abscess at the upper and inner angle of the orbit.

That all four cases occurred within four months emphasises the frequency with which suppuration in the frontal sinus or ethmoid invades the orbit. In view of the anatomical relations of the parts concerned this frequency is scarcely astonishing.

Case 1.—James C., aged 17, was admitted to Barnabas under Mr. Eason on March 11th of this year suffering from left orbital cellulitis. He was obviously ill; he had had a rigor in the Surgery, and his temperature was 103°F. The history of the illness was as follows: Patient was well till fourteen days ago; he then had a nasal catarrh; five days later there was discharge from the nose and bad headache; five days later still—four days before admission—the left eye became swollen and inflamed, and he began to have attacks of shivering, which continued till admission.

On examination the left eye was proptosed and the upper lid much swollen, red, and œdematous; it was very painful on palpation; there was marked chemosis. The patient was transferred to the Ear and Throat Department. On examining the nose, typical creamy pus was seen in both middle meatus, and

on being wiped away returned at once. The patient had a very severe headache, and marked tenderness over the left frontal sinus.

Operation was performed through the left eyebrow. Pus was found on stripping the periosteum from the roof of the orbit; indeed, the periosteum was already separated along the inner wall and roof to some extent, showing how the suppuration had extended outwards from the sinus. The operation was completed by removal of the anterior end of the left middle turbinal and by enlarging the fronto-nasal duct sufficiently to allow of the passage of a rubber tube from the sinus to the nose. The patient made a good recovery, the eye returning to its normal position in about ten days. On cultivation of the pus B. Influenzæ was found in pure culture.

Case 2.—Raphaelo B., aged 43, was admitted into Barnabas under Mr. Ormond in February of this year suffering from left orbital cellulitis with a sinus discharging pus in the upper lid. The man spoke English very badly, making it difficult to obtain a history of the onset of the illness. The patient said his left upper eyelid had been affected for about six weeks; in the Out-Patient Department the lid was so swollen as to make the examination of the cornea very difficult; a diagnosis of abscess pointing in the upper lid was made, and the man was admitted. The day after admission the abscess burst, and under gas it was opened more freely. The patient also stated that the swelling in the upper eyelid had begun when a discharge from the nose, which he previously had suffered from, stopped.

Mr. Ormond transferred the case to the Ear and Throat Department. There was a sinus in the left upper eyelid which latter was reddened and swollen; the sinus admitted a probe for about one inch in an upward and inward direction, and bare bone could be felt. Examination of the nose was negative, no pus could be seen in the middle meatus, even after the application of hemisine and cocain, nor was pus seen on repeated examinations. Nevertheless it was considered certain that the sinus was due to frontal sinus suppuration.

Operation was performed, and on exposing the bone it was found that there was necrosis of the anterior and inferior wall of the frontal sinus about the middle of the supra-orbital margin. The sinus was opened and found to be full of polypoid mucous membrane, which entirely blocked the fronto-nasal duct, thus explaining the absence of pus in the nose. Similar means were taken to procure drainage as in the previous case. The man made a good recovery, except that the wound tended to sink in; this was met by a secondary operation under local anæsthesia at the hands of his dresser, Mr. Starling, when the edges were brought together after being undercut.

Case 3.—Alfred B., aged 18, was sent to the hospital by Dr. Wakefield, in December, 1912, for a sinus in the left upper eyelid. Ten weeks previously the patient had had an abscess at the inner and upper angle of the left eye, which was opened by Dr. Wakefield. Some time after the opening of the abscess some discharge was noticed from the left side of the nose. The sinus has continued to discharge a little pus.

On examination of the nose, creamy pus was seen in the left middle meatus, and returned after being wiped away. From the position of the re-appearance of the pus in the nose it was thought that the disease lay in the anterior ethmoidal cells rather than in the frontal sinus. The position of the sinus in the lid—just above the internal canthus—helped to support this view.

An intra-nasal operation was performed. The anterior end of the middle turbinal was removed, and the sinus in the lid scraped. A probe was found to pass straight into the nose from the sinus, and could be felt by a finger introduced into the nose. Free drainage was maintained for a few days by means of a rubber tube introduced through the nose, and the sinus in the lid healed. Cultivation of the pus gave a pure growth of influenza bacilli, and on a second occasion B. influenzæ and B. catarrhalis.

CASE 4.—Alice B., aged 30, attended in the Out-Patient Department complaining of a painful swelling at the upper and inner angle of the right orbit. The history of the case is as follows: She always, as far as she remembers, has suffered from a nasal discharge, worse when she bends down; she has frequent sore throats, and used to have headaches, but has been free of these for two years.

Five days before admission she had pain at the root of the nose, the day after the pain was worse and extended on to the forehead, and a small lump was noticed at the upper and inner angle of the right eye.

On admission the patient was pale and looked ill; her temperature was normal; there was a fluctuating tender swelling at the upper and inner angle of the right orbit, and the parts around this were œdematous and inflamed. On examination of the nose many polypi were present on both sides of the nose, and there was much creamy pus on both sides.

The operation showed pus in right frontal sinus and ethmoidal cells, and on investigating the septum between the two frontal sinuses pus was seen coming through a hole in the septum; this being enlarged showed the left frontal sinus to contain pus. Without further incision the left fronto-nasal duct was enlarged from the nose and a rubber tube left in the channel thus made. A like channel was made on the right side.

These cases emphasize the importance of nasal examination in all cases of suppuration in the orbit.

THREE CASES OF COLD PARAFFIN WAX INJECTION.

By

W. M. MOLLISON and A. H. TODD.

The following three cases are recorded to show the results obtained by the injection of cold paraffin wax and to emphasize the simplicity of this treatment. All three were cases of depressed bridge of the nose and each arose from a different cause.

The first case was that of a man aged 50, where the deformity was due to syphilis; he had a scar on the nose which was somewhat adherent to the deep tissues; he had also very marked ozæna. The second case was that of a girl, Melinda S., aged 19, who had had a blow on the nose, and the marked depression seen in the photograph was the result. The third case was that of a girl, Florence B., aged 9, whose nasal deformity was due to severe cancrum oris as a result of measles; the disease caused destruction of the incisive portion of the maxilla and spread to the septum. The results, as seen in the photographs, speak for themselves; it only remains to mention the method of operation.

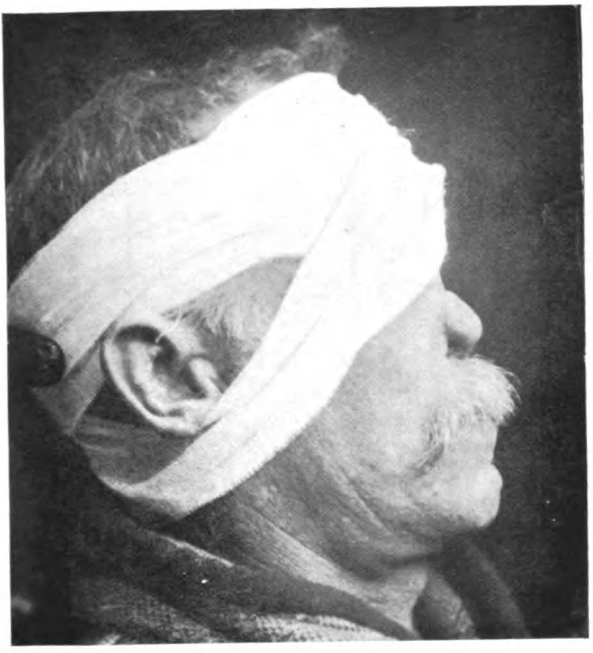
The essential is a special paraffin syringe made so that a solid piston is driven into a cylinder by a lever action; the syringe is Mahn's, and has a needle which is attached by a screw. The paraffin used is sterilised and put up in glass tubes having the same diameter as the cylinder of the syringe. It melts at 48°C. The skin is sterilised with iodine solution, and the part is anæsthetised by injection of a weak solution of novocain. The details of the method are described by Mr. Todd, as follows:—

The skin is sterilised by one application of 3 per cent. solution of iodine in chloroform, and anæsthetised with novocain-adrenalin solution. The latter is very conveniently prepared by dissolving one "Novocain A Tablet" in four drachms of *dis-*

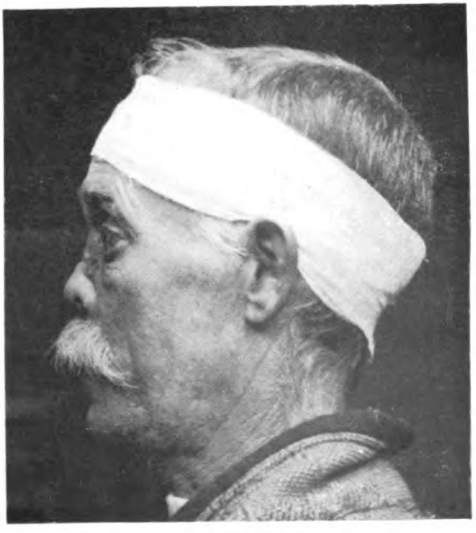
tilled water: tap-water must not be used, for novocain is decomposed by alkalies. For the same reason, the hypodermic syringe and needle must be rinsed with distilled water before use if they have been boiled in tap-water or sodium bicarbonate solution. Novocain has a low toxicity, and the whole amount of solution prepared may be used, if desired, though, as a rule, about two drachms suffice. The skin over the mid-line of the nose is touched with a pointed match, dipped in pure carbolic acid; this blanches it and renders it insensitive; the needle is inserted through the white spot, and the novocain is then injected in every direction until the whole operation area has been infiltrated. One puncture should be enough, and *the point should not be withdrawn until the injection has been completed.* After the needle has been pushed home in one direction it should be drawn back until its point is felt, just under the skin, then the direction should be changed, and a fresh thrust made, injecting all the time. So also, if it be necessary to recharge the syringe, it should be detached at the shoulder, leaving the needle in place. In this manner the whole injection may be performed quite painlessly. In about ten minutes' time the tissues will be insensitive and blanched, and, in addition, any bands of scar tissue that may be present will be rendered particularly obvious by the dimpling of the skin, the rest of the depressed area being "floated up" when the novocain fluid was forced in.

The area being anæsthetic, the syringe charged with wax, and the needle screwed home, enough wax is forced down the needle to appear at the point. The needle is introduced at some little distance from the position in which the wax is to be lodged and is pushed under the skin till the point is felt to be in the desired spot. Injection is then proceeded with. Should there be adhesion of the skin to the underlying tissues, it is better to divide this with a tenotomy knife before injection. The whole process is one of great simplicity; injections can be repeated until the best result has been obtained.

It only remains to add that the patients all expressed themselves much improved.



CASE 1.—Before injection.

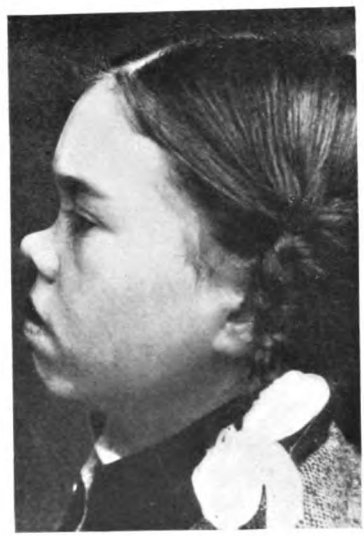


CASE 1.—After injection.

CASE 2.—Before injection.

CASE 2.—After first injection.

CASE 2.—After second injection.



CASE 3.—Before injection.

CASE 3.—After injection.

NEUROLOGICAL STUDIES.

(THIRD SERIES).

EDITED BY

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1. A NOTE ON THE ÆTIOLOGY AND TREATMENT OF SCIATICA.

BY

ARTHUR F. HERTZ, M.D.

SCIATICA may be defined as pain in the distribution of the sciatic nerve, the cause being neuritis in a large majority of cases. The remainder are examples of referred pain, the origin

of which is disease of the hip-joint or other part supplied by the nerve, or disease involving the roots of the nerve either within or without the spinal canal. It has generally been assumed that a primary sciatic neuralgia also occurs, and that sciatica is often a purely functional condition without any organic basis. My experience in a large number of cases during the last two years has, however, convinced me that this is not the case, as I have not seen a single case of sciatica, in which the ankle-jerk on the affected side was not diminished or absent, except when the pain was referred from some such obvious source as a tuberculous or oste-arthritic hip.

In our series of Neurological Studies for 1910 Dr. W. Johnson and I described how the ankle-jerk was present in all normal individuals, and how it was not infrequently lost on the affected side in sciatica. But the method of examination then recommended, in which the patient knelt with one knee on a chair whilst putting most of his weight on the other foot, did not allow of an accurate comparison between the jerks on the two sides. In cases of sciatica I now make the patient kneel on a comfortably cushioned chair with the legs placed symmetrically. When complete relaxation is attained, as shown by the ease with which the feet are plantar-flexed by squeezing the calves, the tendo-Achillis of each side is struck with a rubber-headed hammer. In severe cases the jerk on the affected side is at once seen to be either absent or very feeble compared with the normal jerk on the other side. In all other cases, when the tendons are struck with exactly the same degree of force as each other, the response on the affected is found to be less than on the healthy side, and when the force used is gradually reduced, a point is reached at which the former is absent though the latter is still present. This sign must indicate that either sciatic neuritis is present or that some other organic disease is involving the nerve or its roots. In most cases of sciatic neuritis tenderness is observed along the course of the nerve and pain is produced by the stretching caused by extension of the knee after flexing the hip to a right angle. But the change

in the ankle-jerk is the most constant sign and the only one which could not be a result of some functional disorder and could not be simulated by a malingerer.

If it be admitted that sciatic neuritis is the common cause of sciatica and that in all probability a true sciatic neuralgia does not exist, it is clear that all cases should be treated by absolute rest. As soon as the condition is diagnosed the patient should be put to bed and the leg fixed with a long Liston splint, which should only be taken off when every trace of pain and tenderness has disappeared. By this means the intractable cases of chronic sciatic neuritis, which often require rest for several weeks, would become very rare, and the majority of cases would recover completely within three weeks. Treatment by galvanism, kataphoresis, high-frequency currents, radiant heat, massage, and counter-irritation are of comparatively small importance. I long ago came to the conclusion that in the case of out-patients, the fatigue of a journey to the hospital more than outweighed any good that electrical treatment might otherwise have done, and that they would be far better resting at home. Such forms of treatment are, however, undoubtedly often of value as adjuncts to treatment by rest in severe cases and as after-treatment in all but the mildest cases.

2. NOTES ON FOUR CASES OF CEREBELLAR ATAXIA IN CHILDREN.

By

E. S. TAYLOR, B.C.

THE rarer forms of ataxia occurring in children which are not due to gross lesions of the brain, such as tumour or hæmorrhage, and are not due to ear disease, have been classified by Dr. F. E. Batten into three groups:—

1. Cases in which the ataxia is noticed in early life and in which there is a tendency to gradual improvement—Congenital Cerebellar Ataxia.
2. Cases in which the ataxia develops suddenly after some acute illness in a child previously quite healthy—Acute Ataxia or Cerebellar Polio-encephalitis.
3. Cases in which a child is healthy till a certain time and then gradually develops ataxia—Progressive Cerebellar Ataxia.

The four cases to be described belong to the first two groups under this classification, namely, congenital cerebellar ataxia and acute cerebellar polio-encephalitis.

GROUP I.—CONGENITAL CEREBELLAR ATAXIA.

Case A.—Sarah K., aged 9, was admitted into Mary under Dr. Shaw and subsequently Dr. Hertz in June, 1907, for twitching of the head and unsteady gait. The family history gave no evidence of nervous disease, and two grandparents were alive and well. Four other children were also healthy.

The labour had been normal and the child was bottle-fed. The condition was noticed during the first year, when she began

to show "suppleness of the neck" and a tendency to let her head fall to one side or the other. When two years old she could only sit up with difficulty and at three years was scarcely able to walk. During this time the parents noticed tremors of the arms and legs, which were constantly being moved about. The gait was very unsteady, but the patient never actually fell whilst walking. Her intelligence was good, and her memory was better than that of the other children in the family.

The condition slowly progressed until one year before admission and did not seem to be affected by scarlet fever and whooping-cough. During the year before admission, however, the patient improved slightly.

On admission, the child was well-nourished and cheerful. Her walk was very ataxic, the child jamming her heels down, swinging her arms wildly, and swaying her head. She leaned her body back, and this was apparently overbalanced by her head, so that she was always apparently about to fall backwards. The arms were more ataxic than the legs, but tremors were not noticed.

She answered questions quickly, but had difficulty in speaking long words, and this, with her Scotch accent, made conversation difficult. She could not write or draw, having little control over her arms.

The knee-jerks were brisk, plantar reflexes were flexor, and ankle clonus was absent.

The pupils were equal, and there was no nystagmus. There was no change in her condition while in the hospital.

I have heard recently from this patient's father that she is now a well-developed intelligent girl. "The twitchings of the arms," he writes, "and the unsteady gait continue, but are not nearly so bad, so that when walking on her sister's arm they are scarcely noticeable. She is still, however, unable to draw or write. There is also great improvement in her speech."

Case B.—Phyllis M., aged 9, was first seen in the Neurological Out-Patient Department four years ago. Labour was difficult and instruments were used. The patient was breast fed. When

nine months old it was noticed that she could not crawl about at all, and could not grasp anything owing to co-ordination of the hands. She began to walk when three years old, using the "inside of her feet," but she was continually falling over and showed definite ataxia. She was treated with splints for some months, until, in 1909, she was seen by Dr. Hertz, who diagnosed "congenital cerebellar ataxia." She has gradually improved since this time without treatment of any kind.

On being examined in 1913, the child is found to be quite intelligent and able to do lessons, including arithmetic, quite well. Her speech, however, is slow, and the words are spoken in a hesitating manner. The mother says the child is not usually nervous, but that occasionally, especially during a thunderstorm, she becomes very excited, screams incoherently, and shows much exaggeration of the ataxic movements. There are slight ataxic movements of both arms, and the right leg is more ataxic than the left when she walks. The right knee-jerk is slightly more brisk than the left, and both plantar reflexes are flexor.

When examined for the various signs of cerebellar disease, as described by Babinski, the following are well marked:—

a. *Adiadocokinesia.*—Instead of being able to supinate and pronate the hands in rapid succession, these movements are slowly and clumsily performed.

b. *Asynergia.*—i. When walking forward, patient's body tends to fall backwards and not to follow the legs. This was a well-marked feature in Case A.

ii. When standing up and leaning the head and body backwards, the body and legs are not bent forward in the form of an arc in order to maintain the balance, but the body is bent at the hips, the legs are kept vertical, and the patient falls backwards.

iii. When lying on the back with the knees and thighs fully flexed, the patient is able to remain perfectly steady, showing no ataxia, in contrast to cases of ataxia due to spinal cord disease, in which the ataxic movements are very well marked in this position.

These cases exhibit very clearly the leading features of congenital cerebellar ataxia. In both cases the ataxia was noticed during the first twelve months, and there was backwardness in learning to crawl and to walk; both children showed marked ataxia of the arms and legs, which was only present when sitting or standing up, with normal tendon reflexes and no muscular wasting; both had characteristic speech in which the words were pronounced in a slow, jerky, monotonous manner; and both were bright and intelligent and showed gradual improvement in their condition as they grew older.

Lastly, it may be noticed that nystagmus, which is sometimes present, was absent in both of these cases.

Pathology.—Experimental work by Luciani and others has shown that rapid recovery from symptoms follows removal of the cerebellum alone if the cerebral cortex remains undamaged. Moreover, atrophy or complete absence of the cerebellum may give rise to no cerebellar symptoms. If, however, the cerebral cortex is damaged, the cerebellar symptoms persist. It has been suggested, therefore, by Dr. Batten that in these cases there has been some damage or lack of development in the cerebellum and cerebrum at or before birth, and that as the cerebrum develops it assumes the co-ordinating function of the cerebellum, and in this way more or less complete recovery may take place. It is interesting in this connection that in Case B labour was difficult and instruments were used.

Group II.—Acute Cerebellar Polio-Encephalitis.

Case C.—Maggie D., aged 10, was admitted from the Neurological Out-Patient Department into Clinical under Dr. Shaw in June, 1913, for inability to walk. Her parents were both healthy, and there was no nervous disease in the family. A brother and two sisters were all healthy. Patient had scarlet fever in July, 1912, but there were no sequelæ. Nine weeks before admission patient's two sisters had measles, and one week later the patient herself complained of sick headache and was kept in bed one week. On getting up again she was noticed

to nod her head about from before backwards, though showing no movement when lying down or when asleep. Four days later she was unable to stand up, and complained of a sharp pain in the left chest radiating to the groin. The nodding of the head was quite independent of talking or of any attention paid to her.

On examination, the patient was found to be healthy in appearance and able to lie quite still in bed, but when raised to a sitting posture and asked questions the movements of the head at once commenced. The speech was slow and hesitating, but hardly scanning in character. The knee-jerks were present, and both plantar reflexes were flexor; the pupils reacted normally and were equal in size, and there was no nystagmus. When placed upon her feet she exhibited well-marked "asynergia," falling backwards continually and showing extreme ataxia. On the day of admission the patient complained that as she walked the room "seemed to go round," but this symptom did not persist.

On June 6th, two days after admission, the movements were less marked, and on June 7th she could stand unassisted, the movements of the head were less, and she was able to talk more readily. On June 8th the walking was much improved, and on June 12th patient could walk without assistance. On June 14th the ataxia had almost gone, and the patient was discharged eight days later.

In this case no drug was given as treatment.

Case D.—Phyllis P., aged 7, was admitted into Clinical under Dr. Hertz on July 11th, 1913, for inability to walk. The family history showed no nervous disease, and there were six other children alive and healthy. The patient was a full-term child, and labour was normal. She had measles and chicken-pox when a baby. Two days before admission the child walked to school in the morning, a distance of about one mile, and seemed quite well. At mid-day as she was walking out of school she suddenly fell down and was unable to stand up without assistance. She could not walk alone and was carried back by her sisters. She did not vomit, but complained of headache the next morning.

On admission the patient was a well-developed child and answered questions rationally, but seemed apathetic. Her speech was not normal, the words being spoken in an abrupt manner and in a monotonous tone. Her temperature was 99·2°. Both knee-jerks were present, the plantar reflexes were flexor, and there was no muscular wasting. There was no optic neuritis, the pupils reacted normally, and there was no nystagmus. When lying in bed the patient made no abnormal movements, but on sitting up she moved her head irregularly, and could not maintain the sitting posture without assistance. When standing up she was completely ataxic, with rocking movements of the head in all directions and irregular movements of the arms and body, and when trying to walk she showed a constant tendency to fall backwards unless supported. She was not giddy, and made no effort to support herself when falling. There was intention tremor of both arms. There was evidence of old chronic otitis media in the left ear, but tests carried out showed no other aural defect, the labyrinth being normal on both sides.

A lumbar puncture was performed and 3 c.c. of cerebro-spinal fluid were withdrawn. The fluid was under normal pressure, contained no cells nor organisms, and was quite clear. The patient was at once given urotropine in 10 grain doses every six hours. She had an evening temperature varying from 99° to 101.° Babinski's tests for cerebellar disease were all positive, "adiadocokinesia" and "asynergia" being well-marked.

On July 15th the movements were much less and the intention tremor was very slight, though the ataxia remained unchanged. On July 16th the patient could almost stand alone, and on July 19th she could walk a few steps alone, holding her head to the left. On July 21st the speech was normal, and the patient could walk with her eyes closed. On July 22nd "adiadocokinesia" was still present, but the other signs were not definitely given. There was some frequency of micturition and hæmaturia. The urotropin was, therefore, discontinued. On July 28th the urine was normal, and four days later patient showed no signs of cerebellar disease, her gait, speech, and arm movements being absolutely normal.

These cases exhibit the characteristic features of acute cerebellar polio-encephalitis: the seasonal incidence, both cases occurring during the warm months; the age of the patients; the sudden onset with febrile reaction; the extreme ataxia without loss of power; the characteristic slow jerky speech; and lastly, the tendency to rapid and complete recovery.

Acute cerebellar polio-encephalitis is one result of the acute infection, which also gives rise to cerebral polio-encephalitis and to anterior polio-myelitis. The cerebro-spinal fluid in these cerebellar cases shows no turbidity, only occasionally a few lymphocytes, and only rarely an increased cerebro-spinal pressure, thus emphasizing how localised the infection may remain.

Treatment.—The experiments of Flexner and Clark have shown that urotropin given in full doses as soon as possible has, in many cases, prevented infection in monkeys, and diminished the virulence if not destroyed the organisms in cases already infected. The exceptional rapidity of recovery in Case D may be in some measure due to the urotropin given, but further clinical investigation is required before definite statements can be made.

Prognosis.—The prognosis seems to be always good, both as regards life and the prospects of ultimate complete recovery. One of the earlier published cases of acute cerebellar polio-encephalitis was quite well three and a half years after the illness began, and when seen twenty-nine years later had remained perfectly well. Most of the cases more recently reported have recovered within a year, and in many the total duration of the disease was three months. In Case C almost complete recovery had occurred when the child was last seen ten weeks after the beginning of the illness. Case D is very exceptional in that the symptoms had almost completely disappeared in a fortnight, and the child was perfectly well in less than three weeks.

3. CASE OF POLIO-ENCEPHALO-MYELITIS ASSOCIATED WITH OPTIC NEURITIS AND MYOCARDITIS.*

By

ARTHUR F. HERTZ, M.D., W. JOHNSON, M.D., AND H. T. DEPREE, M.B.

E. K., aged $12\frac{1}{2}$, was admitted into Guy's Hospital on March 5th, 1912, for paralysis of the left side. This had developed suddenly the same day; the patient fell down, and on being picked up was found to have lost the use of his left arm and leg.

On admission his pulse was 54 and temperature 97° F. He was semi-conscious; he could not move his left arm or leg at all, and there was some weakness on the left side of the face. His right pupil was smaller than the left. Both knee-jerks were present, the left being exaggerated; an extensor plantar reflex, diminished abdominal reflex, and ankle clonus were present on the left side only. There was some twitching of the right side of the face. Incontinence of urine and fæces was present. The heart was greatly dilated and a loud systolic murmur was heard at the apex, but there was no history of rheumatism, scarlet fever, or previous cardiac disease. The case was diagnosed as cerebral embolism.

The patient became fully conscious the next day. A tremor now occasionally affected the right arm and leg. There had

*The case was shown before the Clinical Section of the Royal Society of Medicine (*vide* Proceedings, page 91, 1913).

never been any rigidity of the arm and only slight rigidity of the leg; but in the course of three days both became completely flaccid and the tendon reflexes disappeared, but the extensor plantar reflex remained. The knee-jerk and ankle-jerk disappeared from the right side also, but there was only a very slight degree of weakness in the right leg. The temperature was now taken in the rectum and found to be 101·2° F. The pulse was still very slow, but the cardiac condition was otherwise unaltered. On March 8th complete paralysis of the left external rectus muscle developed. Lumbar puncture showed that the cerebro-spinal fluid was under increased pressure; it contained numerous lymphocytes and polymorphonuclear cells in approximately equal numbers, but no organisms were found. Definite optic neuritis was present, which was more marked on the left side than on the right.

On March 16th some atrophy of the left side of the tongue was noticed. On March 20th hæmaturia developed, and the urine contained many pus cells and granular casts in addition to blood; this was probably due to the large quantity of urotropine which had been given. The urine became normal again in about ten days.

The patient gradually regained some power in the left leg and to a less extent in the left arm. Slight wasting of the affected muscles took place, but there was no loss of electrical excitability. In the course of the next four months the knee-jerks reappeared on both sides; the knee-jerk and arm-jerks became exaggerated on the left side, on which well-marked patellar and ankle clonus also developed. The plantar reflex remained extensor on the left side. The temperature gradually fell to normal in the course of three weeks. The systolic murmur became less distinct and the heart less dilated. The paralysis of the left external rectus did not disappear until October.

At the present time (September, 1913) the patient has recovered to a very considerable extent. The left arm, however, is very weak, though the left leg is comparatively slightly affected. The jerks on the left side are still exaggerated, and the plantar re-

flex extensor. The urine is now normal, and the heart is normal except for an occasional faint systolic apical murmur.

The subsequent history showed that the original diagnosis of embolism was incorrect; the sudden onset with unconsciousness and the rapid recovery from acute symptoms would further exclude multiple emboli resulting from infective endocarditis. Acute polio-encephalo-myelitis seems a more probable diagnosis, the infective agent being probably the same organism which more commonly results in simple anterior polio-myelitis. The same toxins which affected the nervous system probably caused the myocarditis.

4. PROGRESSIVE MUSCULAR ATROPHY ASSOCIATED WITH PRIMARY MUSCULAR DYSTROPHY IN THE SECOND GENERATION.*

By

ARTHUR F. HERTZ, M.D., and W. JOHNSON, M.D.

The father, G. H., aged 41, a butcher, came under observation six months ago, complaining of weakness, chiefly in the left hand. He was a strongly developed man and had formerly great muscular power, being capable of carrying "a quarter of beef." His occupation necessitated this daily overuse of his muscles. He first noticed weakness of the left hand in February, 1912, and he ascribed it to an injury to the left index-finger from a chopper. The whole of the left arm gradually became weak, and within six months of the onset the right hand and later the arm became affected. The legs also began to drag about this time, the left being the first involved. About a year after the onset, rather rapid wasting occurred in the hands, the weakness having previously been associated with only slight wasting. This continued for about four months, but no further wasting has occurred during the last two months. The legs have recently become rapidly weaker, and he now frequently falls in the street. Fibrillary tremor has been marked in the arm, leg, and shoulder muscles. The thenar and hypothenar eminences are markedly wasted in both hands, and the interossei to a less extent. The opponens movement is almost absent, while all the

*The cases were shown before the Clinical Section of the Royal Society of Medicine (*vide* Proceedings, page 177, 1913), and before the Neurological Section of the International Medical Congress, London, 1913.

muscles of the forearm and arm are deficient in power; they are also flabby and smaller than they were three or four years ago. The calf muscles do not appear to be wasted, but some weakness is present. The thigh muscles, especially those on the inner side, are definitely wasted. The abdominal and back muscles both show weakness. The strabismus which the patient exhibits has been present since birth. At times there is a tendency to nystagmus. The orbiculares palpebrarum are weak, and there is some tremor of the tongue. Examination of the eyes shows nothing abnormal. It is difficult to obtain any trace of tendon reflexes, with the exception of the triceps-jerk. Cremasteric and abdominal reflexes are not obtained; the plantar reflexes are flexor. There has never been any bladder or rectal trouble. Cutaneous sensation is normal. The Wassermann reaction of the blood is negative.

Since June, 1913, slight bulbar symptoms have developed. The patient experiences great difficulty in coughing, and his voice "seems to go from him" after he has been talking for a short time. He has not had any dysphagia.

S. H., aged 17, the eldest son of G. H., was a well-developed child at birth and normal in every way up to the age of 8. It was then noticed that he walked with his back arched and with his abdomen prominent. Soon his legs began to drag, and he used to fall about a good deal. Within three years of the onset he was quite unable to walk. In raising himself from the floor "he climbed up his legs," and his power of walking upstairs was very early impaired. His face now has the myopathic character, but there is no marked facial weakness. There is much wasting of the scapular, humeral, and thigh muscles, with a corresponding amount of weakness. The hamstrings are much contracted. The calf muscles are large and firm in comparison with the other muscles. The hand muscles are not wasted, and the patient is able to make good use of them. The knee-jerks are absent, but the ankle-jerks are present. No arm-jerks could be obtained. He is clearly suffering from the pseudo-hypertrophic form of primary muscular dystrophy.

F. H., aged 5, another son of G. H., appears to exhibit a very early stage of pseudo-hypertrophic paralysis, his condition being similar to what was noticed at the onset of his brother's illness. He has the same tendency to arch his back and protrude his abdomen when walking. His calves are well developed and unusually firm. There is, however, no wasting present. In rising from the floor he shows a distinct tendency to climb up himself. His condition is still too early for a definite diagnosis to be made, but the association with the other cases makes it highly probable that it is an early stage of a primary muscular dystrophy.

There are two other children in the family—one a boy aged 15, and the other a girl aged 7, both of whom are perfectly normal. Inquiry into the history of relatives on both sides led to negative results, with the exception of a boy, J. H., aged 3, the son of the first patient's brother. For the last six months he has been noticed to be falling about. No wasting is present, but there is a tendency to lordosis of the spine, and to prominence of the abdomen when he walks, and the calves are unusually well developed and hard. It is possible that this boy may also be suffering from the earliest stage of a primary muscular dystrophy.

We regard the father as a case of the pure progressive muscular atrophy type of motor neurone disease, and the two sons and nephew as cases of primary muscular dystrophy of the pure pseudo-hypertrophic type in the two younger boys and of a mixed pseudo-hypertrophic and facio-scapulo-humeral type in the eldest boy, the former apparently being the primary condition, as the paralysis and atrophy of the facio-scapulo-humeral muscles are of comparatively recent development. If these diagnoses are accepted, the most probable conclusion is that the association is accidental, as it is difficult to conceive how there can be any causal relationship between a primary spinal cord disease in one generation and a primary muscular disease in the next generation. On the other hand, it is possible that the father is also suffering from a form of primary muscular dys-

trophy; but it is very uncommon for primary muscular dystrophy to develop at such a comparatively advanced age as 39, the transmission from father to son and to brother's son is very uncommon, the distribution of the atrophy is most unusual, the hands having been first affected and still being the parts most seriously involved, and the progress has been unusually rapid.

Perhaps in the father there is an inherited tendency for muscular atrophy to occur, and the over-use is the exciting cause, the tendency alone being insufficient to cause atrophy without over-use. In the sons and nephew the tendency is greater as it often is in the second generation of inherited diseases, and consequently atrophy and paralysis occurred at an early age without any obvious exciting cause.

5. TWO CASES OF BILATERAL ATROPHY OF THE FACE.*

By

ARTHUR F. HERTZ, M.D., AND W. JOHNSON, M.D.

Case 1.—Thomas T., aged 26, is the only member of his family affected. His father suffered from syphilis, and appears to have died from general paralysis of the insane, but the patient himself shows no evidence of congenital syphilis. He had double otorrhœa from infancy up to the age of 14; there has been no recurrence of this, but he is rather deaf. Six years ago he had a cut on the right side of his face, which did not suppurate and healed rapidly, but it left a small scar. Two years ago he noticed some puffiness under the eyes, which gradually passed away, but his face, especially the right side, from this time became progressively thinner, until his friends thought that he must be consumptive, and advised him to come to the hospital. He was first seen by us in December, 1912; the condition has steadily increased, especially during the last six months. The right side has always been the most affected.

He feels in perfect health, and, except for his face, he is exceptionally well developed. His chest is normal, and there is no evidence of nervous disease. There is no weakness of the facial muscles, all of which react normally to electricity. His tongue is unaffected ; cutaneous sensibility and taste are normal. There is no scleroderma, which is present in one class of hemiatrophy. Pigmentation has been recorded in some cases, and this patient had a few small brown patches over his buttocks, which he had never observed himself.

*The case was shown before the Clinical Section of the Royal Society of Medicine (*vide* Proceedings, page 93, 1913), and before the Medical Section of the International Medical Congress, 1913.

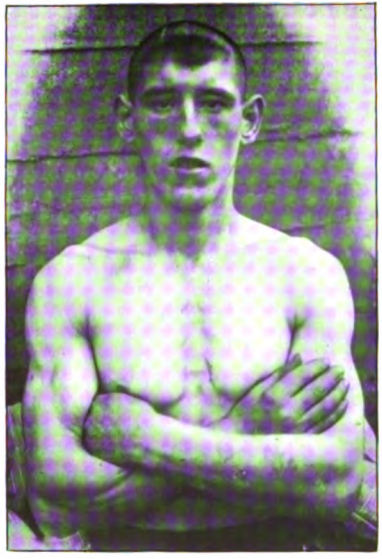


FIG. I.

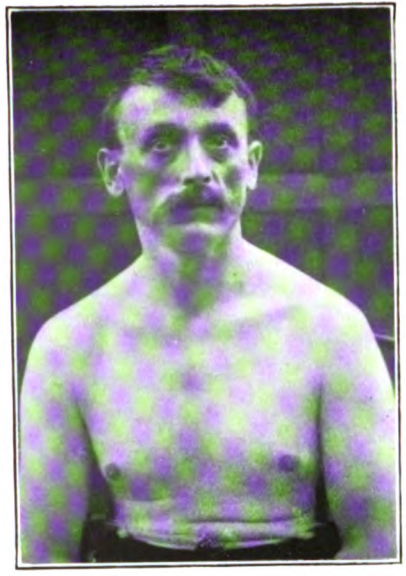


FIG. II.

FIG. III.

TWO CASES OF BILATERAL ATROPHY OF THE FACE.

Case 2.—Carlos F., an Italian, aged 38, had until recently always been in excellent health. He is very well developed and served ten years in the Italian Navy. When he first came to England he followed the occupation of a plaster-modeller for five years, but for the last three years he has worked in a lead factory.

He presented himself for weakness of the right side of his face, complaining that the right eye closed incompletely during sleep. His face shows a well-marked condition of atrophy on both sides, especially the left. The cheeks are extremely hollow, and the tissues covering the bones of the face are very wasted. The facial paresis has improved greatly in the five weeks since he first came to the hospital. He can close his eyes quite firmly, and only when he smiles is any weakness of the right side of his face noticeable. He shows well-marked signs of chronic lead poisoning with great weakness of all the extensor muscles of the wrists and fingers of both sides. He has a blue line on his gums and is extremely constipated ; he has had numerous attacks of abdominal colic. He showed no other signs of involvement of his nervous system; the reflexes are all normal.

The accompanying photograph (Fig. II.) shows the atrophied condition of the face, which is in striking contrast with the one taken ten years ago (Fig. III.). His friends state that they noticed no change in his appearance until four months ago.

We suggest that these cases are of the same nature as the more familiar hemiatrophy of the face, and correspond with the cases recorded by Wolff,[1] Oppenheim,[2] Judson Bury,[3] and Batty Shaw,[4] as examples of "bilateral hemiatrophy of the face," the ætiology of which is quite obscure.

REFERENCES.

1. J. Wolff : quoted by Oppenheim, loc. cit.
2. H. Oppenheim : Lehrbuch der Nervenkrankheiten, p. 1547, 6th edition, 1908.
3. Judson S. Bury: Diseases of the Nervous System, p. 267, Manchester, 1912.
4. H. Batty Shaw : Clinical Society Transactions, xxxviii, 222.

6. TEN CONSECUTIVE CASES TREATED BY HYPNOTISM.

By

F. G. L. SCOTT, B.A. Oxon.

Case I.—Telegraphic Neurosis.

Frank C., æt. 47, had for four years suffered from increasing difficulty in tapping certain letters of the Morse code. Any letter beginning with a "dot" caused him great uneasiness if it came first in a word. Sometimes he would delay even half a minute, his hand trembling violently. At other times the delay would be short, but it was nearly always present and seemed to bear no relation to his confidence on any particular occasion.

Urgent suggestion—apart from hypnosis—caused a temporary improvement in manipulating the key which the patient brought with him, but a relapse followed a return to ordinary work.

He was treated six times, and every effort to induce hypnosis was made, but without success. He was ordered a week's rest and suggestion with electrical treatment was given, but there was no permanent improvement. As a last resource a drowsy state was induced by a small dose of Cannabis Indica, and suggestion took effect so far that he could tap perfectly on the key he brought with him. The hypnotist accompanied him to Paddington Station, and he sent several messages of upwards of 80 words without a falter. A month later he wrote, "great improvement, but am not yet perfect."

Case II.—Hysterical Paralysis.

Geo. H., æt. 45, carter, came up complaining of "paralysis" of the right leg. Three months previously "he awoke one morn-

ing, and when he tried to stand, fell back violently into bed, his legs giving under him." He "thought he had had a stroke." He walked with the aid of a stout stick, deprived of which he could merely shuffle along.

He was very lightly hypnotised and told he could walk if he gripped a penny. He immediately did so with some slight stiffness, but appeared to be quite sure of his footing.

There was no recurrence of the trouble.

Case III.—Tic.

Henry H., æt. 13, complained of a violent convulsive movement of the head whenever he spoke. It was so pronounced as to be highly ludicrous and was a great trouble to his mother, who said it "got on her nerves." It had lasted six or seven months, and had latterly grown worse. The boy was intelligent and well placed in school.

A light stage of hypnosis was induced, and he was ordered to say "how do you do" several times. The motion was very considerable, but disappeared under vehement suggestion; after the experiment repeated questions did not elicit it. The following day the boy only once noticed the motion.

Demonstrated before a class of students his first remark gave rise to the motion, but after this he spoke without the trouble.

A month later the mother said the motion was now only seen very rarely in moments of excitement.

Case IV.—Choreiform Movements.

Ada S., æt. 13, came up for inability to write and choreiform movements. Recently her teacher had reported that the child's writing was becoming very slow and very untidy; previously, as shown by a specimen, it was excellent. For the past month she had developed movements of the right side, and there appeared to be considerable weakness of the right arm. To write her name she required sometimes as much as half a minute, and the writing was very irregular. She was distinctly anæmic and there was a systolic bruit in the mitral area. Though chorea

had been diagnosed, the result of the treatment shows that her condition must have been hysterical.

Deep hypnosis was readily induced, and after the sitting she wrote her name without any tremor and fairly quickly. In subsequent treatments the movements gradually quieted down, till after a week they had practically disappeared, and the child, unasked, said that "she felt her arm stronger." Her mother said she could now lift a cup without tremor, though previously she had often dropped it.

A month later it was found she had returned to school and her writing was quite satisfactory.

Case V.—Obsessions.

Harry D., æt. 18, had, since the age of 10 been troubled by "bad thoughts." These were of a disgusting nature, being chiefly related to defæcating and a perverse form of intercourse. He also imagined he might stab people in the genitals, and brooded on murders and acts of atrocity.

Not even the lightest hypnosis could be induced, and though considerable improvement was soon apparent, later treatments produced no further good effects. Six weeks later his father said there had been great improvement, but the lad was by no means cured. It is probable that the high degree of auto-suggestibility of the patient (manifested in the very numerous odd sensations he described apart from the actual "bad thoughts") and the length of the history militated against a very satisfactory result.

Case VI. Masturbation.

Patrick O., æt. 31, had practised masturbation for 20 years; up to the age of 15 very constantly; from 15 to 30 several times a week; and in the present year at intervals of 10 days or so. He complained of pain in the right arm, very vaguely localised, and "a sharp agonising pain in the back passage as though a red-hot knife were passed up." He was very depressed, and

thought his pains were a judgment of God. He would not eat with his family, but would take his food to his bedroom. He felt dizzy and thought he would fall into the river when working at the docks. He said he would take his life had he the necessary courage.

Deep hypnosis was soon obtained, and at the end of a week he reported his family relations were entirely changed; his work, previously a drudgery, was performed with pleasure; he had had no impulse to masturbate and no pain. The previous night he had celebrated his "return to health" by becoming intoxicated. Under hypnosis he was forbidden to touch alcohol for a week, and the prohibition was effective.

At the end of a month he had only once experienced strong desire to masturbate, but it had been overcome.

Nine weeks after the first treatment (not having been treated for a month) he said the habit had not once been renewed. He complained of pain in the arm, which was readily dissipated by suggestion. A few weeks later he came up to report that he "had been promoted" at the docks.

Case VII.—? Hysterical Chorea.

Annie A., æt. 18, complained of choreic-like movements of the left side, spasmodic flexion and extension of the ankle and wrist, and rapid lifting of the shoulder. The movements were continuous, increased in excitement, and always of the same type. Six months previously she had been treated for supposed chorea affecting the right side.

No efforts could induce hypnosis; at first she could not be persuaded to close her eyes. After several treatments she would close them for 30 seconds. After six treatments there was considerable improvement, but relapse under excitement. As it still was found impossible to induce the slightest degree of hypnosis, the suggestive effect of electrical treatment was tried, but at the end of a month it was reported that there was no improvement at all. The failure of suggestion makes it probable that the patient is really suffering from some organic disease.

CASE VIII.—TRAUMATIC PARALYSIS. DEPRESSION.

John W., æt. 49, was admitted for weakness and trembling of both legs, especially the left. Four years ago he fell downstairs and "injured his back." He is said to have been "unconscious for several months," and on gaining consciousness was completely paralysed in the left arm and below the waist. The condition gradually improved, but he had been "weak in the left side" ever since. Three weeks before admission under Dr. Hale White he was lifting a piano when he felt something snap in his brain, and he nearly fainted. An hour later a very marked tremor and weakness in the legs set in; he walked with a pronounced limp. He was treated in the Electrical Department for about a fortnight with little improvement.

Deep hypnosis was induced, and after three treatments the tremor was less; after four it suddenly disappeared, and his gait became and remained absolutely normal. At his request he was treated for depression, and on discharge he appeared well satisfied with the result.

CASE IX.—STAMMERING. PERSISTENT HEADACHE.

Willie A., æt. 12, was brought up for stammering which dated from an epileptic fit some six months earlier. There was distinct neuropathic trouble in the family. He was an intelligent boy, doing well at school, but stammering kept him back a good deal. Among his schoolmates he was known as "Stuttering Bill." When quiet, the stammer was slight or absent, but the slightest excitement or nervousness brought it on. He was troubled by severe headache, which his mother said "never left him for days."

Treatment caused an immediate improvement in sleep, and the headaches soon disappeared. A gradual improvement in speech was reported by his mother. At the end of a fortnight his treatment was unavoidably interrupted.

A month later his mother said the stammering was very greatly lessened. It now only occurred when he was "taken

unawares," and a word from her at such times would prevent it. He was no longer troubled by headaches, and his general health was much improved.

Case X.—Agoraphobia.

Mrs. K., æt. 45, had six years previously been deserted by her husband; this trouble so overcame her that her health gave way. She described an "attack of palpitation," which prostrated her for several days and was followed by a sense of "oppression in the chest." She became afraid to go out of doors, and for some years would never venture out alone; she became very hysterical, breaking into violent weeping whenever spoken to.

At first, to avoid alarming the patient, suggestion as distinct from hypnosis was tried. She could hardly sit quiet in the chair, and alternately bemoaned the oppression she felt in her chest and broke into hysterical laughter. After six treatments she was quieter, occasionally went for long walks and even to places of amusement, but her sleep was still bad.

Hypnosis was now induced at the request of the patient, and there was a steady and immediate improvement. By the twelfth sitting she had accustomed herself to going out alone and felt very little dread of it. When treatment was discontinued after a few more sittings, she could travel alone by train (a thing she had not attempted for five years) without the smallest apprehension, and she no longer complained of faintness or oppression. Her sleep had improved, but was still not very good.

In was interesting to note that though improvement was gradual (the treatment extended over some ten weeks), there was at no time anything of the nature of a relapse.

CREATININE AND CREATINE: A REVIEW.

By

E. P. POULTON, M.B., B.Ch. Oxon., M.R.C.P.

Contents :

Creatinine is only second to urea in being the most plentiful of any nitrogenous compound of the urine. However, very little was known about its occurrence until Folin[10] described a method in 1904 for estimating both creatinine and creatine quantitatively. This publication opened up a new field of research and gave the necessary impetus to a large number of workers in Europe and America.

The result of all this work has been to establish certain definite facts. However, it seems probable that some of the results will not be substantiated by future work, as within the last two or three years Mellanby and Folin and Taylor have pointed out certain sources of error in the method as usually performed, and quite recently Greenwald and Graham and Poulton have noticed that the presence of aceto-acetic acid in the urine completely upsets the determinations. There can be little doubt that a large amount of the work on this subject is at present resting on very insecure foundations.

The purpose of this paper is to describe briefly these various sources of error, and the methods for obviating them, and at the same time to consider the extent of our present knowledge of this whole subject.

I.—Folin's Method of Estimating Creatinine and Creatine.

The original colour test for creatinine in urine was described by Jaffé; it consists in the addition of picric acid and soda to the urine, when an orange colour is produced.

Folin's method of estimating creatinine was based on this colour test. He found that 0·01 grm. creatinine when dissolved in 10 c.c. water and treated with picric acid and soda, and subsequently diluted to 500 c.c., gave a colour identical with a $\frac{1}{2}$N potassium bichromate solution, *i.e.*, a layer of the potassium bichromate 8 mm. in depth exactly matched a layer of the diluted creatinine solution 8·1 mm. in depth when viewed by transmitted light. The estimation of creatinine in a specimen of urine was carried out as follows: Some $\frac{1}{2}$N bichromate solution was placed in one cell of a Duboscq colorimeter, and the cylinder adjusted to 8 mm. 10 c.c. of the urine was treated with 15 c.c. saturated picric acid and 5 c.c. 10 per cent. soda, and after mixing and allowing to stand for five minutes it was diluted to 500 c.c. with water and put in the other cell of the colorimeter, and the cylinder was adjusted until the two colours matched. The mean of several readings was taken. The amount of creatinine (in a volume of urine V) is $\frac{V}{v} \times \frac{8{\cdot}1}{r}$ where v is volume of urine used for the determination, and r is the scale reading.

The presence of creatine in the urine was determined by a somewhat similar process; it was converted into creatinine by heating 10 c.c. urine with 5 c.c. $\frac{N}{1}$ HCl on the water-bath for three hours, and after cooling and neutralising with 10 per cent. soda, the urine was treated in exactly the same way as before. In this case the amount of creatinine originally present in the urine + the creatinine obtained from conversion of the

creatine were determined together. The creatine present can be calculated from the difference between this quantity and the amount of creatinine originally present in the urine (the so-called pre-formed creatinine).

It has been shown that the orange colour produced in Folin's method of estimation is due to a strong reducing action of creatinine on picric acid, which is converted into diamino-mononitrophenol. This red substance retains its intensity when considerably diluted. The accuracy of this method when applied to the estimation of creatinine has been tested by Mellanby,[34] van Hoogenhuize and Verploegh,[24] and many others, and has been generally regarded as satisfactory, but the nature of the reducing action on which it depends makes it absolutely necessary that it should be carried out exactly as described. The important thing is to take such a volume of urine for the determination, that when diluted to 500 c.c. it will give a reading as near 8·1 mm. as possible. Mellanby states that the temperature of the reagent and also of the water used for dilution should be constant.

The most satisfactory amount of urine to take for the determination is 10 c.c., but, of course, if the urine is concentrated, 5 c.c. may be taken and an equal quantity of water added to it. If the urine is dilute, 15 c.c. or even 20 c.c. may be taken, but in this case there is a difficulty because, when the picric acid and soda are added, the total volume of the reacting substances is greater than usual, and so the orange colour takes more time to develop. The mixture must be allowed to stand at least $7\frac{1}{2}$ minutes. It will probably be safer always to wait this length of time before dilution. Under no circumstances must a volume of urine larger than 20 c.c. ever be taken, as is shown by the following example: 10 c.c. urine gave a reading of 6·39 mm. as a mean of three separate determinations; 10 c.c. of the same urine diluted to 20 c.c. with water gave a reading of 6·56, which is within the limit of accuracy for the method; 10 c.c. of the same urine diluted to 30 c.c. before adding the picric acid and soda gave a reading of 7·72 mm., which indicates a very large error in the determination.

However, there is another method that has often been employed for dilute urines, such as diabetes, in order to get a scale reading within the limits prescribed by Folin. The quantity of urine taken is mixed with picric acid and soda and diluted to 250 c.c. instead of 500 c.c. Folin himself has adopted this plan, and he has been very largely followed in America by Benedict and others. Van Hoogenhuize and Verploegh[24] state that the error caused by this proceeding is too small to be of importance. Mellanby,[36] however, points out that from Chapman's work that while creatinine produces the diamino-mononitrophenol with picric acid, other reducing substances, such as dextrose, etc., produce the mono-amino-di-nitrophenol, which also has an orange colour, but that this substance loses all colour on dilution. Hence, full dilution is absolutely essential, or otherwise too dark a colour may be obtained.

Experiments performed by Graham and Poulton to test this point have shown that there is certainly a definite error in many cases. Thus, 10 c.c. urine treated with picric acid and soda gave a reading of 6·39 mm. when diluted to 500 c.c.; 5 c.c. of the same urine, to which 5 c.c. water were added, was treated with picric acid and soda as usual, and the urine was then diluted to 250 c.c. The reading was 6·00 mm., which was perceptably lower than the previous one.

Weber[54] goes a step further and adds less than the ordinary amount of picric acid and soda, and dilutes only to 100 c.c. However, he admits himself the uncertainty of the results.

In many cases the error from diluting only to 250 c.c. is not very large, but as it adds an element of uncertainty to the determinations it should be avoided.

Greenwald[21] has attempted to get over the difficulty of estimating creatinine in dilute urines, by taking 25 c.c. and adding twice the quantity of picric acid and soda to it and diluting to 250 c.c. This method certainly gives an apparently correct result, but it is open to the objection that it is obtained by compensating the errors already mentioned against one another, and it is possible that this compensation may not take place in all cases.

While the estimation of creatinine has been very generally approved by those workers who have tested it, Folin's method of estimating creatine has been subject to much criticism.

To begin with, it has the disadvantage of being an indirect method. Some authors[8, 54] complain that the brown colour produced by heating the urine interferes with the creatinine+creatine determinations. This certainly is not usually the case. A lot of doubt has been expressed as to whether treating the urine with half its volume of $\frac{N}{1}$ HCl for three hours is really sufficient to convert all the creatine that may be present into creatinine. Mellanby[34] states that five hours is necessary.

Some experiments by Graham and Poulton[20] have shown that three hours is sufficient in cases where the amount of creatine present is small. Weighed quantities of pure crystalline creatine were added to urine. If the urine contained 0·016 per cent. creatine (calculated as creatinine), complete conversion took place in three hours. If the urine contained 0·076 per cent., only 0·037 per cent. was converted in $3\frac{1}{2}$ hours; if heated for $5\frac{1}{2}$ hours 0·068 per cent. was recovered in the urine, so that complete conversion occurred in this case, the difference being within the experimental error of the method. In another case urine containing 0·152 per cent. creatine (calculated as creatinine) was used, and only 0·044 per cent. was recovered as creatinine as a result of three hours' heating.

Since the publication of the method it has been suggested that twice the volume of $\frac{N}{1}$ HCl should be added to the volume of urine taken to ensure complete conversion of all the creatine. This suggestion* would seem to introduce the error of dilution mentioned above. If 10 c.c. urine were used for a determination the addition of double the volume of $\frac{N}{1}$ HCl and its subsequent

*Since this has been written, a very full account of the estimation of creatinine and creatine by Thompson, Wallace, and Clotworthy has appeared in the *Biochemical Journal*, 7, 445, 1913. These authors have obtained very similar results to those mentioned in this paper, but they do not lay stress on the error from dilution. It is possible that the low creatine results noticed by them with 20 c.c. normal HCl may have been due to this cause.

neutralisation would increase the volume to nearly 40 c.c. before the picric acid and soda were added, and this would probably lead to a grave error in the determination. Heynemann[22] who used this method complained of finding that the pre-formed creatinine was sometimes larger in amount than the creatinine+ creatine. This would be explained by this error in dilution.

Klercker[27] has pointed out that the essential thing is to have the correct concentration of HCl present in any given case; if the concentration of HCl is too high some of the creatinine will be destroyed, while if it is too low complete conversion will not occur. Weber[54] gives 3 per cent. HCl as the optimum concentration. In the experiments of Graham and Poulton mentioned above the concentration of HCl was 1·2 per cent. This is amply sufficient for estimating accurately moderate amounts of creatine, if the urine is heated for five hours as Mellanby advises instead of three hours.

In dilute urines where it may be necessary to take 15 c.c. urine to get a reading in the right part of the scale, the addition of half the volume o $\frac{N}{1}$ HCl and its subsequent neutralisation will cause too great a dilution for accurate work. In such a case there would seem to be no objection to adding a small amount of strong HCl, so calculated as to produce the requisite percentage concentration of acid in the urine.

Benedict and Myers[3] have introduced a modification which enables the conversion of creatine into creatinine to be carried out in a quarter of an hour. The urine is heated with HCl in the autoclave under increased pressure. They have shown that this method gives accurate results. However, the same precaution as regards dilutions should be carried out as with the water-bath method.

II.—Sources of Error in the Method.

When Folin first described his colorimetric method he stated that the presence of aceto-acetic acid (diacetic acid), aceto-acetic ethyl ester, acetone, and hydrogen sulphide in the urine completely upset the determinations.

Taylor[52] has found that in addition to these substances, bile pigments and excessive amounts of normal urochromes may have a very disturbing effect.

Folin and Denis[13] have recently observed that both muscle and blood cause an error in creatinine estimations, and they claim that a number of experiments are vitiated from this cause. The estimation of creatinine and creatine in pathological conditions is certainly complicated by the possible presence of these various substances in urine, and the greatest caution is needed in interpreting the results. Dextrose, if not present in unreasonably large amounts, will produce no error, but if urine containing dextrose is heated in the autoclave a serious error is produced. Rose[45] describes a method of overcoming the difficulty. The simplest plan would seem to be to use the water-bath instead of the autoclave in such cases when dextrose is present, and probably no error will be introduced (Greenwald[21]).

Of the other substances mentioned the so-called acetone bodies are particularly important, as they occur in so many conditions. Acetone bodies are excreted in small amounts shortly after a normal person begins taking a diet containing no carbohydrates. They are excreted in starvation. In both cases it is reasonable to ascribe their appearance to abnormal metabolism due to the limited amount of carbohydrates available.

Among pathological conditions they may occur in diabetes, gastro-intestinal diseases, fevers, post-anæsthetic poisoning, cyclical vomiting in children, toxæmic vomiting in pregnancy, eclampsia, acute yellow atrophy, phosphorus poisoning, and perhaps scurvy.

The general conclusion drawn from a number of investigations by different workers has been that the presence of acetone bodies in the urine introduces no perceptible error in creatinine and creatine determinations.

β-oxybutyric acid was investigated by Graham and Poulton.[20] A 1 per cent. solution in urine caused no error in the determination of creatinine; with a 2 per cent. solution the error was scarcely perceptible. Such a concentration would never occur

under pathological conditions, so that the presence of β-oxybutyric acid in the urine as a disturbing factor can be neglected.

In considering acetone and aceto-acetic acid (diacetic acid), it is obvious that any possible error introduced by their presence will only affect the determinations of pre-formed creatinine. During the conversion of creatine into creatinine the urine is heated sufficiently long to destroy all the aceto-acetic acid and to drive off the acetone, so that the creatinine + creatine determinations will be correct.

Acetone, being a reducing agent, causes an orange colour with picric acid and soda, but on dilution to 500 c.c. the colour disappears, so that it is practically impossible to say that there is any difference between such a solution and a corresponding picric acid and soda solution to which no acetone has been added.

The important question is, what occurs if urine containing acetone is treated in the same way. Van Hoogenhuize and Verploegh[24] stated that the presence of acetone caused the colour with picric acid and soda to be darker than normal immediately after dilution, but after a few minutes the colour gradually faded and finally gave a correct reading. Hence the acetone produced no error if successive readings were taken until the readings were constant. A similar result was obtained by Rose[45] and Krause,[28] and it has been generally accepted.

However, Klercker's conclusion[26] was entirely the opposite. He found that the addition of acetone made the colour too light, and as it would be necessary to use a greater thickness of liquid to match the bichromate solution, the readings of the colorimeter would be too high and the amount of creatinine found would be too little.

From experiments by Graham and Poulton there does not seem to be any doubt that Klercker was right. The addition of acetone does produce too high a reading, and the error is quite marked if a 1 per cent. solution of acetone is used, but is negligible with a 0·2 per cent. solution. As a 1 per cent. solution would correspond to an excretion of 15 grms. acetone in the day if the volume of urine is 1,500 c.c., it is only in extreme cases of acidosis, if at all, that any error will arise from this cause.

Aceto-acetic acid, owing to its reducing action, causes a similar colour to acetone when treated with picric acid and soda. Its action when present in urine has been studied on several occasions, and sometimes not very satisfactorily. Rose,[45] Wolf and Osterberg[56] used the ethyl ester instead of the acid. The very great difference between the chemical characteristics of these two substances need hardly be dwelt on here, or the fact that the ester does not break up into ions in solution. Krause,[28] who used aceto-acetic acid, found that it made the colour darker than usual, and drew the conclusion that this error would give the impression that less creatine was excreted than was really the case. This result has not been confirmed by recent work.

Greenwald,[21] when working on diabetic urines in March, 1913, again tested the effect of aceto-acetic acid on Folin's colorimetric method. He found that quite small quantities absolutely upset the results, the colour produced with picric acid and soda being made much lighter than normal. This error would produce the impression that less creatinine had been excreted than was actually the case. It has already been mentioned that there would be no error in the creatinine + creatine determinations owing to the decomposition of the aceto-acetic acid, and so, even if there was no creatine really present, the creatinine +creatine would be greater than the amount of pre-formed creatinine apparently found, and the difference between the two would be ascribed to the presence of creatine.

However, this error might not have been considered of much importance, because until recently it was believed that aceto-acetic acid was only excreted in pretty severe states of acidosis, while acetone produced practically no error.

Recent work has completely altered our views as to the relative importance of aceto-acetic acid and acetone in urine. Arnold, Emden, Folin, and Hurtley[25] have conclusively shown that in acidosis fresh urine contains practically no acetone at all; it is all present as aceto-acetic acid (diacetic acid), or rather, a salt of aceto-acetic acid, and this body is only slowly decomposed on standing.

Rothera's modification of Legal's test was generally regarded as the most delicate test for acetone. This consists in adding 5 grms. $(NH_4)_2 SO_4$ and three drops of a freshly-prepared 5 per cent. sodium nitroprusside solution to 10 c.c. urine, mixing and subsequently adding 2 c.c. strong ammonia. A purple colour develops. Hurtley has shown that this is a far more delicate test for aceto-acetic acid than it is for acetone. Hence, a so-called + test for acetone in clinical medicine means the certain presence of aceto-acetic acid, with or without acetone, even if the ferric chloride reaction is negative.

In a ten days' experiment on a creatine-free diet of low calorie value, and containing no carbohydrates, Graham and Poulton[18] found that no creatine was ever excreted in the urine, though the acidosis was marked. This was completely opposed to the work of Cathcart[7] and others who have repeatedly found creatine under these conditions. Greenwald's observation suggested the possibility that this discrepancy might be due to errors from the presence of aceto-acetic acid in the ordinarily accepted work on creatinine and creatine in these conditions. As Greenwald's results were opposed to those of Krause, it was first of all necessary to see if they could be confirmed. Consequently Graham and Poulton[20] added the sodium salt of aceto-acetic acid in different percentages to urine and made creatinine determinations for each concentration of aceto-acetic acid. The addition of the aceto-acetic acid produced in each case a lighter colour than was obtained with the urine alone, so that there was an apparent diminution in the amount of creatinine in the urine.

In Fig. I. varying percentages of aceto-acetic acid are plotted out against the corresponding errors in the creatinine determinations. The curve approximates to a straight line, so that the error produced is roughly proportional to the concentration of aceto-acetic acid present. There is some evidence that this curve remains the same if the actual amount of creatinine in the original urine is varied. The urine with which the above experiment was made contained 0·116 grms. creatinine per 100 c.c.

These results demonstrate in a very striking fashion that Greenwald was right and that Krause's work was incorrect.

It is clear that this altered colour reaction is not brought about indirectly by the interaction of some other urinary constituent, because the same effect is produced if aceto-acetic acid is added to a solution of creatinine in distilled water. In all urines containing acetone bodies it is essential to remove the aceto-acetic acid before making creatinine determinations.

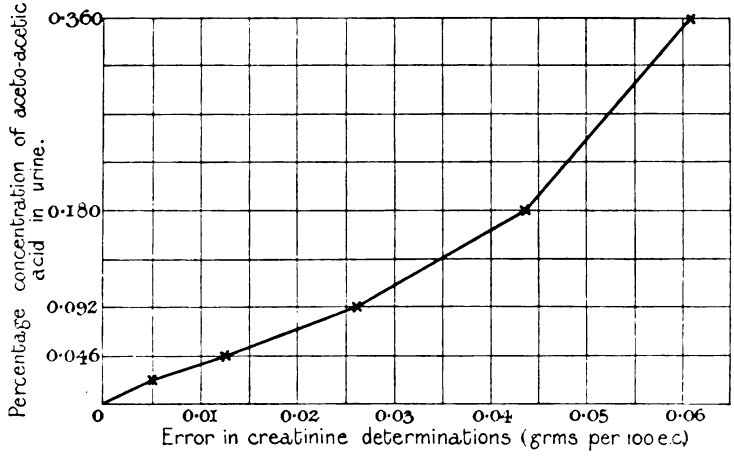


FIG. 1.

The following method described[18] and subsequently modified[19] has been found quite satisfactory for this purpose. The urine (10 c.c.) is measured accurately into a boiling tube fitted with a rubber stopper and 1 c.c. of a 10 per cent. phosphoric acid solution is added. Through the rubber cork are passed two tubes. The first is drawn out to a fairly fine point and dips beneath the surface of the urine; the second connects the boiling tube with a receiver which is, in turn, connected with a filter pump. The boiling tube is placed in a water bath kept between 65° C. and 70° C. The pressure is diminished by means of the pump to about 250 mm., and a stream of air is allowed to bubble through the liquid. Very little liquid is distilled off in the process, so that practically no concentration of the urine takes place.

By this means all the aceto-acetic acid is removed in three-quarters of an hour if present in amounts up to 0·2 per cent. If larger quantities are present the process may be continued for an hour and a half. In cases where an unknown amount of aceto-acetic acid is present it will be safest to heat another specimen of urine in the same way, and test for the presence of aceto-acetic acid at various intervals by the extremely delicate Rothera's reaction.

In order to find out if creatine was broken down by this method, weighed amounts of pure creatine were added to urine. In no case was any increase in the creatinine readings obtained after distillation. The largest amount of creatine added was 0·2 grm. per 100 c.c., which is much more than would be ever likely to occur in urine.

Greenwald has described a method for removing aceto-acetic acid by extracting with ether for two hours, and subsequently blowing away the ether. This method is much more complicated than the one just described, and suffers from the serious defect that the urine must be considerably diluted in the process. This dilution may lead to uncertain results.

III.—Is Creatine excreted in Acidosis?

Owing to the work of Cathcart[7] and Mendel and Rose[38] there has been general agreement that creatine is always excreted, when acidosis is produced in man, by withdrawing carbohydrates from the diet. The possibility that this general conclusion was wrong was indicated by the ten days' experiment of Graham and Poulton on a fat diet mentioned above. The point was of such interest that it has been further tested by Graham and Poulton,[20] special care being taken to avoid the error due to the aceto-acetic acid.

Experiments were carried out on three different individuals. Limited quantities of fat, with or without protein, were taken on the days of the experiments, and the urine was analysed quantitatively for creatinine and creatine. The various results are shown in six columns in Table I. In the first three columns

TABLE I.

No. of Experiment.	Subject of Experiment.	Day of Experiment.	Creatinine + Creatine (calculated as Creatinine).	Apparent Creatinine (Folin's Method).	Apparent Creatine.	Creatinine + Creatine (calculated as Creatinine).	True Creatinine (Distillation Method).	True Creatine.	Aceto-acetic Acid.
			Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
I.	E. P. P. ...	1	1·80	1·82	0	1·80	1·78	0	—
		2	1·82	1·58	0·24	1·82	1·81	0	0·87
		3	1·72	1·42	0·30	1·72	1·72	0	0·87
II.	G. G. ...	1	1·53	1·46	0·05	1·53	1·52	0	0·30
		2	1·43	1·21	0·22	1·43	1·43	0	1·06
		3	1·53	1·17	0·35	1·53	1·52	0	1·46
III.	M. D.	1	2·05	1·93	0·11	2·05	2·03	0	Rothera's test positive.
		2	2·14	2·02	0·15	2·14	2·15	0	"
		3	2·25	2·09	0·15	2·25	2·27	0	"

are placed the results obtained when no precautions were taken to remove the aceto-acetic acid. The first column gives the creatinine + creatine estimation, and the second column gives the *apparent* pre-formed creatinine obtained by applying Folin's method directly to the urine, *i.e.*, without removing the aceto-acetic acid. The difference between the two columns gives the *apparent* creatine (third column). In the fourth column the creatinine + creatine figures are repeated. The fifth column gives the *true* pre-formed creatinine obtained after removing the aceto-acetic acid. The *true* creatine (column six) is the difference between these two columns.

From the first three columns it is obvious that, if the aceto-acetic acid is not removed, there is an apparent diminution in the excretion of creatinine, and at the same time creatine was apparently excreted. The last three columns show the true state of affairs. The amount of creatinine excreted remained practically unchanged throughout each experiment, and in reality there was never any creatine excreted at all.

This conclusion is strengthened on comparing these results with the following two experiments of Cathcart's :—

TABLE II.

Day of Experiment.	Diet.	Creatinine + Creatine.	Pre-formed Creatinine.	Creatine.
		Grams.	Grams.	Grams.
1	Fast ($+H_2O$) ...	1·28	1·14	0·14
2	Fat	1·38	1·00	0·38
3	"	1·28	0·95	0·35
1	Fast ($+H_2O$) ...	1·13	1·02	0·11
2	Fat	1·15	0·87	0·28

The figures are very similar to those in the first three columns of Table I. The pre-formed creatinine figures diminished as the result of taking fat, and the creatine figures showed a gradual rise, and the actual amounts of creatinine and creatine obtained by Cathcart are very similar to the *apparent* creatinine and *apparent* creatine in Graham and Poulton's experiments. They

can thus be explained by the error due to the presence of aceto-acetic acid in the urine which has been recently discovered. Further, Cathcart notices that as soon as carbohydrates are taken, the creatine excretion disappears. This will be accounted for by the arrest of the acidosis. There is one point of difference between Cathcart's experiments and those of Graham and Poulton. In the former case the subject fasted for the first twenty-four hours. In the latter case there was no absolute fast. However, on the first day of Experiment 1, E. P. P. only took half a pint of cream, so that for all practical purposes the experiments were identical.

The conclusion can be drawn that the excretion of creatinine remains unaltered as a result of withdrawing carbohydrates from the diet, even if the experiment lasts ten days, and that no creatine is ever excreted under these conditions.

IV.—Theories of the Origin of Creatinine.

The interest in the metabolism of creatine and creatinine lies in their close chemical relationship to one another, and in their limited occurrence in nature. Creatine is present in the voluntary muscle of vertebrates. Although the voluntary muscle of invertebrates, such as the lobster, resembles vertebrate voluntary muscle in the existence of cross-striation, and this cross-striation has probably a similar function in both cases, the invertebrate muscle never contains creatine. However, there is evidence[34] that among vertebrates creatine is not invariably present in striped muscle. In the developing chick there is muscle on the twelfth day of development, but the muscle contains no creatine. During incubation the creatine appears, and immediately after hatching the growth of the liver and the creatine content of the muscles both increase rapidly together. Creatine does not occur in the urine of adults on a meat-free diet under normal conditions.

Creatinine, on the other hand, is never found in normal muscle, but appears exclusively in the urine. Further, Folin has shown that in health the creatinine output in the urine is a

constant quantity from day to day, but different individuals excrete different amounts. This fact has been verified by Klercker,[26] van Hoogenhuize and Verploegh,[24] and many others. The amount of creatinine is absolutely independent of the total nitrogen in the urine,[11, 26] remaining the same when the subject excreted 16 grms. and 2·25 grms.[18] In other words, it is independent of the amount of protein in the food.

Folin considers that it is the result of endogenous metabolism, and the amount of creatinine excreted offers a measure of the endogenous metabolism of the individual. The same constant output is noticed among women, but the amount is rather less than in men.

Schaffer[49] further states, from one experiment, that the creatinine excretion is also constant from hour to hour. This is opposed to the results of Klercker, van Hoogenhuize and Verploegh, and Leathes.[31]

The very natural conclusion has been drawn from these various considerations that the creatinine of the urine is formed directly from the creatine of the voluntary muscles, and support is given to this supposition by the ease with which creatine can be converted into creatinine in the test-tube by simply boiling, preferably with the addition of a little hydrochloric acid.

A large number of experiments have been designed to find out if this conversion ever occurs in the body. The experiments have been of three kinds :—

A. Feeding experiments.
B. Autolytic experiments.
C. Experiments on muscular work.

A. Folin[10] showed that with a normal individual on an ordinary meat diet, small quantities of creatine occurred in the urine.

If Liebig's extract, which contains both creatinine and creatine, is taken, more or less creatine always appears in the urine, and there is also an increase in the creatinine. At the same time, by no means all the creatine that has been taken by the mouth is recovered in the urine. The body seems to find it

easier to excrete creatinine than creatine. There is no apparent conversion of creatine into creatinine under these conditions, and so Klercker[26] drew the conclusion that there was no biological connection between the two substances.

Peckelharing and van Hoogenhuize[41] found creatine in the urine after injecting it into animals. Folin gave 1 grm. creatine to healthy men on a low nitrogen diet; it was completely used up, none appearing in the urine. If 5—6 grm. creatine were given, a certain percentage did appear in the urine, and this percentage was greater if the diet contained more nitrogen. The possibility that this experiment suggests, is that in nitrogen starvation the creatine was utilised as a foodstuff, a conclusion very different from the prevailing views as regards the value of meat extracts. Mellanby suggested that the disappearance of the creatine might be accounted for by bacterial action, and lately he and Twort[37] isolated an anærobic Gram + bacillus from the human intestine which has a marked power of destroying creatine. This gives a satisfactory explanation as to why, when creatine is given in the food, only part of it is recovered in the urine, and there is no evidence that when given in this way it undergoes conversion into creatinine. Thus, feeding experiments have yielded negative results.

B. Seemann,[48] Gottlieb and Strangassinger,[15 16 17] and later Rothmann from Gottlieb's laboratory,[46] all stated that on autolysis of muscle with antiseptic precautions, a conversion of the muscle creatine into creatinine took place. They also found that by passing warm defibrinated blood through isolated organs similar changes occurred particularly in the liver and kidneys. They inferred that many tissues contained ferments, creatase, and creatinase, which brought about these various conversions.

However, Mellanby[34] has performed various autolytic experiments, and in no single respect confirms the presence of ferments, and has never found any conversion of creatine into creatinine if precautions were taken to prevent bacterial action. He states that the outstanding fact is the stability of muscle creatine. Lepmann[33] has confirmed Mellanby's results.

In trying to decide between these opposing results it is necessary to consider the ease with which creatine breaks up into creatinine under all sorts of conditions, and the fact that the whole estimation is an indirect colorimetric measurement, which also is liable to all the errors mentioned in the earlier part of this paper, as well as the error due to presence of muscle products mentioned by Folin and Denis.[13] Taking these things into consideration, when there are two series of experiments entirely opposed to one another, and with no obvious fallacy to account for the differences, it will probably be correct to take the negative result as being right.

C. Weber[54] perfused a working heart with Ringer's solution, and found that after heating the perfusing fluid with HCl, creatinine was present; there was no alteration in the creatine content of the heart. In another case cinchonine convulsions caused an increase in the output of creatinine, while when one sciatic nerve of the cat was divided, and the muscle analysed after some weeks, the creatine content was found to be less than on the other side.

However, Mellanby[34] in some very detailed experiments on isolated muscles has shown that no conversion of creatine into creatinine takes place in muscular work, and further, that the amount of creatine in muscle is entirely unaffected by the work. Here, again, it will probably be more correct to assume that Mellanby's negative results are right.

Some work recently carried out by Rübsamen and Gusikoff[47] throws some light on the errors incidental to these experiments on isolated muscles. A uterus was kept alive and made to do work in a vessel containing warm oxygenated Ringer's solution. Creatine was obtained, although less in amount than that obtained by Weber with the working heart, because, as it was pointed out, less work was done by the uterus than by the heart. The authors considered that the creatine arose from the uterine muscle in the same way as it arose from the heart muscle in Weber's experiment. However, according to Mellanby,[36] the uterus consists of plain muscle, and does not contain any creatine at all.

Mellanby's results have been confirmed by experiments on man. Peckelharing[40] and Schaffer[49] found that muscular work produced no creatine in the urine and no increase in the creatinine excretion. Spriggs[51] found that in two cases of tetanus the creatinine excretion was not above normal.

Lately Peckelharing[40] has suggested another possibility, viz., that the creatinine of the urine arises from body muscle as the result of muscular tone, and not from muscular work. A student taking a definite diet held himself erect in the military position for four hours with tense muscles. The creatinine was normally 60·6 mgms. per hour; it rose to 75·6 mgms. on the tonus days. A four hours' walk made no difference. This increase may have been due to partial asphyxiation of the muscles.

The general result of all these experiments is that there is no proof that muscle creatine is ever converted into creatinine in the body; as far as the evidence goes, it must be taken as showing that this conversion probably never occurs. Mellanby points out the improbability of the body's transforming such an innocuous substance as creatine into such a strongly basic substance as creatinine.

However, in spite of this evidence it is quite possible that the creatinine of the urine may be derived from muscle, independently of its creatine content. This is the view put forward by Spriggs[51] and later by Schaffer.[49] The latter regards the creatinine of the urine as due to a special process of metabolism bound up with muscular processes and depending on the nutrition of the muscle, and not on muscular activity or muscle tone. The amount of creatinine excreted by the individual will depend on his muscular development. The creatinine co-efficient, which is a constant for any individual, is the amount of creatinine nitrogen in milligrammes per kilo. of body weight. In man this value ranges from 7—11, and is less, according to Schaffer, in individuals with poorly-developed muscles or with more than the normal amount of adipose tissue.

Mellanby's view[34] is completely different. In certain diseases of the liver, such as cirrhosis, engorgement, carcinoma, he found

that the creatinine excretion was low. He suggests that under normal conditions creatinine is formed in the liver from the various substances brought in the blood stream from the different parts of the body, and that this creatinine is transferred to the muscles and deposited there as creatine up to a certain saturation point. When saturation has been reached creatinine is excreted unchanged in the urine.

V.—The Excretion of Creatine in Physiological Conditions.

Creatine is not excreted normally in adults. It is true that Krause[29] disputes this statement when he says that creatine normally occurs in the urine of women at different times in the sexual cycle, but the amounts of creatine found by him are irregular, and often small enough in amount, to be put down to occasional errors in the determinations. His results are opposed to those of other workers.[23] [22]

However, creatine does appear to be a normal constituent of the urine in children up to the age of 13—15 years. This was shown by Rose,[44] and has been confirmed by Folin and Denis.[12] Some of the children had always been brought up on a vegetarian diet.

Creatine also appears to be excreted in normal pregnancy. Heynemann[22] confirmed this result with some very careful experiments. He does not believe in the suggestion of van Hoogenhuize that the excretion of creatine in these normal cases is due to a partial derangement of the liver functions.

Heynemann also showed that creatine occurs in the urine in lactation. Mellanby[36] points out that this appearance has nothing to do with the involution of the uterus, as, among other things, the latter contains no creatine. He considers that it is connected with the active production of milk.

VI.—The Excretion of Creatinine and Creatine in Pathological Conditions.

A large number of empirical facts have been stated with regard to the excretion of creatinine and creatine in pathological

conditions. So far there is no satisfactory theory for linking these various facts together, and this anomalous state of things has been commented on by various authors.

According to the prevalent view, that creatinine arises in the normal metabolism of muscle, it is comparatively easy to imagine that in many pathological conditions a wasting, destruction, or utilisation of muscle tissue occurs, and that in these conditions there is an excretion of unaltered creatine due to abnormal muscular metabolism. This is the view advocated by Schaffer, and it has been used to explain the excretion of creatine in such widely different conditions as starvation, fever, carcinoma of the liver, and diabetes.

There seems to be no doubt that there was acidosis present in many of the pathological conditions in which creatine has been found, and, owing to the error due to aceto-acetic acid, many of these results will require revision. It is quite possible that in future it will be found that the excretion of creatine does not occur by any means so frequently as is generally supposed at the present time.

It will be of interest to consider briefly some of these pathological conditions, and to see what evidence there is for any abnormal behaviour in the excretion of creatinine and creatine.

Starvation.—The view held at present is that the output of creatinine diminishes in starvation and that creatine appears in the urine; the latter fact is usually explained by the breaking down of muscle tissue. In Cathcart's starving man[6] about 0·3 grm. creatine was excreted on the second day, and about this amount was excreted on the other days of the experiment, except on the fourth day when the excretion rose to 0·44 grm. The creatinine output was low. The creatine disappeared at once on taking a diet of starch and cream.

In comparing these figures with those for *apparent* creatine in Table I. it will be seen that, except for the fourth day in Cathcart's case, the figures for the creatine were about the same in the two cases. It would be unlikely that the amount of acidosis would be any less in the starvation case than that which occurred

in Graham and Poulton's experiments, so that there is considerable probability that all the creatine in starvation was due to the error of the aceto-acetic acid. If this is the case, the figures for the creatinine + creatine will give the *true* pre-formed creatinine values. The behaviour of the pre-formed creatinine deduced in this way is interesting. The normal value for the individual was about 1·36 grm. As the result of starvation, it gradually fell to 0·9 grm. During this time the nitrogen excretion fell from 16·45 grms. to 7·8 grms.

In Benedict and Diefendorf's case[1] the amount of creatine was no greater than in Cathcart's case. Its presence is probably explicable on the same hypothesis. The *true* pre-formed creatinine output, deduced as above, seemed to be fairly constant throughout the fast.

Artificially produced high temperature (heat stroke).—In three experiments by Graham and Poulton[18] a high body temperature was produced artificially in man by means of a steam bath. In the first two experiments a diet was taken containing a very high carbohydrate content. In one case a rectal temperature of over 100·4°F. was maintained for three hours and three quarters, and in the other case a rectal temperature of over 104° F. was maintained for an hour and a quarter. In neither experiment was the output of nitrogen increased, and there was also no alteration in the amount of creatinine excreted. In the last experiment a diet was taken deficient in calorie value and containing no carbohydrates. A rectal temperature of over 103° F. was maintained for two hours. There was no increase in the output of nitrogen or creatinine.

Recently Myers and Volvic[39] raised the temperature of three rabbits artificially for periods of two or three days. There was an increase both in the nitrogen and the creatinine output. It seems reasonable to consider that this rise in the nitrogen output was probably due to the utilisation of protein owing to the increased energy production set up by the high temperature, as originally explained by F. Voit[53] and later by Graham and Poulton,[18] and that it was not due to the effect of the high

temperature itself breaking down the body protoplasm. Similar results were obtained by Richter[43] and Formanek[14] and are to be explained in this way.

The rise in the creatinine output, running parallel with the total nitrogen excretion, may, perhaps, be considered as due to an increase in the endogenous protein metabolism.

Fever.—Leathes[32] showed that the output of creatinine in the urine was increased in fever produced by injecting an anti-typhoid vaccine into man. This rise of temperature only lasted a few hours, and it was during these few hours that the increase of creatinine occurred. This result has been confirmed by a number of observers—van Hoogenhuize and Verploegh,[24] Klercker,[27] Schaffer and Coleman,[50] Ewing and Wolf,[9] Wolf and Lambert,[55] particulary in cases of pneumonia and typhoid. The excretion of creatinine was almost invariably increased during the pyrexia, and in many cases ran parallel with the increased output of total nitrogen. During convalescence both the total nitrogen and the creatinine diminished to the normal level. Klercker noticed in cases of pneumonia, where a rise in the total nitrogen output occurred after the crisis, due to absorption of the exudate, that there was no corresponding increase in the creatinine.

Most of these observers have also found that creatine was excreted during the febrile period and sometimes in convalescence. In typhoid cases Schaffer and Coleman found that as much as 0·56 grm. creatine was excreted in one day, but the excretion was usually much less than this. Acidosis was often present in these cases, as during certain periods of the experiments the diet contained little carbohydrate. At other times very large amounts of carbohydrate were given which had the effect of practically arresting the nitrogen loss, and at the same time the amount of creatine was diminished or disappeared altogether. They ascribed this diminution of creatine to the action of the carbohydrate preventing the destruction of muscle protoplasm. The recently-discovered error, due to aceto-acetic acid, suggests the possibility that very little if any creatine was really

excreted, especially as, in most cases, the amounts of creatine obtained were very similar to those excreted in Graham and Poulton's experiments already described.

If this is the case the *true* pre-formed creatinine can be calculated by adding the amount of creatinine and creatine together, and in most cases it will be found that the values so obtained during pyrexia are very distinctly greater than during convalescence. In this way Leathes' original conclusion is confirmed. There can be no doubt that in fever the output of creatinine is increased.

Diseases of Pregnancy.—Van Hoogenhuize and ten Doesschate[23] found that the excretion of creatinine in eclampsia was high, while considerable amounts of creatine appeared in albuminuria and eclampsia. Heynemann[22] distilled the urine to get rid of the acetone, and found that creatine was excreted in eclampsia. Ewing and Wolf[9] found very low values for the creatinine output in toxic vomiting, results that may have been due to the presence of aceto-acetic acid.

Cyclical Vomiting.—Mellanby[35] described a case of cyclical vomiting in a child six years old. The child excreted creatine during the whole time the observations were carried out. This was considered by Mellanby to indicate abnormal metabolism; however, it has been shown recently that creatine is a normal constituent of the urine of children. During the attacks of vomiting acidosis was noticed, and the amount of creatine in the urine rose. This could obviously be explained by the presence of aceto-acetic acid. Mellanby states that the creatine rose before the acidosis came on. The amount of creatine certainly varied to some extent beforehand, but these variations lose their significance if the excretion of creatine in childhood is a normal phenomenon. The probability is that in cyclical vomiting there is no abnormal excretion of creatine.

Exophthalmic Goitre.—In acute cases, Schaffer[49] noticed that the excretion of creatinine was low and that creatine appeared in the urine, but not in very large amounts. These cases support his argument that creatinine depends on metabolism of

muscle and not on the total endogenous metabolism which is probably increased in this disease.

Diseases of the Liver.—Mellanby[34] described six cases of cirrhosis of the liver, two cases of engorged liver from mitral stenosis, and two cases of carcinoma of the liver. In all these cases the creatinine was diminished, and in the carcinoma cases large quantities of creatine were found.

Van Hoogenhuize and Verploegh[24] did not find much diminution of creatinine in cirrhosis of the liver, but in carcinoma they obtained a similar result to Mellanby. All these experiments are of importance, as considerable theoretical conclusions have been drawn from them, viz., that the liver is intimately connected both with creatinine and creatine metabolism. This is the conclusion of Boljaiskij,[5] who investigated cases of carcinoma, cirrhosis, abscess, and wounds of the liver.

Mellanby prefers to believe that the creatine in carcinoma cases comes from the wasting of the muscles due to cachexia, and it is only the creatinine metabolism that is intimately concerned with the liver.

Diabetes.—Dreibholtz[8] and Krause and Cramer[30] have investigated creatinine and creatine in diabetes. They state that creatine is always present, even if there is no marked acidosis present. All the experiments on diabetes are liable to the error due to the presence of aceto-acetic acid. Then there is the difficulty of the dilution of the urine, which may be a serious factor in cases where three or more litres of urine are excreted daily. Dreibholtz thought that the amount of creatinine excreted in the day was not less than usual.

Nervous and Muscular Diseases.—Benedict and Myers[4] investigated a number of women in an institution for the insane. In certain cases creatine was excreted. The creatinine output was rather low. Spriggs[51] found that the normal amount of creatinine was excreted in cases of locomotor ataxy, general paralysis, and spastic paraplegia. He also found a very low creatinine co-efficient in two cases of muscular dystrophy and in one case of amyotonia congenita, and a rather low co-efficient in my-

asthenia gravis. He argues that creatinine is connected with the nutritional metabolism of the muscle fibre and is not a substance formed in the act of contraction.

VII.—Summary.

In the present state of our knowledge none of the theories of creatinine metabolism are entirely satisfactory. The one definite thing seems to be that creatinine is a product of endogenous nitrogen metabolism, as suggested by Folin, and has no relation to the nitrogen of the food.

There are certain facts that agree with such a theory. There is the rise in creatinine which occurs in fever and which runs parallel with the rise of nitrogen. The rise of nitrogen, which presumably indicates an increase in the endogenous nitrogen metabolism of the body, is regarded partly as toxic in origin and partly as a utilisation of protein to supply the increased energy requirements of the body. The rise in creatinine may have a similar cause.

Again, one of the effects of starvation is to cause a gradual diminution in the nitrogen output during the first few days, and at the same time the creatinine excretion diminishes. This diminution in the nitrogen output is no doubt chiefly brought about by the gradual disappearance from the body of nitrogen of exogenous origin, but at the same time, there is probably also a diminution in the general endogenous nitrogen metabolism, and the lessened excretion of creatinine may be due to this cause.

There is still the question as to whether the production of creatinine takes place in the liver from substances conveyed in the blood stream. The observations on liver diseases certainly indicate this possibility.

According to the views of Spriggs and Schaffer, the origin of creatinine is limited to the nutritional processes of muscle. The observations of Spriggs that in chronic muscular diseases the creatinine excretion is very low, offers most significant support to this view. On the other hand, it is difficult to reconcile

the increase in the creatinine output in fever with this view, unless it is considered that the increased endogenous metabolism in this condition arises almost entirely in the muscles. This, of course, may be the case. At present the balance of evidence is in favour of this view, and it is also possible that the liver may be concerned in the final elaboration of creatinine from the muscles before it is excreted.

In this paper some stress has been laid on the fact that there is no evidence that the creatine of muscle is ever excreted in the form of creatinine, and if the creatinine of the urine arises from the muscles the creatine probably takes no part in this formation. Creatine in muscle may well be an end product from creatinine metabolism, as Mellanby has suggested. The suggestion has also been made in this paper that in a large number of pathological conditions the apparent excretion of creatine is probably due to an error in analysis. However, in those cases where this explanation will not apply, the origin of the urinary creatine can only be regarded as at present unknown. The most obvious explanation would be to attribute it to some abnormal process in muscle; on the other hand, in one case Mellanby has connected it with an entirely different process, viz., lactation.

REFERENCES.

1. Benedict and Diefendorf, Am. Jour. Physiol., 18, 362, 1907.
2. Benedict and Myers, Am. Jour. Physiol., 18, 377, 1907.
3. Benedict and Myers, Am. Jour. Physiol., 18, 397, 1907.
4. Benedict and Myers, Am. Jour. Physiol., 18, 406, 1907.
5. Boljaiskij, Zentralbl. f. d. gesammt, Phys. u. Path. d. Stoffwechsels, 6, 413, 1911.
6. Cathcart, Jour. Physiol., 35, 500, 1906-7.
7. Cathcart, Jour. Physiol., 39, 311, 1910.
8. Dreibholtz, Diss. Greifswald, 1908.
9. Ewing and Wolf, Arch. of Int. Med., 4, 330, 1909.
10. Folin, Zeit. Physiol. Chem., 41, 223, 1904.
11. Folin, Am. Jour. Phys., 13, 66, 1905.
12. Folin and Denis, Jour. Biol. Chem., 11, 253, 1912.
13. Folin and Denis, Jour. Biol. Chem., 12, 141, 1912.

14. Formanek, Sitzungsber d. k. k. Akad. d. Wiss. Math. Nat., Wien., Abt. III., 101, 278, 1892.
15. Gottlieb and Strangassinger, Zeit. Physiol. Chem., 52, 1, 1907.
16. Gottlieb and Strangassinger, Zeit. Physiol. Chem., 55, 295, 1908.
17. Gottlieb and Strangassinger, Zeit. Physiol. Chem., 55, 322, 1908.
18. Graham and Poulton, Quart. Jour. Med., 6, 82, 1912.
19. Graham and Poulton, Jour. Physiol., 46, Proceedings, xliv.
20. Graham and Poulton, Proc. Roy. Soc., B., 1913 or 1914.
21. Greenwald, Jour. Biol. Chem., 14, 87, 1913.
22. Heynemann, Zeit. f. Gebürtshilfe, 71, 110, 1912.
23. Van Hoogenhuize and ten Doesschate, Annales de Gynecol. et l'Obstetrique, Jan., Feb., 1911.
24. Van Hoogenhuize and Verploegh, Zeit. Physiol. Chem., 57, 161, 1908.
25. Hurtley, Lancet, 184, 1160, 1913.
26. Klercker, Bioch. Zeit., 3, 45, 1907.
27. Klercker, Zeit. f. Klin. Med., 68, 22, 1909.
28. Krause, Quart. Jour. Exp. Physiol., 3, 298, 1910.
29. Krause, Quart. Jour. Exp. Physiol., 4, 293, 1911.
30. Krause and Cramer, Jour. Physiol., 40, lxi., 1910.
31. Leathes, Jour. Physiol., 35, 125, 1906.
32. Leathes, Jour. Physiol., 35, 205, 1906.
33. Lepmann, Zeit. Physiol. Chem., 57, 475, 1908.
34. Mellanby, Jour. Physiol., 36, 447.
35. Mellanby, Lancet, 2, 8, 1911.
36. Mellanby, Proc. Roy. Soc., B86, 88, 1913.
37. Mellanby and Twort, Jour. Physiol., 44, 43.
38. Mendel and Rose, Jour. Biol. Chem., 10, 213, 1911-12.
39. Myers and Volvic, Jour. Biol. Chem., 14, 489, 1913.
40. Peckelharing, Zentralblatt f. Exp. Med., 1, 72, 1912.
41. Peckelharing and van Hoogenhuize, Zeit. Physiol. Chem., 64, 262, 1910.
42. Peckelharing and van Hoogenhuize, Zeit. Physiol. Chem., 69, 395, 1910.
43. Richter, Virchow's Archiv. f. Path. Anat. u. Physiol., 123, 118.
44. Rose, Jour. Biol. Chem., 10, 265, 1911-12.
45. Rose, Jour. Biol. Chem., 12, 73, 1912.
46. Rothmann, Zeit. Physiol. Chem., 57, 131, 1908.
47. Rübsamen and Gusikoff, Arch. f. Gynäkol., 95, 461, 1911.
48. Seemann, Zeit. f. Biol., 39, 333, 1907.
49. Schaffer, Am. Jour. Physiol., 23, 1, 1908.
50. Schaffer and Coleman, Arch. of Int. Med., 4, 538, 1909.
51. Spriggs, Quart. Jour. Med., 1, 1, 1907.
52. Taylor, Jour. Biol. Chem., 9, 19, 1911.
53. Voit, Sitzunsber. d. Ges. f. Morph. u. Physiol., München, 1895, 120.
54. Weber, Arch. f. Exp. Path. u. Pharm., 58, 93, 1907.
55. Wolf and Lambert, Arch. of Int. Med., 5, 406, 1910.
56. Wolf and Osterberg, Am. Jour. Physiol., 28, 71, 1911.

A CASE OF SPLENOMEGALIC POLYCYTHÆMIA OR ERYTHRÆMIA.

By

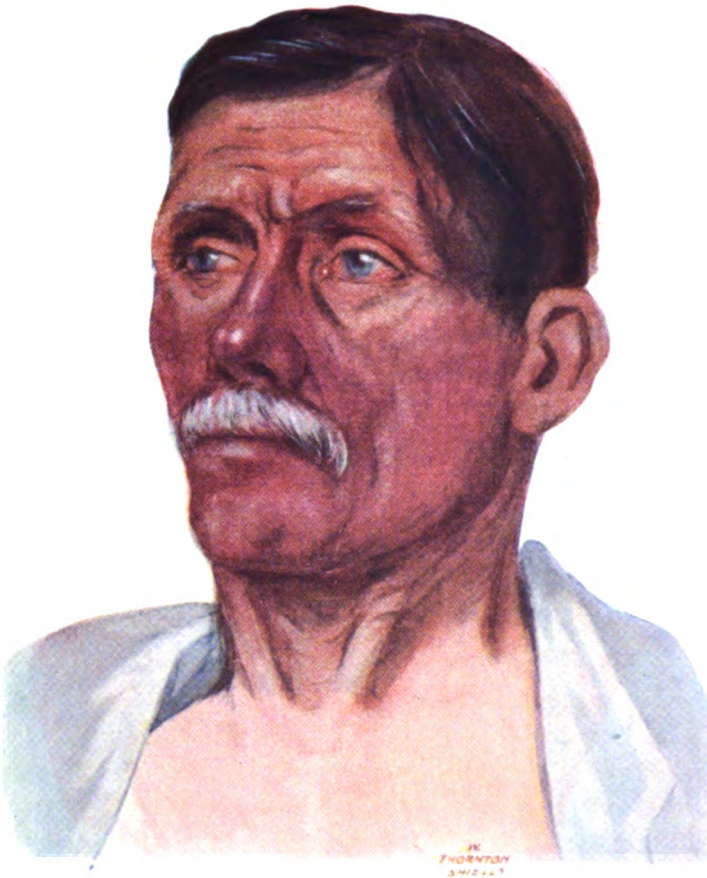
LAURISTON SHAW AND HERBERT FRENCH.

THERE are already a large number of cases of typical splenomegalic polycythæmia on record and much of the literature upon the subject has been collected in an article by Dr. F. Parkes Weber entitled "Polycythæmia, Erythrocytosis, and Erythræmia," in the *Quarterly Journal of Medicine* for October, 1908, page 85.

The clinical features of the following case differ but little from previous descriptions of the disease; but this opportunity of recording it is taken in order to depict in the coloured plate the striking facial appearance which often accompanies the malady. When the patient, Job G., æt. 58, walked into the Out-Patient Department he seemed at the first glance to be a healthy, outdoor worker of a very weather-beaten type, but on closer inspection one was struck with the cyanotic tinge of the seemingly weather-beaten skin; moreover, he was a riveter working in the London Docks and had never been exposed to any particular external conditions likely to cause the change in his complexion. He was 58 years of age, married to a healthy wife, with seven living children—two boys and five girls; two others had died at the ages of 12 and 22 respectively of phthisis. When 18 the patient had had small-pox mildly, but he had had no other serious illness. Twenty-five years ago he had lost the sight

of his right eye as the result of injury by a piece of steel, and he now wore a glass-eye in its place. His present illness began twelve years ago when he was 46; the first thing he noticed was that his face was becoming red. At this time there were no other symptoms, but his appearance went against him in seeking employment; although he had never been more than a moderate drinker and had then for some years been a teetotaler, those to whom he applied for employment were afraid from his looks that he drank too much. The red colour of his face gradually deepened, but it was not until he was 56—ten years after he first noticed the curious alteration in his colour—that he began to develop further symptoms of which the chief were: frequent headaches situated mainly in the frontal region; attacks of giddiness occurring about two or three times a week, coming on at any time of the day or night, and often severe enough to cause him to hold on to something or to sit down; trembling attacks occurring three or four times a week, mainly between 7 and 8 a.m., and lasting about a quarter of an hour; and an increasing tendency to shortness of breath on exertion. He had also been growing gradually less acute of hearing, though this deafness was not extreme. In November, 1912, he had considerable pain in his right foot, not definitely localised to any one part; this pain came on without any injury or obvious cause, and was associated with a numbness of the foot and a sensation also of pins and needles in it; there was some swelling over the right instep, but no redness or discolouration. The pain was sufficiently severe for him to be admitted to a hospital for it, and he remained twelve days as an In-Patient, by which time the foot was apparently well again.

He was sent up to Medical Out-Patients by Dr. Floyd, of Gray's, Essex, on the 30th May, 1913, and was admitted into Clinical Ward. The immediate symptoms which led to his being sent up were as follows:—Two days previously he was helping to lift a heavy piece of iron when he felt his mouth fill with something hot, and he spat out about a tablespoonful of blood; this bleeding continued on and off for about forty hours, during



THE FACIES IN A WELL-MARKED CASE OF SPLENOMEGALIC POLYCYTHÆMIA.

which time he thinks he spat up about a pint of blood altogether. He had occasionally suffered from persistent bleeding before, once after an injury to his lip and also after extraction of teeth, when bleeding had sometimes persisted for as much as thirty-six hours. The radial arteries were markedly thickened and tortuous. The pulse was of full volume and high tension, the rate averaging from 68—80 per minute, and the respiration rate averaged 20; the blood pressure by the Riva Rocci sphygmomanometer measured 170 mm. Hg. The cardiac impulse was felt in the fifth space an inch outside the nipple line, the first sound was prolonged at the apex and the aortic second sound was markedly accentuated, but there was no bruit.

The urine was of specific gravity 1022, acid in reaction, and contained neither albumen nor sugar. The lungs seemed normal; there were no abnormal apical signs to suggest phthisis as the source of the hæmoptysis. The tongue was clean, the teeth fairly good, and there was no complaint of dyspeptic symptoms or of constipation. There was no pyrexia. The spleen could be felt as a hard mass, slightly tender, projecting an inch and a half below the left costal margin during expiration and coming proportionately further down during deep inspiration.

A blood examination showed that there were 10,000,000 red corpuscles per cubic millimeter; the hæmoglobin measured 125 per cent. by Haldane's method; the leucocytes numbered 7,500 per cubic millimeter; there was no particular abnormality in the differential leucocyte count and the red corpuscles as seen in films looked normal; there were no nucleated red corpuscles, and none exhibited poikilocytosis or material variation in size. The liver was not obviously enlarged; there was no œdema. Various forms of treatment were tried; he could not take iodide of potassium comfortably; inhalations of oxygen did not make any material difference to his colour; venesection gave him some relief. On one occasion 16 ounces were removed, upon another 22 ounces, and upon another 24 ounces; all by the short hollow needle method without incision. The patient himself was convinced of the benefit he received from copious blood-letting.

He said he felt better for two or three weeks after each venesection, but objectively there was little change in his condition, unless, perhaps, a little improvement in his colour.

He was a straightforward case of erythræmia or chronic polycythæmia with cyanosis and enlarged spleen as it used to be called. As has been stated above, the main object in recording the case is to depict his striking facies.

A CASE OF PARATHYROID INSUFFICIENCY.*

By

ARTHUR F. HERTZ, M.D.

The patient, C. H. G., a clerk, aged 47, first noticed an enlargement of his thyroid gland when he was 30. It gradually became very large, and in 1908 the greater part of it was removed. After this he remained perfectly well until August, 1910, when he was in very good health and weighed 191 lbs. He then quite suddenly became extremely depressed, very nervous and restless. He had an irresistible desire to keep moving and slept very little, walking about the greater part of the night. He was exceedingly tremulous, so that he found it difficult to write; there was a continuous fibrillary twitching of the eyelids. His general appearance was similar to that of a severe case of Graves' disease, except that his eyes were sunken instead of being prominent, and no thyroid gland could be felt. Though his appetite increased, and he ate enormously and was never sick, he lost weight rapidly, so that he was only 144 lbs. when I first saw him with Sir Bertrand Dawson on the 2nd of December, 1910. He experienced some difficulty in swallowing, the food appearing to stick in his throat; and he also had some irregular intestinal pains. The X-rays showed that the dysphagia was due to irregular spasmodic contractions of the œsophagus, and perhaps a similar condition accounted for the intestinal pain.

*The case was shown at the Medical Section of the International Congress, London, 1913.

His pulse was constantly about 120, and he was greatly troubled with palpitation; his face and neck were deeply flushed. His hair had ceased to grow, but it did not become thinner; formerly it required to be cut every ten days, and now six weeks had elapsed without any increase in length occurring; this was also noticeable to a less extent on his face. He had become completely impotent. Instead of opening his bowels once a day as before, he now passed three or four large, but otherwise normal, stools each day. His urine was scanty, but otherwise normal. The secretion of sweat was unaffected. We thought that the condition was due to disturbance in the secretion of some of the ductless glands, possibly as a result of the operation, which might have injured the parathyroid glands at the same time as the greater part of the thyroid was removed.

On being put to bed and given four pints of milk a day in addition to an ordinary full diet, his pulse became slower and his abdominal discomfort disappeared, but his weight continued to fall, till it reached 130½ lbs. on December 13th, and he found it excessively difficult to stay in bed on account of his restlessness. Dried thyroid gland aggravated the symptoms, and Moebius' antithyroid serum made him sick. He was then given large doses of opium and bromide, which had the effect of making him slowly gain weight till he was 159 lbs. on February 6th, 1911; though the pulse was slower and the restlessness and tremor less marked, his other symptoms were unaltered.

He was allowed to get up in February and return to work. His weight at once began to fall, though slowly, and in spite of continuing with the same diet and taking opium and bromide he only weighed 127½ lbs. on June 18th. He continued his work, but on that day began to take 1/10th gr. of dried ox parathyroid gland (Armour) four times daily, and four days later he discontinued the opium and bromide. He gained 6½ lbs. in the first four days, 12 lbs. the next five days, 5 lbs. the next four days, and 5 lbs. the next six days, making 28½ lbs. in nineteen days. At the same time he became stronger every day, slept better, was less restless, the tremor and dysphagia disappeared, his hair began to grow, the quantity of urine in-

creased, the stools diminished in bulk, and the pulse fell till it became slower than normal.

In a month he was almost well, but it was not until he had been taking parathyroid for six months that his sexual functions returned. By January 11th, 1912, he was weighing 161 lbs., and at Christmas, 1912, he weighed 175 lbs. He then discontinued taking the parathyroid preparation. Though he has now (August, 1913) gained another four pounds, he is more restless and his pulse is faster than it was in 1912; he has therefore been advised to continue to take 1/10 gr. of dried parathyroid gland daily.

The remarkable effect of treatment with dried parathyroid gland after everything else had failed makes it very probable that the group of symptoms from which the patient suffered was the result of parathyroid insufficiency.

SUMMARY OF WEIGHT AND TREATMENT.

Date.	Weight.	Remarks.
August, 1910 ...	191 lbs.	Illness began.
December 2 ...	144 „	Rest, over-feeding.
„ 13 ...	130½ „	Opium and bromide.
January 20, 1911 ...	144 „	
February 6 ...	159 „	Returned to work, but continued opium and bromide.
June 18	127½ „	Parathyroid began.
„ 21	134 „	Discontinued bromide and opium.
„ 26	146 „	
„ 30	151 „	
July 5	156 „	
„ 12	158 „	
January 11, 1912 ...	161 „	
December 25 ...	175 „	Discontinued parathyroid.
August 8, 1913 ...	179 „	

CASE OF RECOVERY FROM A DUODENAL ULCER PRODUCED BY A BURN.

By

ARTHUR F. HERTZ, M.D.,

AND

W. E. DIGBY, M.R.C.S., L.R.C.P.

THE occasional association of a duodenal ulcer with a burn or scald was first recognised by James Long[1] in 1840. The most important contributions to the subject since that date were made by Curling[2] in 1841 and by Perry and Shaw[3] in 1893. The latter authors found that between 1826 and 1892 a duodenal ulcer was found in 0·4 per cent. of all cases, upon which autopsies were made at Guy's Hospital, but in 3·3 per cent. of patients dying from the effect of burns. It seems that in recent years the frequency of the association has diminished, possibly owing to the improved treatment of burns resulting in the less frequent occurrence of septic infection, although the present case shows that the latter is not an essential factor. The ulcer is almost invariably found in the usual situation in the first part of the duodenum; it was single in about half the recorded cases, two or more ulcers being present in the others. It occurs at every age, but twice as frequently in women as men. In seven out of Perry and Shaw's twenty-nine cases death resulted from perforation, and in thirteen from hæmorrhage; in the remainder death was due to the burn, no symptom of ulcer having been noticed during life. Death followed perforation between the third and eleventh day and hæmorrhage between the fourth and thirty-seventh day.

So far as we are aware nothing has ever been written on the subject of duodenal ulcers resulting from burns from the clinical point of view, no case having been recognised during life. The following case, which appears to have been of this nature, is therefore of great interest, but as the patient recovered it is impossible to say with complete certainty that a duodenal ulcer was present.

Elizabeth J., aged 35, a cook, was admitted into Mary Ward on 23rd February, 1913, for weakness and increasing pallor, with a provisional diagnosis of infective endocarditis. She had always enjoyed good health and had never suffered from indigestion. She remained very well until four weeks before admission, when she scalded the dorsum of her left foot whilst at work in the kitchen. The next day she felt weak and had no appetite, and seven days later she noticed that she was getting paler. The scald took three weeks to heal, but it caused her very little inconvenience, and did not become septic. From the date of the scald she became progressively paler and weaker; she could not do her work properly and had no appetite. Four days before admission she consulted a doctor, who advised her to come into the hospital. She had become so weak that she could not stand up without fainting. The doctor found that her temperature was 99·8° F., and her pulse 120; her heart was slightly dilated, and there was a soft systolic murmur at the base. The day before admission she vomited gastric contents of a darkish colour.

On admission she appeared to be very anæmic; her face was waxy in appearance, and there was no trace of colour in her cheeks. She complained of no pain. Her temperature was 97·6°; her pulse was 114 and her blood pressure 108 mm. of mercury. Her teeth were good, and her tongue was moist and clean. Her appetite was very poor, and she had become extremely constipated. She complained of no flatulence or distension, and the abdomen moved normally on respiration. Nothing abnormal could be felt on palpation; the spleen was not enlarged. Deep pressure showed the presence of very slight tenderness on the right side

at the level of the umbilicus. The cardiac impulse was felt in the mid-axillary line; on auscultation a faint systolic murmur was audible at the apex, and a louder systolic murmur at the base. Her respiratory and nervous systems were healthy, and her urine was normal. A blood count showed that she had 2,200,000 red corpuscles and 6,875 leucocytes to the cubic millemetre, and the hæmoglobin was 25 per cent. of the normal.

She was put on a light diet, and as her bowels had not been open for several days, she was given in turn cascara sagrada, castor oil and white mixture, but without effect. On February 25th she was ordered an enema. This acted well, and on examination the fæces were found to contain blood in large quantities. She vomited during the night, the vomit containing distinct traces of blood.

Acute duodenal ulcer resulting from the burn on her foot was diagnosed. She was ordered citrated milk and subsequently horse serum; no purgatives were given, her bowels being kept open by enemata. She was restless, but became more quiet with morphia. The pulse rate varied between 104 and 116 on February 24th, 25th, and 26th; between 94 and 112 on February 28th and 29th; between 84 and 112 on March 1st and 2nd; between 84 and 104 on March 3rd; and between 84 and 92 on March 4th. The temperature was 98° on admission; it rose to a maximum of 100·5° on February 24th, 101·4° on February 26th, and 101·8° on February 27th. The temperature was irregular on February 28th and March 1st, dropping to normal on March 2nd and rising slowly to 101° on March 6th; it rose still further to 103° on March 9th and 10th, when she developed a thrombosed vein in the left leg, which no doubt accounted for the return of the pyrexia.

As blood was still present in the stools on February 26th, she was starved completely; fresh sheep's thymus, calcium lactate (x gr. three times a day), and horse serum were tried. As the latter made her vomit fluid containing a trace of blood, and the thymus and calcium lactate seemed to have no effect in controlling the hæmorrhage, they were omited. From the beginning

she was given subcutaneous injections of ½ gr. iron and ammonium citrate twice a day.

On March 10th the blood disappeared from the fæces and the patient was given fluids. Her appetite returned and she felt better. Her blood count was now 2,200,000 and hæmoglobin 28 per cent. On March 12th she was given farinaceous diet, and on the next day eggs. She had now a slight colour in her cheeks, and she was given a mixture containing iron and arsenic instead of iron injections. On March 26th she was put on to a light full diet. Her colour was much better and she ceased to be depressed. The blood count was now 2,800,000; on March 23rd it was 3,100,000. On March 30th she was given full diet, and she got up on April 4th. On April 9th the blood count was 3,625,000 and hæmoglobin 65 per cent. She was discharged on April 15th feeling quite well, her blood count being then 3,900,000. During the next four months she had some slight indigestion, but on October 27th she reported herself as well and strong.

REFERENCES.

1. J. Long, London Medical Gazette, New Series, I., 793, 1840.
2. T. B. Curling, Medico-Chirurgical Transactions, XXV., 260, 1841.
3. E. C. Perry and L. E. Shaw, Guy's Hospital Reports, L., 171, 1894.

SOME NOTES ON THE EXCRETION OF ACETONE BODIES.

By

E. L. KENNAWAY, M.A., M.D.

Contents:

I.—The Chemical Relationships of the Acetone Bodies.

The term "acetone bodies" is a convenient collective title for a group of three compounds, namely, acetone, aceto-acetic acid, and β-oxybutyric acid, which are closely allied to one another. Their chemical relationships may first be briefly considered.

The work of Knoop and of Dakin[1] has thrown much light upon the exact manner in which fatty acids are oxidised in the body. The chain of carbon atoms forming a fatty acid is not oxidised all at once, or at different points at random; it seems that for some reason the third carbon atom, counting from the acid group at the end of the chain, is most susceptible to oxidation, and the first oxygen atom is therefore attached at this point. If butyric acid, $CH_3CH_2CH_2COOH$, is oxidised in this manner, either β-oxybutyric acid, $CH_3CHOHCH_2COOH$, or aceto-acetic acid, CH_3COCH_2COOH, will be produced. Owing to the ease with

which these two latter acids are converted into one another in the body, it is not at present possible to say which of them is the primary oxidation product.

The conversion by the tissues of β-oxybutyric acid into aceto-acetic acid, and the reverse change, has been demonstrated by many different workers (Neubauer, Dakin and Wakeman, and others). The one reaction is an oxidation, the other a reduction.

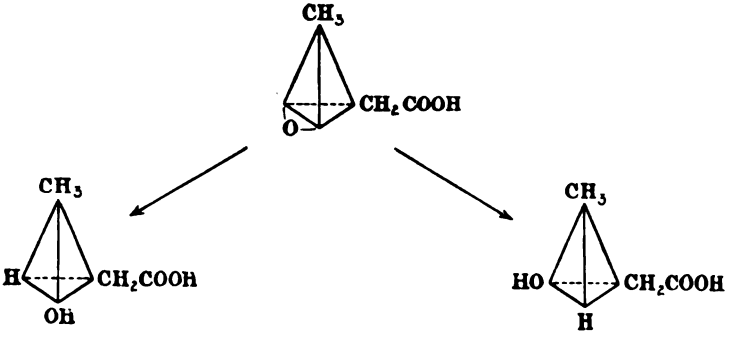
$$CH_3CHOHCH_2COOH \underset{-2H}{\overset{+2H}{\rightleftharpoons}} CH_3COCH_2COOH.$$

Neubauer[2] showed that when β-oxybutyric acid was administered to a diabetic, both this acid and aceto-acetic acid were excreted in increased amounts; and similarly, when aceto-acetic acid was given, the output of both the acids was again increased. One must conclude that in the body either of the two acids is convertible into the other.

This phenomenon has been further investigated by Dakin and Wakeman,[3] who found that ground-up liver tissue from a dog could effect the reduction of aceto-acetic to β-oxybutyric acid, while a cell-free watery extract of liver, if supplied with oxygen or oxyhæmoglobin, brought about the contrary oxidative change. The reaction is thus a reversible one, and Neubauer's observations suggest that in an intact organism this reaction has a certain equilibrium point, an increase in the amount of either member of the pair of compounds leading to the formation of some of the other one.

Of the three compounds under consideration, β-oxybutyric acid alone contains an asymmetric carbon atom, and can, therefore, exist in the form of two optical isomers. It is a rule with very few exceptions that a naturally occurring optically active compound exists in any one situation in the form of one only of its two isomers. So far as is known, the higher animals produce only the levo-rotatory β-oxybutyric acid, and the simplest method of estimating this substance in urine, namely, by the polarimeter, depends upon this fact. But it was shown by Neubauer that yeast when supplied with aceto-acetic acid inserted

the hydrogen atoms in a different way, producing the dextro-rotatory compound. These two methods of reducing aceto-acetic are represented below :—



Acetone is produced by the splitting off of CO_2 from aceto-acetic acid.

$$CH_3COCH_2COOH \longrightarrow CH_3COCH_3 + CO_2.$$

This change is accelerated by an acid reaction, and is irreversible. It occurs spontaneously in an acid urine containing aceto-acetic acid.

II.—The Acetone Bodies Occurring in Urine.

a. *Under normal conditions.*—Very small amounts of acetone can be obtained from normal urine by methods which would cause the conversion of any aceto-acetic acid present into acetone. It is, therefore, at present undecided whether one only or both of these compounds is present. Scott-Wilson,[4] using a new method of estimation, found the 24-hourly yield of acetone in man to be from 2 to 4 milligrams; the older and less reliable analyses gave considerably higher results.

One cannot at present say definitely whether β-oxybutyric acid is present normally. Magnus-Levy, who himself elaborated the method of isolation of this substance, was unable to detect its presence even when working with large amounts of urine, but he admits that it might nevertheless have been present, as the separation of small amounts from urine is a very difficult matter.[5]

b. *Under abnormal conditions.*—Some of the conditions under which abnormal amounts of acetone bodies appear in the urine will be dealt with in a subsequent section. Here one may consider merely the acetone bodies which are present in the urine in any case which would be said clinically to show acetonuria. It is commonly stated in the books that in the mildest forms of acetonuria acetone alone is excreted; if the condition becomes more marked, aceto-acetic acid appears also; while in still more severe cases, β-oxybutyric acid is added to the other two. It appears now that there is no basis of fact for this statement, which is due to the misinterpretation of qualitative tests (see Section III.) and to defects in quantitative methods of estimation.

Arnold[6] showed as long ago as 1900 that amounts of acetone detectable by the nitroprusside test were to be found only in the most marked forms of acetonuria, such as severe cases of diabetes, although aceto-acetic acid was present in abundance. In all milder cases of acetonuria, aceto-acetic acid was again present, but no acetone could be detected. His method was to extract two portions of the urine with ether, one portion being made alkaline and the other acid; the extract from the former would contain any acetone that is present, but no aceto-acetic acid, since the salts of this acid are insoluble in ether; the extract from the acid urine will contain aceto-acetic acid as well. The extracts were tested for aceto-acetic acid by Arnold's own method (see Table, page 168), and for acetone by the nitroprusside test, and it was only in the cases yielding large amounts of aceto-acetic acid that traces of acetone could be found.

Hurtley[7] has recently drawn attention to these experiments, which have been generally overlooked, at any rate in this country. The statements generally made as to the more frequent and abundant presence of acetone in urine would not have become current if Arnold's work had been recognised, and they are based upon a misapprehension of the meaning of the nitroprusside test (see Section III.). It seems probable that such acetone as is present in urine is formed chiefly in the urine after secretion by the decomposition of aceto-acetic acid.

The term "acetonuria" is, therefore, an unsuitable one. The condition is sometimes spoken of as "diaceturia," and in the light of Arnold's work this is certainly preferable; but chemists object to the name "diacetic acid" for aceto-acetic acid, so that it seems better to retain the term "acetonuria," using it to denote an excretion of acetone bodies, and not of acetone especially.

It is well known that in cases of marked acetonuria a considerable amount of acetone is excreted in the breath; in severe diabetes this may amount to several grams daily. At first sight this does not accord well with Arnold's results. If there is so much acetone in the breath there must be acetone in the blood, and if there is acetone in the blood one might expect it to occur in larger amounts in the urine. The reason for this difference between the expired air and the urine may be that the lung and the kidney are not supplied with the same blood. One may suppose that any acetone formed in the blood can escape during the passage of the blood through the lung, since acetone is readily diffusible. This blood is then conveyed in a few seconds down the aorta and through the kidney, and this may be done before any appreciable fresh formation of acetone has taken place. During the comparatively slow passage of the blood through some of the systemic capillaries, and along the venous system, some formation of acetone may occur; or possibly acetone is produced in some of the viscera, such as the liver; in either case this blood must pass through the lung before reaching the kidney. Further, the complex chemical conditions in the blood are, of course, different in the arterial and venous vessels, and it may be that in the latter, where the reaction is tending toward the acid side, the conditions for the formation of acetone from aceto-acetic acid are more favourable.

The question of whether aceto-acetic acid is invariably accompanied in the urine by β-oxybutyric acid is a difficult one. All the more recent observers agree that β-oxybutyric acid is always to be found when considerable amounts of aceto-acetic acid are present,, but the lesser degrees of acetonuria require

further investigation. β-oxybutyric acid gives no colour reactions, and the methods for its chemical isolation or quantitative estimation become unsatisfactory when very small amounts are dealt with. The old statement quoted at the beginning of this section was that β-oxybutyric acid was present only in the more severe cases of acetonuria, but this probably means that small amounts were not detected by the older methods. A case of diabetes was recently in this hospital in which the urine gave only a faint nitroprusside reaction, and the alveolar CO_2 was found by Dr. Poulton to be within normal limits. The aceto-acetic acid output per day was only 20 mg. An estimation of β-oxybutyric acid gave as a result a daily output of 14 mg., and this relation between the amounts of the two acids is just what one would expect from a comparison with a series of more marked cases of acetonuria in which the ratio of β-oxybutyric to aceto-acetic acid rises progressively.[21] One may note that on the old system this urine would have been stated definitely to contain acetone, but no aceto-acetic or β-oxybutyric acid, whereas it probably contained in reality both these acids, but no acetone.

One frequently sees the statement that the absence of a simple test for β-oxybutyric acid is no disadvantage clinically, since it is never present in urines which do not give the nitroprusside test. This statement is pure assumption, and it is important that it should be recognised as such. Probably it is true that every urine which gives a distinct nitroprusside reaction would have sufficient β-oxybutyric acid for estimation by a good method. But obviously this has nothing to do with the question whether β-oxybutyric acid ever occurs in urine unaccompanied by aceto-acetic acid, and we have direct evidence that it does so occur. Stadelmann and others have observed the excretion of considerable amounts of β-oxybutyric acid in the absence of aceto-acetic acid and acetone.* It is quite possible that in some pathological conditions the body has difficulty in oxidising

*These results are quoted as credible by Magnus-Levy (5), who is a very sceptical critic. I have not seen the original papers.

β-oxybutyric acid either to aceto-acetic acid or to carbon dioxide and water by some other route. It would, of course, be a laborious investigation to examine for the presence of β-oxybutyric acid a large number of urines which did not give the nitroprusside reaction. The cases, however, need not be taken at random. If a patient had defect in the power of decomposing β-oxybutyric acid the concentration of this substance in the blood would rise, and like any other acid compound it would produce increased respiration with resultant diminution of the alveolar carbon dioxide[8] and a certain type of alteration in the dissociation curve of the hæmoglobin.[9] If the acid accumulated to a sufficient extent the increased respiration would become obvious as "air-hunger." It would be well if the urine of any patient who showed these changes for no assignable reason were examined for the presence of β-oxybutyric acid.

III.—Qualitative Tests for the Acetone Bodies.

Hurtley[7] has recently given an elaborate account of the tests for aceto-acetic acid and acetone, and has introduced a new test for the former substance. Full details of the methods of performing the various tests will be found in his paper, so that it is unnecessary to describe them all in detail here. The subjoined table is compiled from his paper, and serves merely to indicate the uses of the different tests.

The tests for acetonuria most commonly employed are the ferric chloride test, and Rothera's modification of Legal's test. Legal's test consists in the addition of a few drops of fresh 5 per cent. sodium nitroprusside solution to urine made alkaline with caustic soda; if aceto-acetic acid is present, a red colour appears. Creatinin, a normal constituent of urine, gives a similar colour which changes in a few minutes to yellow. Rothera[10] showed that if the urine were made alkaline with ammonia instead of with soda, and were saturated with ammonium sulphate, creatinin gave no colour on the addition of nitroprusside, while the delicacy of the test as a means of detecting acetonuria was increased.

Test.	Author.	Colour.	GIVEN BY		
			Acetone.	Aceto-acetic Acid.	Other Substances occurring in Urine.
Ferric chloride	Gerhardt	Red-brown to purple	0	+	Substances excreted after taking various aromatic compounds (salicylates, salol, aspirin, etc.). Colour more violet than that due to aceto-acetic acid.
Nitro-prusside	Rothera	Pink	+ 1 in 20,000	+ 1 in 400,000	None known that give similar colour.
Iodine	Riegler	Iodine decolourised	0	+	None known.
Para-amino-aceto-phenone	Arnold	Purple*	0	+ 1 in 40,000	None known.
Isonitroso-acetone	Hurtley	Violet or purple	0	+ 1 in 50,000	None known.

*The urine should be shaken with animal charcoal and filtered before applying Arnold's test. If this is not done, a urine, though containing a high percentage of aceto-acetic acid, gives a reddish-purple colour very similar to that given by the reagents alone; by artificial light the two colours can scarcely be distinguished. In any case the test is inferior to Rothera's, both in simplicity and delicacy.

Rothera described this as a test for acetone. Hurtley has pointed out that the test is given both by acetone and by aceto-acetic acid, and is about twenty times more delicate in regard to the latter (see Table). Legal stated originally that his reaction was given by both substances, but this has been overlooked, and the test is now firmly established in the books as one for acetone only.

In carrying out Rothera's reaction, it is important to note not only the maximum depth of colour attained, but also the rate of its development. In this way one gets a better idea of the quantity of aceto-acetic acid present. The greatest depth of colour is reached in from five to twenty minutes, a larger

amount of the acid causing a quicker development. In testing by this method one may adopt some such arbitrary scale as the following:—(1) quick-strong; (2) slow-strong; (3) quick-weak; (4) slow-weak. A "quick-strong" reaction shows a deep permanganate colour within a few seconds of the addition of nitroprusside; a "slow-weak" reaction may show no pink at all for some minutes, and it is then most marked in the lower part of the test tube where the ammonium sulphate solution is most concentrated.

In my experience the quantitative indications of the ferric chloride and Rothera tests are roughly as follows:—(1) a "quick-strong" Rothera shows the presence of more than 0·25 per cent. aceto-acetic acid; one five-hundredth of this amount, or 0·0005 per cent. (1 in 200,000) will give a "slow-weak" reaction; (2) the ferric chloride reaction is not obvious when less than 0·07 per cent. aceto-acetic acid (1 in 1,400) is present, though with care it can be observed with considerably less; four times the above amount is required to give a strong red-brown colour, looking black by reflected light. When this reaction is faint the precipitate of phosphates should be filtered off.

The relative delicacy of the Rothera and ferric chloride tests is illustrated by the following experiment:—A diabetic urine, giving a rather slow-strong Rothera test and fairly strong ferric chloride test, was found on quantitative estimation to contain 0·235 per cent. acetone + aceto-acetic acid reckoned as aceto-acetic acid.* It was diluted with a normal urine: at a dilution of 1 in 20 (1 part aceto-acetic acid in 8,500) the ferric chloride reaction was extremely faint, and would not have been detected without special attention. Rothera's test, on the other hand, gave a quite distinct "slow-weak" reaction at a dilution of 1 in 500 (1 part aceto-acetic acid in 212,000) and was just perceptible at double this dilution. When this thousand-fold dilution was carried out with water instead of with urine the Rothera colour was more distinct. In this experiment, then, the

* 1 part acetone is derived from 1·76 parts aceto-acetic acid.
1 part aceto-acetic acid yields 0·558 part acetone.

Rothera test was at least 25 times more delicate than the ferric chloride test. This difference in the sensitiveness of the two reactions is a fortunate circumstance, since it enables one to form a better idea of the amount of aceto-acetic acid present.

A simple dilution experiment, such as the one described above, illustrates the long over-looked fact that aceto-acetic acid is at any rate greatly predominant over acetone in urine, since acetone is not detectable by Rothera's test at anything like these dilutions (see Table and Rothera's paper[10]).

The examination of acetonuric urines by test-tube reactions could only lead to erroneous statements as long as Rothera's test was regarded as one for acetone only, and a urine was thought to contain no aceto-acetic acid unless it gave the ferric chloride reaction. A urine containing 0·01 per cent. aceto-acetic acid and no acetone at all, and giving a positive Rothera and negative ferric chloride test, would be said to contain acetone and no aceto-acetic acid, whereas it was urines of this very type, giving the one reaction and not the other, which Arnold[6] used in demonstrating the presence of aceto-acetic acid and the absence of acetone. This misapprehension of the meaning of Rothera's test is, of course, the source of the statement that acetone is present alone in slight forms of acetonuria, while the appearance of aceto-acetic acid as revealed by the ferric chloride test is a sign of a more severe disturbance.

The fact that the urine of persons taking salicylates gives a colour with ferric chloride similar to that produced by aceto-acetic acid is not of much importance so long as Rothera's test is performed as well. A urine which gave the ferric chloride reaction at all would give a quite distinct Rothera reaction if the colour with ferric chloride were due to aceto-acetic acid. If a person were taking salicylates and, as happens in salicylate poisoning, excreting aceto-acetic acid, the Rothera test would show the presence of the latter substance, and would provide a sufficient basis for judging of its amount. In any case of doubt the urine can be boiled in a flask for a few minutes: if not acid, it must be acidified with acetic acid. If the urine is then

cooled and tested with ferric chloride, the colour, if due to aceto-acetic acid, will be very much less than before, the greater part of the acid having been converted into acetone. The colour due to other substances is not affected by this preliminary boiling.

It is generally stated that the colour with ferric chloride, if due to aceto-acetic acid, disappears on warming, while that due to salicylates is unaffected or intensified. So far as my experience goes, the colour in either case becomes paler and redder on warming, and in the case of aceto-acetic acid a reddish flocculent precipitate appears on boiling.

IV.—Conditions under which Acetonuria occurs.

a. *Acetonuria in healthy persons.*—Acetonuria is readily produced in a healthy person by abstention from carbohydrate food; the subject of such an experiment may take either protein alone, or fat alone, or both protein and fat, or no food at all.* As an example one may quote an experiment† in which a diet of eggs and cream was taken; on the third day the urine gave slight ferric chloride and strong Rothera reactions, and 1·33 g. aceto-acetic acid and 2·74 g. β-oxybutyric acid were excreted.

A diet of fat alone produces a much more intense degree of acetonuria than a diet of protein alone; similarly, if the diet consists of protein and fat, the acetonuria is diminished by increasing the protein and increased by increasing the fat. If a day of protein feeding be interposed in a period of starvation, the output of acetone bodies falls. The following three conditions will then produce acetonuria in ascending degree :— (1) a diet of protein; (2) starvation; (3) a diet of fat.

If carbohydrate be then added to any of these diets, or taken alone during a fast, the acetonuria is lessened or stopped according to the amount taken. This phenomenon, of course, finds clinical application when bread or dextrose is given to a diabetic

*For references on this subject, see Leathes, "Problems in Animal Metabolism," Lecture V.

†An experiment upon himself by Dr. Graham of St. Bartholomew's Hospital.

showing a dangerous degree of acidosis. The experiments of Satta[19] indicate that at least 80 to 100 grams of carbohydrate are required to prevent acetonuria in a healthy person, and in harmony with this he did not succeed in producing acetonuria by adding large amounts of fat to a mixed diet. It is obvious that the quantity of carbohydrate supplied is of importance in this connection, for a well-nourished man who abstains from carbohydrates for a few days and shows marked acetonuria will still have a considerable store of glycogen, and of carbohydrate combined with protein, while the body is capable of producing sugar from some of the amino-acids of protein, and probably from fat as well.

The experimental evidence obtained from normal persons shows, then, that acetonuria is produced by inadequacy in the supply of carbohydrate and is intensified by the metabolism of large quantities of fat. Starvation in itself increases the amount of fat oxidised, because the body has then to obtain the greater part of its energy from this source. The action of protein is, at first sight, less clear; when given as the sole food, acetonuria appears; when added to a diet of fat, or given in starvation, the acetonuria is diminished. Now most proteins contain some combined carbohydrate, and can yield a further quantity in the body from their amino-acids. Evidently, then, protein acts as a food poor in carbohydrate; enough of it cannot be taken to meet the whole needs of the body in this respect.*

The production of acetonuria in a normal person illustrates very well the complexity of the oxidative processes in the body. For oxidation in the tissues three things are required :—(1) oxygen; (2) a substance which the body can oxidise; (3) some means of making the oxygen combine with the substance. A healthy man living on protein and fat is well supplied with the first two of these requisites, yet he is deficient in the capacity to utilise β-oxybutyric and aceto-acetic acids, which are readily

* The question of the production of acetone bodies from protein will be discussed later.

oxidised under normal conditions. Evidently it is the third factor that has failed, and this failure seems to be due to lack of carbohydrate.

One has now to consider whether the acetonuria seen in pathological conditions is of the same nature.

b. *Acetonuria in pathological conditions.*—Magnus-Levy states that he can detect the smell of acetone in 10 per cent. of the patients in a general hospital. There would seem to be here a wide field for clinical research.

The mere fact that a person is in a hospital does not, of course, prove that any acetonuria which he may show is associated with his disease. Frew[11] examined by Rothera's test the urines of 662 children in the wards at Great Ormond Street. In 62 per cent. of the cases a positive result was obtained, and in the great majority of these the acetonuria was first detectable about twelve hours after admission, was most marked twenty-four hours later, and had disappeared by the fourth day. When the cases were grouped according to the type of disease (affections of the digestive system, of the nervous system, etc.), the percentage incidence of acetonuria was found to be approximately the same in each class, and the same figure was obtained in a series of simple surgical cases. Among the out-patients acetonuria was extremely rare, and could, as a rule, be assigned to some obvious cause.

The acetonuria seemed, then, to be brought on by the new conditions of hospital life. The diet given was not deficient in carbohydrate; however, it was found that the administration of dextrose stopped the excretion of acetone bodies within twelve hours. Since, then, the deficiency lay neither in the supply of the ordinary forms of carbohydrate, nor in the power to absorb and assimilate dextrose, the end-product of carbohydrate digestion, one must conclude that the capacity to digest carbohydrate food had undergone a transitory impairment resulting from the change in diet or in mode of life. One would expect that the younger children would be more liable to this disturbance, and the incidence of acetonuria was found to fall fairly regularly

from 84 per cent. at 3 to 4 years of age, to 46 per cent. at 11 to 12 years. Again, every one of 11 breast-fed infants was found to show acetonuria after the change to artificial food on admission.

These results show that if it be required to ascertain the significance of acetonuria in any clinical case, attention must be directed in the first place to the efficiency of the carbohydrate supply; there may be defect in the amount given, or in the processes of digestion, absorption, or assimilation and oxidation.

In diabetes, when the body is to a greater or less extent unable to utilise dextrose, the tissues are in a state of virtual carbohydrate starvation, which is no doubt the cause of the acetonuria. The amount of acetone bodies produced by a patient on a strict diet may be lessened by giving carbohydrate; apparently an increase in the concentration of dextrose in the body leads to the utilisation of a larger amount, perhaps by simple chemical mass-action. However, the condition in some cases may be more complex, for a diabetic may still show acetonuria when retaining more carbohydrate than would suffice to prevent any production of acetone bodies in a healthy person (Mohr[12]).

Acetonuria has been observed in a great variety of pathological conditions. Mohr mentions in this connection many acute fevers, advanced malignant disease, gastro-intestinal affections, scurvy, eclampsia, death of the fœtus in utero, etc.; also the action of anæsthetics, of many poisons, and of thyroid extract. Other clinical conditions which might be added to this list are cyclical vomiting, pernicious vomiting in pregnancy, and the rare sequel of anæsthesia known as delayed chloroform poisoning.

Obviously the intake of carbohydrate may be inadequate because the patient lacks appetite, or is under dietary restrictions, or vomits, though these simple factors seem to have been sometimes overlooked. In other cases some defect in the processes of digestion, absorption, or assimilation might well be present: the question could readily be investigated by the administration of dextrose. Mohr applied this test in a number of examples of some of the conditions mentioned above, and concluded that acetonuria is in all cases due to lack of carbo-

hydrate. This generalised statement is certainly somewhat in advance of the facts, but at any rate it appears that no one has demonstrated a form of acetonuria which persists when any considerable amount of carbohydrate is being assimilated.

The appearance of acetone bodies in the urine after anæsthesia is no doubt in many cases due partly to restriction of diet before and after the operation, but it is also an effect of the anæsthetic. Telford and Falconer[13] found that acetonuria, as demonstrated by the ordinary qualitative tests, occurred in about the same percentage of cases (89 per cent.) whether the anæsthetic were chloroform, ether, ethyl chloride, or chloroform and ethyl chloride. In twenty-five control cases which were prepared as for operation in the same way, but were not anæsthetised, no acetonuria resulted. No particulars are given of the mode of preparation.

The condition known as delayed chloroform poisoning, in which marked acetonuria, hyperpnœa, drowsiness, and coma occur, is in these respects similar to diabetic coma. It may follow anæsthesia by ether, ethyl chloride, or nitrous oxide, but is much more liable to appear after chloroform (Guthrie[14]). This sequel of anæsthesia is said to be at any rate very much less frequent if the patient be not subjected to any rigorous preparation, but be well supplied with carbohydrate before the operation, which is "substituted for the last meal" (Waugh[15]).

Spriggs[16] has discussed the nature of delayed chloroform poisoning from the physiological standpoint. He concludes that the metabolism of the patient is at the outset in an abnormal state owing to the inadequate supply of food; to this condition is superadded the effect of the anæsthetic, which in some persons produces persistent vomiting, and this in turn, of course, renders the condition already existing more serious. Guthrie[14] has pointed out the similarity between delayed chloroform poisoning and cyclical vomiting, and regards them as manifestations of the same disorder. He has observed that many children who are subject to cyclical vomiting have an extreme craving for carbohydrate food, which suggests an inability to utilise this

class of food-stuff to the normal extent. In both delayed chloroform poisoning and cyclical vomiting there is a great accumulation of fat in the cells of the liver and kidney, and chloroform has been shown experimentally to produce a peculiar type of fatty change in the liver.[17] For this reason some authorities recommend the avoidance of chloroform in cases where the functional activity of the liver is thought to be specially affected. At the Bristol Royal Infirmary "chloroform is no longer used as an anæsthetic in any case of eclampsia or in any cases of pregnancy where acidosis is present." and this change "has been followed by a marked improvement in the mortality rate of cases of eclampsia" (Swayne[18]).

V.—The Source of the Acetone Bodies.

The acetone compounds must be formed in the body either synthetically or by the break-down of protein, fat, or carbohydrate. We have no evidence in favour of a synthetic origin, and it is *a priori* unlikely. The action of carbohydrate food in arresting acetonuria seems to exclude an origin from this source. One must then consider whether proteins or fats or both of these are the mother-substance in question.

In cases where the amount of acetone bodies excreted is large, it seems that at any rate the greater part is not derived from protein, since (1) no parallelism is observed between the amounts of these compounds and of nitrogen and sulphate in the urine (Satta[19]), and (2) in severe cases of diabetes the quantity of protein katabolised, as indicated by the output of nitrogen, could not yield at the most more than perhaps one quarter of the acetone bodies excreted (Magnus-Levy[5]). There is, of course, nothing in these results to exclude a partial origin from protein.

By a process of exclusion, then, one must regard the fats as the chief source, and there is also much direct evidence for this. Firstly, a diet of fat alone is the most potent means of producing acetonuria in a healthy person. Secondly, acetonuria arising from whatever cause is in almost all cases intensified by an increase in the amount of fat in the food. This is true not

only of clinical cases, but also of the experimental pancreatic and phlorrhizin glycosurias in animals. Thirdly, many fatty acids which occur naturally yield acetone when perfused through the liver.[1]

One must conclude that at any rate the greater part of the acetone bodies excreted is derived from fat, but one cannot as yet explain why lack of carbohydrate causes these substances to accumulate in the body. When the supply of carbohydrate food is diminished or withheld, more fat must be oxidised in order to supply energy, and it seems that under these circumstances the katabolism of fat is unable to proceed in the normal manner; the β-oxybutyric and aceto-acetic acids produced are not oxidised as they are when administered under normal conditions.* The proper course of fat katabolism is then dependent upon a free supply of carbohydrate, and upon the ability of the tissues to utilise this.

One must not overlook the possibility of the origin of acetone bodies from protein also. Various amino-acids which occur in proteins yield aceto-acetic acid when perfused through the liver, and are stated to increase the output of acetone bodies when administered to diabetics.† Further, Scott-Wilson[4] has shown that in normal human urine there is a distinct parallelism between the amounts of acetone and of nitrogen; in the dog this parallelism persists even when large variations in the amount of nitrogen are induced by changes in the diet and by starvation (Fritz Voit[20]).

VI.—The Acetone Bodies as a Cause of Acidosis.

The acid, alkaline, or neutral reaction of a fluid depends upon the relative amounts of H and OH ions in it. The normal blood, when tested by electrical methods and not by indicators, is found

*The reversible reaction between these two acids makes it difficult to decide which of them fails to be oxidised in acetonuria. An interesting discussion of this point is given by Neubauer (2).

†For references, see Neubauer in Abderhalden's Biochemisches Handlexicon, page 371.

to be practically neutral, that is, it contains approximately equal numbers of H and OH ions. If the uniform distribution of these two ions throughout a fluid be disturbed, the reaction of the fluid will not be the same in its different parts. Now various cells possess the power of altering the distribution of ions; a segregation of ions may occur within the limits of a unicellular organism, as is seen in the acid-containing digestive vacuole of amœba. In the course of development in the higher animals different capacities for dealing with and reacting towards different ions are sorted out into the various descendants of the fertilised ovum. Thus, the nerve cells in the respiratory centre become extremely sensitive towards acids. The cells of the digestive glands acquire the power of secreting acid or alkaline solutions from the neutral blood. In a carnivorous animal like the dog the acid gastric and alkaline pancreatic juices may be approximately decinormal, and it is one of the most remarkable things in physiology that two gland cells supplied from the aorta with the same blood should be capable of preparing such acid and alkaline solutions from it.

Some cells of the kidney transfer any excess of H or OH ions from one side of them to the other, *i.e.*, into the tubule. This is one of the functions of the kidney ; it serves not only to remove particular chemical compounds, but to regulate the reaction of the blood. Hasselbalch found variations in the acidity of the urine 25,000 times as great as those which occurred in the blood; the constancy in the one fluid is dependent upon the variations in the other. It is obvious that some such regulating organ is required if acids and alkalies are to be introduced with the food, produced in the course of metabolism, and removed from the blood at different times during digestion by the glands.

There must, however, be limits to the efficiency of the kidney in this respect. In the first place, contact with excessive amounts of acid or alkaline ions may damage the kidney cell, and a vicious circle be thus set up. In the second place, the kidney would be more favourably situated for regulating the reaction of the blood if it were interposed upon the course of the whole

blood stream, as the lung is. Acid substances might be produced in the body at such a rate that they could do damage, however efficient the kidney were; it is a question of the ratio between the rate of production of acid and the fraction of the whole blood passing through the kidney.

The maintenance of the reaction of the blood within normal limits does not depend wholly upon the kidney; it is assisted by the presence of proteins in the blood. If, for example, hydrochloric or sulphuric acid be added to a solution containing protein, the reaction of the solution is not altered as much as it would be if the proteins were absent;* the protein, though itself neutral, can in the presence of acid act like an alkali. If the blood were a protein-free solution, the variations in its reaction would be much greater, however efficient the kidney were.

When the body produces acids like β-oxybutyric or acetoacetic acid, these substances can be dealt with in various ways.

1. Normally they are oxidised to water and carbon dioxide, that is, the strong acid is converted into a weak one which decomposes, the CO_2 escaping through the lungs. In acetonuria the power to affect this oxidation has failed to a greater or less extent, and the acids must be dealt with in other ways.

2. The acids will combine with inorganic basic radicals present in the tissues and tissue fluids, and be excreted with them. This is a harmful process because it disturbs the concentration and distribution of inorganic radicals upon which the proper working of various organs, the heart, for instance, depends.

3. The presence of protein in the tissue fluids will lessen the alteration of reaction which would otherwise result from the appearance of these acids. However, the capacity of the proteins to do this is, of course, limited, and abnormal quantities of any acid will lessen the power of the proteins to deal similarly with the other acids still being produced by normal metabolic processes.

*This is well shown in some experiments by Mathison,, Journal of Physiology, xliii., page 360, 1912.

4. A certain amount of base is taken in with the food. A vegetable diet is richest in inorganic bases, but is, of course, unsuitable in the case of a diabetic. In practice, sodium bicarbonate is added to the diet, the result of this being to substitute CO_2 for the stronger acid.* One might expect this to be a very efficient means of treatment, but the ultimate fate of the majority of severe diabetics shows that its power is distinctly limited. The alkaline salt can only be introduced into the blood-stream; the extent to which it reaches the cells which are being damaged by acid is not under control from outside. Further, one might perhaps question whether the administration of large quantities of sodium salts over long periods is altogether innocuous; various familiar physiological experiments upon cardiac and striped muscle show that the Na ion has very powerful effects.

Doses of sodium bicarbonate certainly increase the amounts of β-oxybutyric and aceto-acetic acid excreted. Dr. Ryffel has suggested that when this salt is introduced into the blood-stream the organic acids must distribute themselves by mass action between the proteins and the sodium; of the two compounds thus produced one can pass out through the kidney and the other cannot.

5. The body can also neutralise acids by means of ammonia which would otherwise have been converted into urea. A healthy person on an ordinary diet excretes about 87 per cent. of the total nitrogen of the urine in the form of urea, and 3 to 5 per

* (1) 1 part $NaHCO_3$ neutralises 1·24 parts of β-oxybutyric or aceto-acetic acid. Since the two acids differ in molecular weight by less than 2 per cent. they may be regarded for the present purpose as one substance.

(2) 1 drachm (=3·88 grams) $NaHCO_3$ neutralises 4·8 grams β-oxybutyric acid; 2 drachms four-hourly would neutralise 58 grams of the acids in a day; such amounts are excreted in many cases of severe diabetes. The largest day's output of the two acids on record appears to be 153 grams (5).

(3) 1 drachm $NaHCO_3$ yields about 1 litre CO_2. 25 drachms would yield about 7 per cent. of the amount of CO_2 produced by a man at rest in twenty-four hours.

cent. in the form of ammonia. In severe cases of diabetic acidosis as much as 40 per cent. of the nitrogen may be excreted as ammonia, though such a proportion is quite exceptional. In such a case the amount of ammonia would be about 10 grams, which would neutralise 60 grams of β-oxybutyric acid. It would be well, then, if a person who was producing 100 grams of the acid daily could use more ammonia for this purpose, and surprise is sometimes expressed because this does not occur. Some writers seem to expect all the ammonia produced in the body to be played directly upon the source of β-oxybutyric acid, like water upon a burning house. The question of whether a molecule of ammonia is converted into urea or into an organic ammonium salt may depend simply upon whether it meets first the organic acid or the urea-forming mechanism. One must remember that the formation of urea is itself a protective mechanism which prevents the body from being poisoned by the highly toxic substance ammonia, and one would hardly expect the body to be capable of switching off this process to any required extent.

All the experimental evidence goes to show that β-oxybutyric acid and aceto-acetic acids have no toxic action apart from their acid character. From the clinical standpoint, then, the most important indications for treatment and prognosis in a case of marked acetonuria will be obtained by observing the degree of acid intoxication present; the acetonuria *per se* is of quite subordinate importance. One must inquire, then, in what way any quantitative estimate of acidosis can be obtained. Here the ordinary chemical methods fail. The most accurate estimations of the daily output of β-oxybutyric and aceto-acetic acid give no definite information as to the strain which these amounts of acid have imposed upon the neutralising powers of the body. One may learn whether the case is particularly severe in character, but one will not learn whether the patient is or is not in immediate danger. Estimations of urinary ammonia show the extent to which the organic acids have been neutralised by one of several possible methods. Estimations of

the acidity of the urine show merely the amount of acid which has been removed, whereas the life of the patient may be terminated by the further amount which ought to have been removed.

There is no doubt that at present the most valuable method of observing the degree of acidosis consists in estimations of the alveolar carbon dioxide. Measurements of the hydrogen ion concentration in the blood, and of the dissociation of hæmoglobin, may give similar and possibly more valuable information, but the methods required are more elaborate and the results obtained cannot as yet be interpreted in the light of abundant data. The pressure of carbon dioxide in the alveoli seems to be directly proportional to its pressure in the blood, which is in turn dependent upon the amount of stimulation by acid substances to which the respiratory centre is exposed. An estimation of alveolar carbon dioxide gives, then, at any rate some indication of the degree of acidosis prevailing within the body. It is the activity of the kidney cell which limits the value of all analyses of urine as a means of judging of the actual state of affairs within the tissues.

REFERENCES.

1. Dakin, Oxidations and Reductions in the Animal Body, London, 1912.
2. Neubauer, Verhandlungen des deutschen Kongresses für innere Medizin, xxvii., page 566, 1910.
3. Dakin and Wakeman, Journal of Biological Chemistry, viii., 97 and 105, 1910.
4. Scott-Wilson, Journal of Physiology, xlii., page 444, 1911.
5. Magnus-Levy, Ergebnisse der inneren Medizin und Kinderheilkunde, I., 352, 1908.
6. Arnold, Centralblatt für innere Medizin, xxi., 417, 1900.
7. Hurtley, Lancet, April 26th, 1913.
8. Beddard, Pembrey, and Spriggs, Journal of Physiology, xxxvii., page xxxix., 1908, and Lancet, June 19th, 1909.
9. Barcroft, Journal of Physiology, xlii., page 44, 1911.
10. Rothera, Journal of Physiology, xxxvii., page 491, 1908.

11. Frew, Proceedings of the Royal Society of Medicine (Anæsthetics), 1912, page 60.

12. Mohr, Ueber diabetische und nicht-diabetische Autointoxicationen mit Säuren (Acidosis), in von Noorden's Sammlung klinischer Abhandlungen, Heft 4, Berlin, 1904.

13. Telford and Falconer, Lancet, ii., page 1341, 1906.

14. Guthrie, British Medical Journal, page 1158, October 17th, 1908.

15. Waugh, Proceedings of the Royal Society of Medicine (Anæsthetics), 1912, page 73.

16. Spriggs, British Medical Journal, page 1162, October 17th, 1908.

17. Goodhart, Guy's Hospital Reports, lxiv., page 301, 1910.

18. Swayne, Proceedings of the Royal Society of Medicine (Obstetrics and Gynæcology), 1912, page 315.

19. Satta, Hofmeister's Beiträge, VI., page 22, 1904.

20. Voit, Deutsches Archiv für klinische Medizin, lxvi., page 564, 1899.

21. Kennaway, Pembrey, and Poulton, Journal of Physiology, xlvii., page x., 1913.

A STUDY OF FOUR HUNDRED AND FORTY CASES OF INGUINAL HERNIA.

BEING A PAPER READ BEFORE THE GUY'S HOSPITAL PUPILS' PHYSICAL SOCIETY ON MARCH 1st, 1913.

By

W. E. TANNER.

Mr. President and Gentlemen,—To-night I propose to take you away from the fields of philosophic medicine to consider a subject of great interest to us and of extreme importance to the community.

My reasons for occupying your time with a "Study of Four Hundred and Forty Cases of Inguinal Hernia" are numerous. In the first place, it is quite impossible for one whose clinical experience is very limited to be a reliable exponent of the more philosophic side of our work, for many years' experience are required before one can deal profitably with that mass of fact and fiction which constitutes the basis of accurate or inaccurate diagnosis and treatment; secondly, the majority of us know nothing concerning the after-history of our patients, and because one had seen more cases of inguinal hernia than any other surgical affection one felt that the after-results of operation on that condition would be more accurately judged than those of a more obscure surgical lesion; thirdly, although the views of experienced surgeons are more valuable than those obtained from mere statistical evidence, yet the latter, broadly interpreted, gives a firmer basis to our ideas.

The material upon which this paper is based was obtained by sending inquiries to four hundred and forty patients who were operated upon during the whole of the year 1908, and part of 1907 and 1909. Replies were received from two hundred and ten, and of these, one hundred (including all recurrent cases) were personally examined.

Inguinal hernia rarely occurs in mammals. It is met with, however, in the horse and in the female of the canine species. In the horse it is far more frequent in stallions than in geldings. In the latter the canal is usually reduced in size by castration. In many cases there is a hereditary predisposition, and in some it is undoubtedly congenital. In the pig, inguinal hernia is far from unusual, and is congenital in them if not hereditary.[1]

With these exceptions, however, it is so rare that the extreme frequency of the condition in man must, as has been suggested by Keith,[2, 3] be in some measure due to changes which have taken place in the pubo-femoral region as a result of the adoption of upright progression.

In man the external oblique muscle has become attached to the iliac crest to help in balancing the body, and Poupart's ligament has thus been formed by a separation of the lower tendinous fibres of the external oblique muscle from the muscular digitations arising from the lower ribs. In the orang, also an upright primate, the muscular digitations of the external oblique arising from the lower ribs have no attachment to the iliac crest, but terminate directly in the pillars of the external abdominal ring. In the gorilla the external oblique muscle is inserted into the iliac crest.

In all primates, except man, the internal oblique and transversalis muscles arise from the anterior border of the ilium, the sheath of the ilio-psoas muscle, and arching over the spermatic cord, end in a long insertion into the iliac pectineal line. By acting as a powerful sphincter of the inguinal canal they prevent hernia. This sphincteric action is also present in a lesser degree in man, and must be an important factor in the prevention of

hernia, because a large number of patent funicular processes have been demonstrated in normal adults. In the orang the abdominal wall becomes narrow inferiorily in the pubic region, and, therefore, the tendency to hernia is much less than in man, in whom the pubic symphysis has become raised in adaption to his method of walking.

The Causes of Hernia.

These factors may be divided into predisposing and exciting.

Predisposing Causes.

Sex.—Eighty-four per cent. of our cases were in males. This increased predisposition is due to several factors of which the most important is the descent of the testicle, for females do not show such a marked tendency to hernia at birth or during the early years of life as males. Further, in males, both during infancy and adult life, exciting causes are more often present.

Heredity.—There can be no doubt that inheritance also plays some part as a predisposing cause. A large number of patients gave a history of this condition in their parents. The father and five brothers of one female had been operated upon for hernia. It would be interesting to know whether the male parent transmits the tendency more to sons than daughters. In the case of the female it is stated that she predisposes to femoral hernia in males as well as females. In this hereditary transmission there are two factors, namely, the presence in the children of a congenital sac, and also the children of weak and unhealthy parents are more liable to suffer from pulmonary and intestinal disturbance which will act at a critical period when the abdominal wall is weak and underdeveloped.

Age.—It is generally stated that the occurrence of hernia is commonest during the most active period of life. This is not true of our cases, for although thirty-six per cent. occurred between the ages of ten and thirty years, thirty-one per cent. were noticed at birth and during the first year of life. In females the age frequency remains fairly constant during the

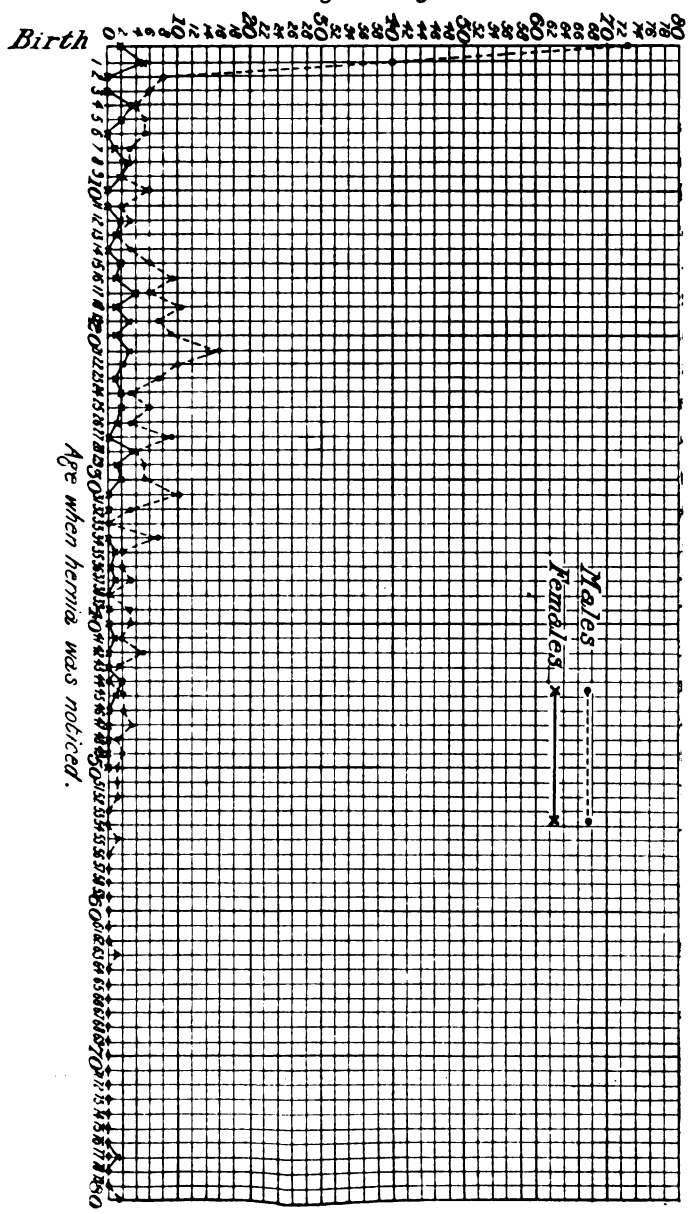
first thirty years of life and then declines. The accompanying curve illustrates these facts. The cases of direct hernia appeared between the ages of twenty-seven and forty-six years.

Age when "swelling" was first noticed.

Oblique inguinal hernia.

	Males.	Females.
At birth	73 cases	... 2 cases.
During the first year	40 „	... 5 „
Between ages of one and ten	58 „	... 21 „
„ „ ten and twenty ...	58 „	... 16 „
„ „ twenty and twenty-five	44 „	... 9 „
„ „ twenty-five and thirty	30 „	... 8 „
„ „ thirty and thirty-five...	22 „	2 „
„ „ thirty-five and forty ...	12 „	
„ „ forty and fifty... ...	20 „	3 „
„ „ fifty and sixty... ...	3 „	
„ „ sixty and seventy ...	1 case	... 1 case.
„ „ seventy and eighty ...	1 „	... —

Type of abdomen.—There are two types of abdomen which are associated with hernia and are seen either in children under seven years of age or in males over thirty. These types of abdomen do not result from the presence of a hernia, but are associated conditions which show that, apart from the presence of a congenital sac, a weakness of the abdominal muscles is an important predisposing cause. In the commoner type there is a median prominence corresponding to the two recti muscles, and two lateral ones corresponding to the oblique abdominal muscles. In the other type, which is sometimes associated with visceroptosis and the presence of an incomplete hernial sac, the abdomen is very protuberant below the umbilicus, and the upper parts of the transversales muscles are contracted. In pronograde animals the viscera are supported by the muscular walls of the abdomen, perivascular and peritoneal ligaments are unimportant. In them the peritoneal reflection from the posterior abdominal wall is a straight line from the stomach to the anus.


Number of cases during each year of life.
Birth
Males
Females
Age when hernia was noticed.

With the adoption of the erect attitude, the peritoneal reflection from the posterior abdominal wall becomes complex, as in man or the gorilla, the perivascular fibrous tissue becomes more developed, and is especially well marked in the region of the cœliac axis and superior mesenteric arteries. In orthograde primates the muscular mechanism for supporting the viscera is still present. The transversalis is the most important muscle of visceral support. The oblique abdominal muscles act as supports to the viscera and as balancing muscles, and are antagonistic to the action of the diaphragm. In a normal man the abdominal muscles are called reflexly into play when a change is made from the prone to the erect position. Visceroptosis may be due to a failure of this normal reflex from the intestine to the abdominal muscles with a change of position.

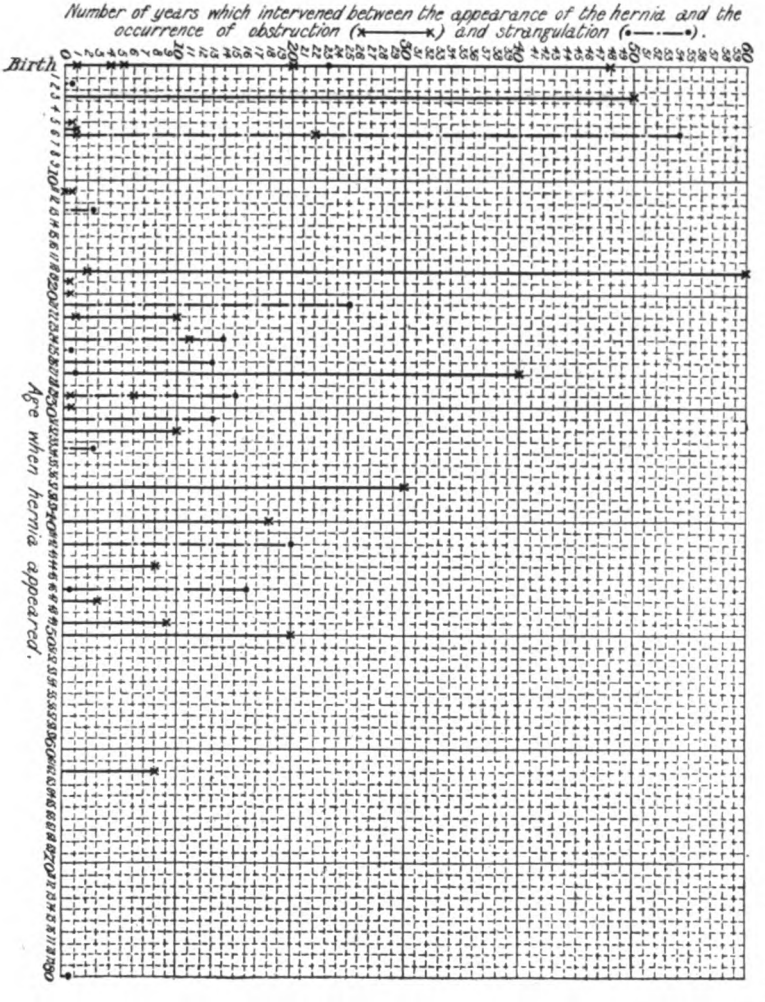
In our cases the hernia was commoner on the right side. This increased frequency on the right side is not more marked at birth than in later life, so that the later descent of the right testicle does not account for it. One believes that it is due to the fact that the iliac colon covers the left internal ring in a certain number of cases during strain. Also it is quite common to find a piece of small intestine or omentum lying in a well-marked fossa on the right side which is bounded externally by the cæcum (which may be dilated), and internally by the right lateral false ligament of the bladder which forms a hood over the internal ring.

Indirect Hernia.

	Males.		Females.
Right side	207 cases	...	35 cases.
Left „	131 „	...	28 „
Double „	26 „	...	5 „

Direct Hernia.

	Males.		Females.
Right side	4 cases	...	—
Left „	1 case	...	1 case.
Double „	4 cases	...	—


Number of years which intervened between the appearance of the hernia and the occurrence of obstruction (×———×) and strangulation (•—·—•).
Birth
Age when hernia appeared.

The Exciting Causes.

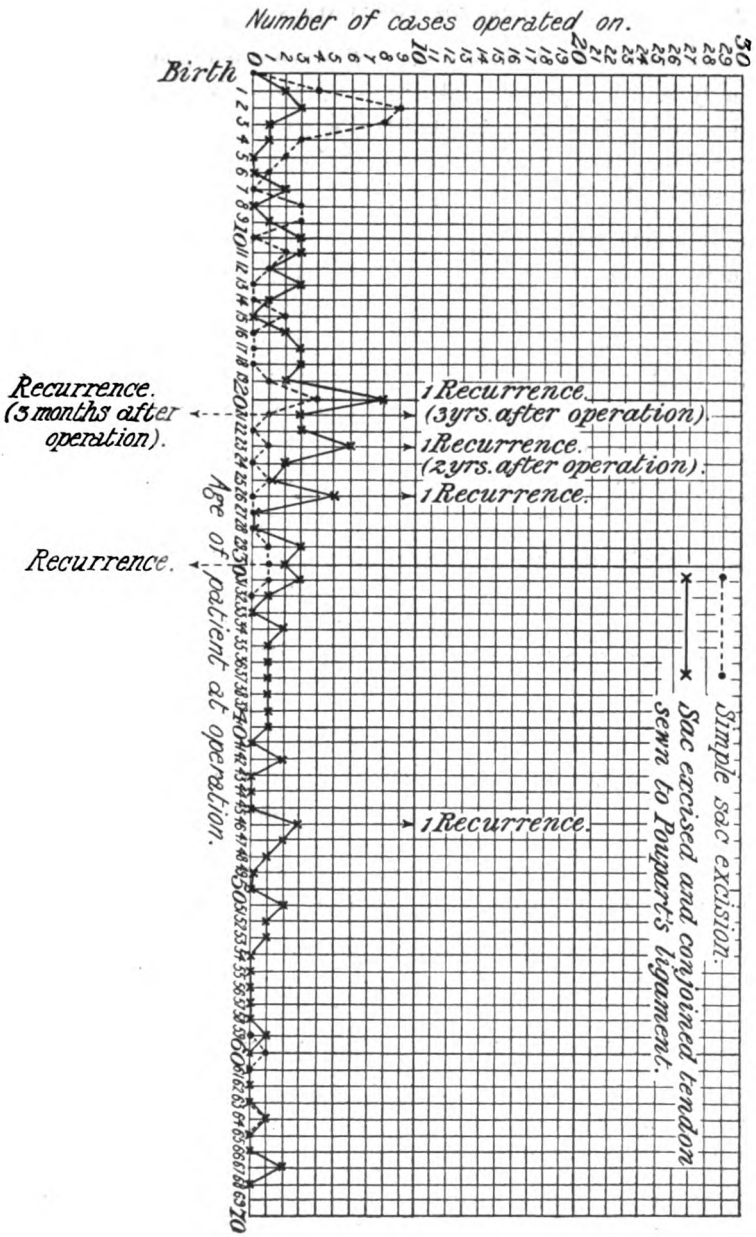
The important exciting cause of hernia is an increase of intra-abdominal pressure. If the inguinal canal were guarded by muscular tissue only, hernia would occur rarely. The increase in pressure may be gradual, but hernia rarely occurs from this cause. It is occasionally met with, however; Genge-Andrews has informed me of a case in which a hernia developed subsequent to the appearance of a large retroperitoneal sarcoma. Sudden and intermittent increase is probably the most common cause, and is of special importance in infancy and childhood. Most of our cases in adults were men engaged in heavy manual work; a large number gave a history of chronic bronchitis. In these cases one believes that the inefficiency of the inguinal canal resulted from factors acting in infancy and early adult life. A definite history of strain was given in thirty-seven cases, and in several, pain in the inguinal region occurred many months before the appearance of a swelling. In many, a sudden increase or decrease of obesity was considered the exciting factor. Large hydroceles and tumours of the cord and testicle may precede the appearance of a hernia.

The Nature of a Hernial Sac.

Hamilton Russell maintains that, with the possible exception of some cases of direct inguinal hernia, all herniæ descend into congenital sacs. In the majority of the cases this is doubtless true, and one may mention briefly the clinical and pathological facts which support this view :—

1. The large number of cases occurring at birth and during the first year; these formed thirty-one per cent. of our cases in males. In eighty-four per cent. the swelling was noticed before thirty years of age. In females ten per cent. appeared at birth or during the first year, and eighty-nine per cent. before the age of thirty.

2. The sudden descent of herniæ in adults into sacs having the same microscopical characters as congenital ones.


Number of cases operated on.
Birth
Age of patient at operation.
Recurrence. (3 months after operation).
Recurrence.
1 Recurrence. (3yrs. after operation).
1 Recurrence. (2yrs. after operation).
1 Recurrence.
1 Recurrence.
Simple sac excision.
Sac excised and conjoined tendon sewn to Poupart's ligament.

3. The success of operation by simple removal of the sac.

4. Patients who have had a radical cure on one side frequently return a year or more later to have an operation performed on a hernia which has appeared subsequently on the other side. In such cases there was a potential hernia on that side although the sac was empty.

5. Edmund Roughton[4] has looked for a sac on the other side in children and young adults. He found a sac in ten cases out of the eighteen in which he looked for an empty sac.

6. *The results of post-mortem examination.* a. Murray demonstrated fifty-two congenital diverticula in two hundred post-mortems on subjects who had never had a hernia during life.

b. Genge-Andrews and I have demonstrated eighteen sacs as a result of thirty-one post-mortems on males. The majority were of the "congenital" type. Two were of the "retrofunicular"[5] type, and in another a patent processus vaginalis with two lateral off-shoots was found. One diverticulum passed upwards between the external and internal oblique muscles, and the other was situated outside the external ring. In eight females who were examined, three sacs were found.

7. At the operation on our cases the majority of the sacs were of the funicular type, fifty-nine were congenital, thirty-seven were associated with imperfect descent of the testicle, and in four cases a retrofunicular sac was found. The latter variety is generally called the "infantile" type, but as it is commonly met with in adults, the terms "retrofunicular" or "intrafunicular" are preferable. A few of our cases were associated with tumours of the cord and testicle, large hydroceles, and tumours of the round ligament. In these cases traction on the peritoneum may have caused the formation of the "acquired" sac.

An interstitial sac was present in the case of a man aged 54, who was admitted with a strangulated oblique inguinal hernia. At the operation a dilated cæcum and ascending colon were found in the sac. These were reduced and the sac was excised, but as symptoms of strangulation persisted, a laparotomy was per-

formed. This revealed the fact that the gut was sloughing owing to strangulation as result of reduction from one sac into another at the first operation. The patient died, and post-mortem peritonitis due to sloughing gut was found.

In another case in a boy, aged 18, the mesosigmoid and bladder formed part of the posterior wall of an incomplete sac. This type of case is due to a gradual downward displacement of the iliac peritoneum, so that the original posterior wall of the sac becomes the fundus, and finally part of the anterior wall. The posterior wall thus becomes deficient and its place is taken by a descent of the cæcum of the right side and sigmoid on the left.

At the operation on a man, aged 70, who had a left irreducible hernia, a bilocular sac containing omentum, small intestine, large intestine, and bladder was found. The latter was inadvertently opened, but was sutured, and the patient recovered.

In the direct form the sac is usually acquired, but the presence of an oblique sac is a predisposing cause of direct hernia. At the operation on a man, aged 62, an oblique sac containing small intestine and a direct one containing omentum were found. The gut was returned, the sacs were excised and the conjoined tendon sewn to Poupart's ligament. When examined four years later, the conjoined tendon was thin and stretched, a bubonocele was present, and a truss was being worn.

Changes in the Sac and its Contents.

At the first the sac may be completely reducible ; very soon, however, it becomes adherent to surrounding structures, and new vessels are developed on its outer surface, especially if irreducible omentum be present. The neck may become thickened and sometimes obliterated. The pressure of a truss is especially liable to induce these changes. In thirty-four cases the contents of the sac were irreducible at the operation. The causes of irreducibility were (1) adhesions between the sac wall and its contents; (2) matting together of the contents; (3) the presence of bridles in the sac.

CONTENTS OF THE SAC AT OPERATION.

Oblique Inguinal Hernia.

	Right side.	Left side.
Males—		
Empty	128 cases	86 cases.
Omentum only	35 „	35 „
Small intestine	33 „	25 „
Cæcum and ascending colon	5 „	2 „
Transverse colon	—	1 case.
Descending colon	—	1 „
Pelvic colon	—	5 cases.
Appendix	9 „	1 case.
Meckel's diverticulum	1 case	—
Bladder	—	1 case.
Vas deferens and epididymis	1 „	—
Females—		
Empty	26 cases	18 cases.
Omentum	4 „	5 „
Small intestine	1 case	2 „
Fallopian tube	—	2 „
Ovary	1 „	2 „

Direct Hernia.

	Right side.	Left side.
Males—		
Empty	5 cases (4 double).	5 cases (4 double).
Omentum	2 cases.	—
Small intestine	1 case.	—
Females	One case on left side containing omentum.	

CONTENTS OF THE SAC OF IRREDUCIBLE OBLIQUE HERNIA.

Omentum	23 cases.
Small intestine	9 „
Small intestine and sigmoid	1 case.
Small intestine, cæcum, and appendix	1 „
Appendix	1 „
Cæcum and perforated appendix with abscess	1 „
Inflamed cæcum and appendix and twisted small intestine	1 „

In the majority of irreducible herniæ fluid was also present. The commonest cause of irreducibility was the presence of omentum adherent to the sac wall.

In two cases gut was adherent to the sac wall. In one case this was due to the presence of a perforated appendix with an appendicular abscess. In the other, an inflamed appendix, cæcum, and small intestine were present.

In five cases the intestine became incarcerated. In one of these the swelling was globular and of a doughy consistence, with entire absence of pain or tenderness.

Strangulation occurred in nineteen cases.

CONTENTS OF THE SAC OF STRANGULATED OBLIQUE HERNIA.

Omentum	9 cases.
Small intestine	9 „
Cæcum and ascending colon	2 „
Pelvic colon	1 case.
Meckel's diverticulum	1 „

In the five cases in which drainage or excision of gut was necessary, the patients died. In the case in which Meckel's diverticulum was strangulated, the cardinal signs of strangulation, namely, complete obstruction with fæcal vomiting, were absent.

THE TREATMENT OF HERNIA.

Herniæ in children may disappear permanently if they are controlled efficiently by a truss, but such a fortunate occurrence is extremely rare. In our cases only four disappeared in this way, the evidence of a former hernia being a thickened cord and a decided weakness in the inguinal region.

There are a large number of advertisements in the lay journals which state that rupture is curable without operation or the use of a truss. I have investigated this matter, and find that "Rupture Specialists" are unable to cause a permanent disappearance of the swelling, but merely control it by means of an "appliance," which is an instrument applying constant pressure over the inguinal canal.

One does not propose to deal with the advantages and disadvantages of trusses, but thinks it advisable to use a light

truss in children under one year old on account of the difficulties of post-operative nursing, which render sepsis a most likely complication, and the possibility of spontaneous cure. In any case an operation should be performed before the patient reaches sexual maturity in order to avoid the possible morbid attention to the sexual organs inducing a moral degeneracy; further on, reaching boyhood, being unconscious of the former physical defect, a boy would strengthen both mind and muscle by taking part in strenuous games. The use of a truss is also indicated in cases of direct hernia unless a really curative operation is devised.

The contra-indications to operative treatment are few. The physical condition of the patient must be the deciding factor: operation is definitely contra-indicated in severe general disease, in cases in which the reduction of the contents of large hernial sacs involves the risk of paralytic distension of the gut, in recurrent and direct hernia unless satisfactory results are obtained by filigree implantation or some modification of the plastic operations usually performed. There is no question that a man who has had a radical cure for simple hernia is a better life. In only two men amongst the present series is a truss now being worn in the "cured" cases. To ensure a "radical cure" the sac must be excised completely, tight suturing must be avoided, and convalescence must not be shortened unduly.

In the majority of our cases the external ring and external oblique aponeurosis were divided. This procedure did not weaken the canal, for, when examined three or four years later, the external ring was quite small in the majority of the cases. The external ring tends to become secondarily enlarged when, owing to a weakness in the posterior wall of the canal, abdominal contents are able to protrude through the external ring and induce an atrophy or splitting similar to that produced at the internal ring in long-standing cases.

The external ring should not be unnecessarily divided, but unfortunately this has to be done in the cases which are most liable to recur.

Treatment of Reducible Oblique Herniæ.

Simple sac excision.—The sac was excised in thirty-one males under the age of twenty without recurrence, and the after result was perfect. The canal was not weakened, and in many the side operated upon was stronger than the other, no doubt because a sac was also present on the side not operated upon. The operation was performed twelve times on males between the ages of twenty and thirty, with two recurrences. One of these occurred within three months, and was due to imperfect removal of the sac.

In the other the sac had been drawn through the oblique muscles, and on examination the weakness was found to be at the situation where the sac had been passed through the muscles. The operation of simple sac excision was performed on three males, aged thirty-two, sixty, and sixty-four respectively, and on twelve females without recurrence. The operation by simple sac excision affords a radical cure at any age, and the success of this operation depends as much upon the size and weight of the contents as on the duration of the hernia.

Table showing the number of years the Hernia was present before operation in the cases which recurred.

Reducible Oblique Hernia.

a. *Simple sac excision.*

Age when "swelling" appeared.	Age when operated on.	Duration of swelling before operation.
21	21	Less than 1 year.
28	30	2 years.

b. *Sac excised and conjoined tendon sewn to Poupart's ligament.*

Age when "swelling" appeared.	Age when operated on.	Duration of swelling before operation.
20	21	1 year.
20	23	3 years.
43	46	3 "
Birth	26	26 "

IRREDUCIBLE AND STRANGULATED OBLIQUE HERNIA.

a. *Simple sac excision.*

	Age when "swelling" appeared.	Age when operated on.	Duration of swelling before operation.
(Irreducible)	18	20	2 years.

b. *Sac excised and conjoined tendon sewn to Poupart's ligament.*

	Age when "swelling" appeared.	Age when operated on.	Duration of swelling before operation.
(Strangulated)	Between 1 year and 10 years	58	Over 48 years.
	?	25	?
	29	49	20 "
	21	48	27 "

DIRECT HERNIA.

Age when "swelling" appeared.	Age when operated on.	Duration of swelling before operation.
46	62	16 years.
30	30	Less than 1 year.
46	47	1 year.
30	45	15 years.
38	38	Less than 1 year.

The practice of bringing the stump of the sac through the oblique muscles is unnecessary and predisposes to recurrence.

Closure of the canal in addition to complete removal of the sac.—The inguinal canal was closed by two methods. In the minority the layers of the abdominal wall were sutured behind the cord, which, therefore, ran subcutaneously to the scrotum. In the majority the conjoined tendon was sutured to Poupart's ligament behind the cord and the external obilque closed in front of it to attempt to maintain the valvular action of the canal. The use of interrupted absorbable sutures, tied not too tightly, is to be preferred, for by using these one knows that once primary union is obtained, the sutures will give no further trouble. Silk and salmon-gut sutures often tear through the structures they are supposed to approximate, and later become absorbed gradually by a process of aseptic suppuration which leaves the tissues weaker. Many surgeons, however, use silk because they consider that the fibrous tissue which develops around and finally replaces the sutures causes the approximated

structures to be attached more firmly to one another and better able to withstand strain. A marked proliferation of fibrous tissue was noted in the cases in which unabsorbable sutures were used. Suturing the conjoined tendon to Poupart's ligament in addition to complete removal of the sac was performed on thirty-one males below the age of twenty without recurrence. The after results were not so good, however, as in the thirty-one cases in which simple sac excision was performed: the cord was thickened, the veins were congested, and in many there was a decided weakness at the outer end of the canal with a diffuse swelling when the patient coughed.

Three recurrences occurred in thirty-three operations on males between twenty and thirty, and one in twenty-nine operated upon between thirty and seventy. In twenty females no recurrence occurred.

We may now consider if the conjoined tendon atrophies as a result of suturing to Poupart's ligament. If the conjoined tendon is not thin and weak there is abundant evidence from clinical and pathological sources to show that it does not atrophy, but the mechanism of the canal is altered. The lower muscular fibres of the internal oblique are replaced by fibrous tissue, and the parieties are weakened so that a diffuse swelling appears over Poupart's ligament, which is most marked when the patient strains. Recurrence is not prevented by suturing a thin and atrophied conjoined tendon to Poupart's ligament. There can be no doubt that in the past the canal has been closed as a routine measure in cases in which it was quite unnecessary to do so.

Sac excision is quite efficient in cases which are operated upon early, but in those cases in which the internal ring has become very enlarged or an incomplete sac is present, an attempt must be made to close the canal.

Operation on Irreducible Oblique Hernia.—The contents of the sac were irreducible in thirty-four cases, and of these fifteen were traced. In two cases the sac was excised, and the canal was closed in thirteen. One case in which the sac was closed by a purse-string suture recurred.

Operation on Strangulated Oblique Hernia.—The Hernia was strangulated in eighteen cases. The after history of eleven was traced. In seven the gut was viable and was returned to the abdomen. Four out of these seven cases recurred. Two of the cases in which the canal was closed, and one in which the sac was excised, recurred. Four cases were fatal.

1. Boy, aged 15, right strangulated oblique hernia; testicle in inguinal canal; Meckel's diverticulum in the sac; report does not state how long gut had been strangulated. 2/1/08: Herniotomy, orchidectomy; diverticulum excised. 4/1/08: Conjoined tendon sewn to Poupart's ligament. 5/1/08: Laparotomy; lateral anastomosis of small intestine. 5/1/08: Died; peritonitis.

2. Man, aged 38, left strangulated congenital hernia; sac contained gangrenous pelvic colon constricted at the internal ring. 1/2/08: Bowel resected, lateral anastomosis of ilium to colon. 7/2/08: Colotomy. 7/2/08: Death.

3. Man, aged 54, right strangulated oblique hernia, "very large old-standing hernia." 19/11/08: Very large meal. 20/11/08: Diarrhœa and appearance of irreducible hernia. 20/11/08 Operation; very large sac containing cæcum and ascending colon; gut reduced, sac excised and closed; laparotomy showed peritonitis due to sloughing gut; gut drained. 26/11/08: Died; p.-m.; sloughing gut; Mr. R. P. Rowlands, "old interstitial hernia reduced from one sac into another at the operation."

4. Man, aged 46, right strangulated oblique hernia; Sac contained gut, omentum, and sanguineous fluid. Herniotomy by means of knife and hernia director; peritoneal aspect of gut slightly lacerated at the neck of sac; sewn up. Sac and omentum excised. Died. P.-m., deep suppuration in wound; peritonitis; bronchitis; pneumonia.

The prognosis in strangulated hernia was extremely bad in cases which had reached the stage of fæcal vomiting, which was present in three fatal cases.

Operative treatment of Direct Hernia.—Six of the nine cases of direct hernia were traced. At the operation on all cases the conjoined tendon was sewn to Poupart's ligament. In this variety the hernia is usually diffuse owing to weakness of the

posterior wall of the canal, and strangulation occurs very rarely. The results of operation were very unsatisfactory, for five cases recurred.

In 1905 McGavin[6] suggested the use of silver filigrees in cases in which the probability of cure by ordinary surgical procedures was doubtful, and he has been highly gratified by his results. The presence of the filigree causes an exudation of lymph, which becomes organised so that fibrous tissue grows around and between the wires of the filigree. A solid plaque is thus formed which converts the inguinal region into a sound resistant area which cannot stretch or bulge.

Post-Operative Complications.

Suppuration was mentioned as having occurred in seventeen cases, and was not associated with the use of non-absorbable sutures. Deep suppuration occurred in one case in which the bowel was lacerated. *Hæmatoma* was mentioned in eight cases. It is specially liable to occur when large adherent scrotal sacs are removed. *Hæmorrhage* from the cut end of omentum in one case. *Thrombosis of femoral vein* in two cases of reducible oblique hernia. Four patients complained of chronic pain in the cicatrix. One man had a patch of anæsthesia on the inner side of the groin.

The Causes of Recurrence.

Suppuration, unless it involves deep sutures, does not seem to induce recurrence, for it only occurred in two recurrent cases. A much more important cause is imperfect removal of the sac. There must be no pouch of peritoneum into which abdominal contents may force their way. The ligature round the sac must not slip. A Staffordshire knot is more satisfactory than a purse-string suture. One recurrence resulted from slipping of the ligature, and in two the sac was closed by a purse-string. Dragging up the neck of sac by sutures passed through the abdominal wall and then tied produces a weakness in the muscles which sometimes forms the starting point of a recurrence.

The tension produced by the forcible approximation of the conjoined tendon to Poupart's ligament weakens the muscular fibres above the canal.

Recurrence occurs so rarely in females that it is natural to seek for some anatomical condition which tends to produce recurrence in males. The spermatic cord drags on the inner wall of the internal ring, and if any part of the cord is included when ligaturing the sac, a pouch of peritoneum is formed when the testicle is replaced in the scrotum.

The unsatisfactory results in the direct variety are due to an unsuitable operation. In these cases the use of filigrees would probably produce better results. Other causes of recurrence are: The patient getting up too soon; neglect to relieve tension on sutures by flexion of the hip and knee; post-anæsthetic vomiting.

I must now conclude this short and imperfect treatment of a very important and common surgical condition. It would be difficult to appreciate justly how much one has learned from this investigation, and one cannot help feeling what a great advance in our knowledge would result if a committee were appointed to inquire into the after results of the treatment of medical and surgical diseases by this Society, which is one of the oldest scientific institutions in London.

I should like, in conclusion, to express my gratitude to the Surgeons and Assistant Surgeons for permission to examine their cases, to Mr. C. H. Fagge and Mr. F. D. Saner for many suggestions, and to Mr. E. C. Hughes who kindly examined some of the cases for me.

REFERENCES.

1. McQueen and Williams' Text-book of Operative Veterinary Surgery.
2. Keith's Text-book of Embryology.
3. Keith's Hunterian Lectures on the Upright Posture, its Evolution and Diseases, February, 1913.
4. Lancet, 1912, vol. 2, page 1792.
5. Since this paper was written Murray has published in the Lancet, 1913, vol. 1, page 746, an interesting paper on the ætiology of cysts connected with Hernia Sacs.
6. McGavin, Lancet, 1907, vol. 2, page 1445. Lancet, 1912, vol. 2.

PARASYPHILIS OF THE NERVOUS SYSTEM, WITH SPECIAL REFERENCE TO SOME OF ITS RARER MANIFESTATIONS.

(FROM THE NEUROLOGICAL DEPARTMENT OF GUY'S HOSPITAL.)

By

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Being a Thesis for the Degree of Doctor of Medicine in Oxford University (1912).

FOURNIER[1] applies the name parasyphilis to that group of diseases in which syphilis is the essential cause, but which are only indirectly the result of the syphilitic virus. That is to say, that a person who has had syphilis is liable at some later period to fall a victim to premature decay of certain groups of neurons, but this decay shows itself so long after infection that it could hardly be considered a direct effect. It is possible, however, that this definition will have to be modified. Syphilis, like malaria, is a protozoal disease, and like it, too, is of prolonged, even lifelong, infectivity. That being so, scientific exactitude may come to consider these neuronic degenerations as direct results of syphilis in spite of their extremely tardy development. When the *raison d'être* of Wassermann's reaction is satisfactorily explained, a great step will have been taken in deciding whether

parasyphilis is a direct or only an indirect result of syphilitic infection. Nevertheless, the term "parasyphilis" is a convenient one whereby to differentiate a certain type of neuronic degeneration, due to the long-continued devitalising influence of the virus upon the nervous elements and in which no gross lesion is present to account for it, from those where a gummatous condition, circumscribed or diffused, exists, or where definite vascular changes have occurred.

The following is Fournier's classification of parasyphilitic affections:—

I. *Acquired Syphilis.*

1. Acute hystero-neurasthenia of the secondary period.
2. Different neurasthenic manifestations of a more advanced stage.
3. Tabes dorsalis.
4. General paralysis of the insane.
5. A special form of epilepsy.
6. A special form of muscular atrophy.

II. *Congenital Syphilis.*

Numerous dystrophies, non-developments, malformations, cachexias, possibly some true epilepsies, juvenile tabes, and juvenile general paralysis.

In this very comprehensive category it is strange he found no place for optic atrophy.

The diseases commonly accepted as parasyphilitic affections of the nervous system are general paralysis of the insane, tabes, and tabo-paresis, a combination of the first two.

Ferrier,[2] in his Lumleian Lectures on tabes dorsalis, asserts that he shares with Fournier, Mott, and most modern neurologists their belief in the essential pathological identity of tabes and general paralysis. In his opinion "they are merely different manifestations of the same polymorphic disease." Both these conditions are primary progressive nerve tract degenerations. But there are certain other nervous diseases of this type, and I shall endeavour in this paper to show that in some in-

stances they are parasyphilitic, for we meet with them in patients giving a positive Wassermann reaction, whether syphilis is admitted or denied; and besides, we find them more or less frequently, but at all events sometimes, combined with the recognised parasyphilitic manifestations—general paralysis and tabes. The diseases with which I shall concern myself are three in number, viz.: primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy.

Most of my material has been collected in the various departments of Guy's Hospital, but I am indebted to Dr. T. E. K. Stansfield for permission to investigate cases of general paralysis in the London County Asylum at Bexley, and some of it has been abstracted from various English and foreign publications, the source of such cases being referred to in the text.

All these diseases are of the same primary character, viz., a degeneration of neuronic groups.

Tabes dorsalis is the manifestation of a degeneration of the spinal sensory protoneurons; general paralysis, of the cortical association neurons; primary optic atrophy, of the optic nerve neurons; primary lateral sclerosis, of the upper motor neurons; progressive muscular atrophy, of the lower motor neurons.

In the case of tabes and general paralysis the essential ætiological factor is the same, syphilis; the old, oft-quoted alternative factors, "alcohol, hard work, exposure," are the same. The average interval between infection with syphilis and the declaration of symptoms is the same. The average age incidence is practically the same, the symptoms and terminations are analogous; signs of gross syphilitic lesions are infrequent, since both usually follow mild attacks of syphilis, and the two diseases are frequently combined in one individual. Moreover, both may attack the subjects of either acquired or congenital syphilis.

Some cases of primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy show an extraordinary analogy to the above; extraordinary that is, if they are to have a different ætiology from tabes and general paralysis, but natural enough if considered as parasyphilitic manifestations. Many

give histories of syphilis, others have signs of syphilis remaining on their bodies, or they give positive Wassermann reactions. Evidences of gross syphilitic lesions, too, are rare, since the primary stages have in most cases been mild. Alcohol, hard work, and exposure are, again, amongst the most commonly accepted ætiological factors. The average interval between syphilitic infection and the appearance of symptoms is about the same as in tabes or general paralysis. The average age incidence is practically the same, though in primary lateral sclerosis the onset is so gradual that the patients do not seek advice at the commencement. All three are frequently found associated with tabes, less often with general paralysis; cases occur where two of them are associated together, or where multiple combinations are exhibited. About ten per cent. of general paralytics have tabes, too (Byrom Bramwell,[3] Mott[4]); some four per cent. of general paralytics have primary optic atrophy (Mott[5]); primary optic atrophy is found in from ten per cent. (Osler[6]) to twenty per cent. (Norris and Oliver[7]) of tabetics; and twenty per cent. of tabetics exhibit some form of muscular atrophy (Dejerine[8]). Further undoubted examples of juvenile primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy exist. Holt[9] remarks that there is a group of muscular atrophies attacking children which are the result of spinal degeneration. The progressive muscular atrophy of the hand type (Aran, Duchenne) and the progressive muscular atrophy of the peroneal type (Charcot, Marie, Tooth). The ætiology, symptoms, and course of these conditions point to their being in the same class, though proof that the anterior cornual cells are degenerated is lacking in the latter type. These diseases sometimes attack children, though usually after puberty. The first-named may in its latest stages affect the bulbar nuclei. Some of these cases are very possibly examples of juvenile parasyphilis.

The following remarks respecting alternative or contributory causes and sex incidence apply chiefly to the two parasyphilitic diseases which have been so fully worked out, but if the rarer

forms are investigated with the same care, I have little doubt but that they will be found equally applicable. We shall see later, when dealing with the evidence on the connection between syphilis and parasyphilitic nervous degeneration, that in some 90 per cent. of tabetic patients precedent syphilis can be proved. Nevertheless, when the text-books on medicine are consulted, it is found that numerous alternative or contributory causes are suggested, not only to account for tabes and general paralysis of the insane, but also the other primary tract degenerations. The most usually mentioned of these ætiological factors are a neuropathic diathesis, chill, sexual excess, trauma, alcoholic excess, and bodily or mental overwork. It is to one or other of these that the patient or his friends usually ascribe his condition. In many instances, however, careful scrutiny will bring out the fact that the earliest symptoms of the disease were present before the alleged causative chill, injury, or excess came into operation. The process of decay is often exhibited in the early stages by increased nervous excitability, exaggerated sexual, mental, or intellectual activity—a period of functional irritability, which so impresses the patient or his friends that they interpret it as the cause of the underlying disorder; but this increased activity is soon followed by exhaustion and loss of function. Erb,[10] in discussing these factors in regard to tabes, gave percentages in a large series of cases where one or other of them was the supposed origin of the illness. He found that 28 per cent. suggested a neuropathic diathesis, but that in a good number the only ground for this was insanity in a distant relative. He got a larger percentage—42—of personal neurosis, but dismisses the subject thus: "One could obtain a similar percentage of neuropathic diathesis in persons suffering from a cold in the head or diarrhœa."

Chill and exposure are terms thoughtlessly used, and there is probably no disorder which has not been laid at their door. Erb found it suggested by 34 per cent. of his series. Sexual excess was given by 15·8 per cent., trauma by 5 per cent., and alcoholic excess by 18 per cent. Overwork he considered as

the most important of the contributory factors, but difficult to estimate. He found 27 per cent. attributed the condition to fatigue in such pursuits as war, exploration, and hunting. None, however, of these figures approach the 90 per cent. of syphilis.

Particular stress has been laid by some writers upon sexual excess, the idea apparently originating with the very early investigators of tabes and general paralysis 70 or 80 years ago. If it were a considerable factor one would expect that every prostitute who survived the wear and tear of her calling would eventually succumb to parasyphilis, but there is no evidence of this. Indeed, it has been said that such women do not suffer from tabes or general paralysis. This proposition, however, is erroneous, and has arisen from the fact that women do not give "prostitution" as their profession when seeking advice, but mention some alternative or subsequent occupation. Sexual excess is rather to be regarded as an early symptom than as a cause of nerve tract degeneration. I have recently seen it suggested that, although the neuronic degeneration in progressive muscular atrophy is essentially similar to that in tabes, it is rather to be laid to the account of a "yet-to-be-discovered toxin" than to that of syphilis. This is somewhat like setting up an altar to an unknown god, a proceeding natural enough for the philosophers of ancient Athens, but to be deprecated in an age of exact science. A man proved to have committed a certain crime must necessarily be suspected when that crime is exactly reproduced, but failure to definitely bring it home to him is no justification for hanging someone else about whom nothing is known. If the lesions found in progressive muscular atrophy were utterly unlike those produced by any agent of disease yet known to us, an "undiscovered toxin" would be as fair and scientific an hypothesis as Halley's when he deduced the orbit of the comet which bears his name, and predicted the date of its return. If Erb ever encountered this undiscovered toxin theory he is strangely undemonstrative about it.

It would appear, then, that syphilis is the essential factor in the ætiology of these tract degenerations, though it is not, and probably never will be, proved in absolutely every case. Contributory causes play their part, not in determining the onset, syphilis alone is responsible for that, but in determining which group or groups of neurons shall be primarily affected. There is some evidence to show that where special strain or stress falls upon one definite group, parasyphilis, if it occurs, will be manifested by degeneration of that group.

That syphilis alone is capable of producing tract degeneration is shown by the cases due to congenital disease, in which the age incidence often is too young to allow contributory causes to have much part, and in which the suggestion of such is rare. Still[11] mentions a series of seven juvenile general paralytics, four girls aged 4, 4½, 8, and 9 years at the time of onset, and three boys aged 7, 7, and 10 years. All had well-marked hereditary syphilis. The progress of the disease was typical in each. Two of them had primary optic atrophy in conjunction. Mott[12] gives the results of examination in twenty cases of juvenile general paralysis. The average was 17 years, and there was a history of congenital syphilis in nearly all.

Contributory causes in parasyphilis seem to have been introduced, or at least largely retained, as a sort of compromise from the days of the struggle between the "syphilites" and the "non-syphilites" over the ætiology of tabes and general paralysis. The "non-syphilites" were beaten, but allowed to march out with the honours of war, their colours were not taken from them, but relegated to a position in the rear. And there in all likelihood they will remain. Human nature demands nowadays a remote and an immediate cause for every happening. We know that syphilis can and does, without any outside aid, sometimes produce very terrible effects in its early stages. And it is not unreasonable to presume that when the virus acts through a period of years upon the delicate tissues of the brain or cord, the subsequent degeneration of certain tracts may be solely due to that action. Still, I do not wish to convey the idea that con-

tributory causes play no part, for anything which lowers the vitality or resistance of an individual or certain of his tissues is calculated to accelerate and intensify a slowly progressing degeneration which has already been originated by some other cause. And, as I said above, the function of the contributory factor is to be considered as one of localisation rather than of causation.

Syphilis is known to be the cause of a vast amount of disease—it is even said that it enters into the ætiology of one-third of all the diseases of the world; it has been shown to be the essential cause of the two most common primary nerve tract degenerations. Yet there is a distinct tendency nowadays to hesitate in accepting this wide-spread infection as the main ætiological factor in other tract degenerations, and to put them down to the account of causes with regard to some of which, to put it mildly, there is considerable difficulty in understanding the modus operandi. This tendency may really be due to a delicacy in ascribing conditions of disease to a primary cause which already bears such a terrible load of responsibility: a sort of humanitarianism, a feeling that it may be just the last straw to break the camel's back. Such delicacy is highly commendable when considering diseases where the pathological conditions found after death are not on all fours with those of degenerations of established syphilitic origin; but where they are, as is the case with most examples, of primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy, the delicacy becomes prudery. In investigating a case, the question should be boldly asked, Can two precisely similar results be demonstrated to be due to two totally dissimilar causes? Would it not be more scientific to believe that such and such a history is incomplete, since syphilis is not disclosed, than to scan the different points mentioned in search for one which has conceivably, or inconceivably, been suggested in some text-book?

With regard to sex incidence the proportions given by different compilers of statistics vary between three to one and twenty to one in favour of males. No doubt, truth lies between

At all events, more men are smitten with parasyphilis of the nervous system than women. Why this should be so is rather an interesting study. Beyond question more men acquire syphilis than women. A young man contracting the disease, as is usually the case, from a prostitute, is naturally unable to infect so large a circle as she has the opportunity of doing. When he does pass the infection on, the recipient is probably a member of the same class as the first, and becomes another centre of dissemination for the male sex. So we can only expect to find the disease commoner in men than women. Does this account for the whole discrepancy between the sexes with regard to parasyphilis? Probably not. In the first place, a large proportion of prostitutes never reach the parasyphilitic age. Secondly, very many women who marry syphilised men are protected by Colles' law. The explanation which is often advanced that women are not so liable to contributory causes is not, I think, a sound one, partly because I do not believe contributory causes play a sufficiently large part in the ætiology, and partly because the great majority of women are exposed to stress or strain just as frequently as men, though such stress may take a different form. The sex incidence in congenital parasyphilis is, of course, equal. Still[13] finds that in the cerebral palsies of children the number of boys attacked is to the number of girls as three is to two.

	Boys.	Girls.
Infantile hemiplegia	32 ...	19
Spastic paraplegia	14 ...	12
Spastic diplegia	14 ...	8
Spastic monoplegia	1 ...	0

It will be seen that in spastic paraplegia the sex incidence is almost the same, and this may be significant with regard to congenital syphilis.

I shall not attempt to prove that every example of primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy is parasyphilitic. Primary optic atrophy undoubtedly occurs as a sequela or complication in lead poisoning, some

specific fevers, diabetes, yet it is not an invariable complication and investigation may conceivably show that it is only manifested in persons who have a congenital or acquired syphilitic taint. Also, up to the present so few cases, comparatively, of primary lateral sclerosis or progressive muscular atrophy have been proved to be parasyphilitic. Even in tabes, where the percentage of unquestionably parasyphilitic patients is so high as 90, the other 10 per cent. are not proven; and until they are, one cannot deem unreasonable those who suppose that some other agent may possibly, though very rarely, cause progressive bilateral degeneration in the posterior columns of the cord. For if one poison, to wit, the virus of syphilis, can produce the disease, why should not another under favourable conditions occasionally do the same? The explanation of the neuronic degeneration as due to the prolonged infectivity of syphilis would support the supposition that if perchance some other poison were to exercise an equally protracted influence (as lead, for instance, is capable of doing), it might produce a similar result. Therefore, we cannot yet with absolute certainty proclaim the "no syphilis—no tabes" dictum. Occasionally an example of locomotor is encountered which is typical, is not amenable to treatment, and yet gives a negative Wassermann, whilst no history of syphilis can be elicited. The following is a case in point. Albert L., 35 years of age, has been troubled with staggering gait for some five or six years. Twelve months ago he began to have attacks of giddiness and nausea, and developed perforating ulcers and lightning pains. He noticed the loss of sexual desire nine months ago. His pupils are unequal and of the Argyll-Robertson type. His knee and ankle-jerks are absent. Romberg's sign is present. There is analgesia over the distribution of the ulnar nerves, and Biernacki's sign is present. The left external rectus is paralysed. Wassermann's reaction is negative, he denies syphilis, there are no marks of the disease about him, and treatment with antisyphilitic remedies for a considerable period has in no way benefited him.

That premature decay of nervous elements can occur from some other cause than syphilis is proved by the disease known as disseminated sclerosis. This is a nervous disorder of earlier average age incidence than parasyphilis, and although the causative agent or agents have not yet been unmasked, no connection with syphilis has been made out. The degeneration is not confined to groups of neurons, but seems to occur utterly at random. Loss of the accommodation reflex in the pupil is commoner than loss of the light reflex, and there are no true combinations with the recognised parasyphilitic manifestations. Wassermann's reaction is nearly always negative where it has been tried. Nevertheless, those cases who exhibit signs of lateral sclerosis and primary optic atrophy, and no other signs of dissemination, may be true parasyphilitic combinations; and there is grave doubt when Argyll-Robertson pupils are present. Of course, a positive Wassermann in disseminated sclerosis would not exclude multiple gummata.

I do not, therefore, attempt to gather every case of primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy into the parasyphilitic net. But I think the analogies to tabes and general paralysis, together with the fact of their frequent occurrence in combination with other diseases, constitute strong evidence that at all events some cases are parasyphilitic in character. From time to time articles appear in the literature in which the connection of syphilis with these three diseases is recognised and brought out. Thus, Mott[14] brackets together general paralysis of the insane, tabes, and primary optic atrophy, and describes them as "Really a single morbid entity, owning one cause, with an insidious onset, progressive in character, and uninfluenced by anti-syphilitic remedies; in fact, a primary neuronic dystrophy. They may occur successively or simultaneously in the same individual." And again, "It is probable that Erb's spinal paralysis and certain cases of amyotrophic lateral sclerosis may be primary post-syphilitic dystrophies."

The recognition of progressive muscular atrophy as a parasyphilitic manifestation is much further advanced on the Continent, particularly in France, than in England. Fournier[15] placed a "special form of muscular atrophy" in his classification of parasyphilitic affections. Other observers who have published cases to show the parasyphilitic nature of progressive muscular atrophy are Pousard,[16] Lannois et G. Levy,[17] Leri,[18] Camus,[19] Lannois et Poret,[20] van Gehuchten,[21] Merle,[22] Kinneir Wilson,[23] and Rose et Rendu.[24] These amongst many. The last-named authors point out that amyotrophies in tabes due to peripheral neuritis affect the lower limbs, whereas those of central origin have a preference for the upper extremities, though the legs are not exempt. In the arms the type is usually the Aran-Duchenne, or else the shoulder girdle is most affected. As a rule the degeneration is symmetrical, though one side may be more advanced than the other. Raymond[25] recorded the case of a man who, eleven years after syphilitic infection, suffered from lightning pains in the right shoulder, and four years later developed a progressive atrophy in the muscles of the right hand, spreading to the arm and shoulder. In the following year the left arm was similarly attacked. The muscles at the back of the neck were atrophied equally.

The Cerebro-Spinal Fluid in connection with Parasyphilis of the Nervous System.

Before proceeding further it will be well to give some consideration to the cerebro-spinal fluid, for it is but natural that its composition, reactions, and abnormalities must enter largely into any investigation of the tissues which it bathes. This fluid is a secretion from the choroid plexuses throughout the central canal, but mainly from those of the lateral ventricles. It fills the central canal, and through the foramen of Majendie reaches the subarachnoid space, and is finally absorbed into the lymph stream. It, however, differs in composition from both lymph and blood-serum. It is a clear, colourless fluid with very few cellular elements. There is no albumen in its composition, the only proteid being a globulin.

In syphilis and parasyphilis there is marked lymphocytosis of the cerebro-spinal fluid. This ebbs or flows along with the activity of the disease, and is therefore useful as an index, not only to that activity or quiescence, but to the effect of anti-syphilitic remedies. Their use in parasyphilis produces no good effect on the disease, and no effect in cerebro-spinal lymphocytosis, but in syphilitic meningitis, for instance, as the patient recovers, so the lymphocytosis decreases. This lymphocytosis occurs in some other diseases, *e.g.*, sleeping sickness and Herpes Zoster, and so cannot be considered absolutely pathognomonic, but its presence is a valuable sign when combined with other facts.

In general paralysis of the insane and tabes there is an increase of proteid (albumen) in the cerebro-spinal fluid. If in a case of parasyphilis the amount of albumen, the lymphocytosis and hæmolytic power of the fluid be observed side by side, they will show varying curves during the progress of the disease. Lymphocytosis will be found to be marked at the commencement and during exacerbations, and less in the intervals; but the increase with succeeding seizures shows a progressive diminution to the final stage, by which time the power to react seems to be abolished. The albumen content increases irregularly, whilst the hæmolysis progresses steadily with the disease. It may be absent in the early stages, but when it does occur it is not subsequently lost (see case 19 for exception). There would appear to be no co-relation between the three phenomena.

There are certain precipitation tests which may be carried out on the cerebro-spinal fluid with a view to helping the diagnosis of parasyphilis. Noguchi[26] found that by boiling two parts of cerebro-spinal fluid with five parts of a 10 per cent. solution of butyric acid in normal saline for a few seconds, then adding one part of normal caustic soda solution and again boiling, a flocculent precipitate was produced in syphilitic and parasyphilitic cases. The test is not specific for syphilis, as all forms of meningitis give it, but it will distinguish, for instance, between general paralysis of the insane and dementia præcox.

If equal parts of centrifugalized cerebro-spinal fluid and saturated solution of sodium sulphate are mixed together and boiled, a resultant turbidity will indicate syphilis.

The Wassermann Reaction.

As Professor Wassermann's serum test for syphilis bulks so largely nowadays in the diagnosis of syphilitic and parasyphilitic disease, a short resumé of the principles underlying the test will not be out of place. Pfeiffer's[27] experiments with bacteriolysins gave the clue for the study of immunity. He found that by injecting a guinea-pig with small but increasing quantities of cholera vibrios, the animal became immunized against cholera, so that vibrios subsequently injected into the peritoneal cavity died and disappeared. As there are practically no leucocytes in the normal peritoneal cavity, Pfeiffer concluded that the substance causing the phenomena was a secretion, and as it dissolved microbes he called it a bacteriolysin. Bordet[28] carried on the investigations. He found that when the chromocytes of one species of animal, *e.g.*, a sheep, are introduced into the body of another species, the second animal elaborates a material which enables the serum to dissolve the corpuscles of sheep in general. The serum contains a hæmolysin.

The term antigen is applied to any alien substance (toxin, microbe, chemical) which on introduction into the tissues of an animal sets up therein a defensive reaction, with the production of special agents of defence. The substance so produced is called the antibody. This consists of two parts: one destroyed by heating to 56° C. (thermolabile) known as alexin, cytase, or complement; the other resisting such temperature (thermostabile), and known as the sensitizer, immune body, amboceptor, or simply as the antibody. Multiplication of terms is always confusing, and I shall adhere to "complement" for the thermolabile substance and "antibody" for the thermostabile one.

Complement is always present in the serum of every individual animal in health or disease, whereas antibody is only the product of immunization. Every antigen has its corresponding antibody.

Bordet in 1901 demonstrated the phenomenon of "absorption or deviation of the complement." A serum loses its hæmolitic powers when either the antibody or complement is absent. If the antibody is linked up with the antigen (complement being present), both antibody and complement are rendered inactive.

Wassermann[29] applied this to the diagnosis of syphilis by the existence (as he thought at the time) of syphilitic antibodies and antigens in the serum of syphilitics.

The classic experiments to find the deviation of the complement:—

1. Immunize a rabbit against sheep's blood by repeated injections of washed corpuscles. An antibody is produced which, with the complement normally present in the rabbit's serum, makes the latter hæmolytic to sheep's corpuscles. If the complement be destroyed by heating to 56° C. the serum loses its hæmolytic properties, but they can at once be restored by adding a small quantity of blood serum from some other animal (another rabbit or a guinea-pig, etc.) which contains complement.

2. The second part of the experiment is the neutralization or deviation of the complement, so that hæmolysis no longer takes place when guinea-pig serum is added to antibody and sheep's corpuscles. This is effected by both antigen and antibody being present together in the fluid examined. The suspected serum is mixed in varying proportions with a solution of the liver of a syphilitic fœtus which contains the antigen (lipoid substance). A small quantity of guinea-pig serum containing a known amount of complement is added and made up to 2 c.c. with normal saline. The tubes are incubated at 37° C. for one hour, and then the sensitized blood corpuscles are added (*i.e.*, washed sheep's corpuscles in complement-free serum of immune rabbit). The mixtures are then incubated for two hours, placed on ice overnight, and the amount of hæmolysis estimated. The tubes contained antigen (in the extract of liver), and if the serum to be examined contained antibody (*i.e.*, if it came from a syphilitic individual), the antigen and the antibody will have united with the complement (in guinea-pig serum) before the addition of the

sensitized corpuscles. The complement will have been fixed, and no hæmolysis takes place. If, on the other hand, the serum contained no antibody, the complement remains free to effect the hæmolysis of the subsequently added corpuscles.

In the antigen test the serum is used instead of the extract of syphilitic liver, and tested against a serum containing a known amount of antibody.

Cerebro-spinal fluid may be used instead of the blood serum. Plant considered the tests specific for the diagnosis of the disease, but that it did not indicate the organ affected. If the central nervous system is not implicated, the reaction will usually be positive with the serum and negative with the cerebro-spinal fluid. This is natural when we remember that the latter is essentially a secretion. But even in general paralysis the cerebro-spinal fluid is very occasionally negative whilst the serum is positive. Plant[30] tested both fluids in 95 cases of general paralysis, and got a positive reaction with the serum in every case, and in all but one with the cerebro-spinal fluid.

Such, then, was the original experiment, and the explanation was very simple, and fitted the ascertained facts beautifully. There was antigen without doubt in the syphilitic liver, and if the serum came from a syphilitic patient it would naturally contain the specific antibody, and the two together would also naturally fix the complement of the added guinea-pig serum. A specific test for syphilis had been found, and the satisfaction of the profession was great. And our satisfaction remains, for the test is, to all intents and purposes, specific for syphilitic disease, although we now know that is has nothing whatever to do with specific antigens or antibodies. The beautiful explanation has gone to pieces, and no convincing new one has taken its place. We know the test is a good one, but beyond that all we can say is that some unknown substance links with the lipoids of the liver (used as antigen) and fixes the complement. This unknown is not elaborated by the spirochæte, but by the tissues or individual cells as a result of syphilitic infection. It is not a specific antibody. Its presence

may possibly be due to an alteration in the surface tension of the lipoid cell-envelope.

The original explanation of the reaction was shattered when Lavaditi[31] and Marie[32] found that the syphilitic liver could be replaced by normal liver, that syphilitic cerebro-spinal fluid did not contain any substance which would kill the spirochæte; and then that any normal tissue containing lipoid substances would do as well (Landsteiner and Porges[33]). Finally, that since the lipoids are soluble in 80 per cent. alcohol, an alcoholic extract of normal liver would serve as the antigen. Then Landsteiner, Muller, and Potzl[34] showed that certain malarias also gave the reaction. We must say, therefore, that the reaction is probably specific for protozoal diseases, and in cases where we cannot exclude malaria a positive Wassermann may be due to malaria and not syphilis. Clinically, however, the test may be considered as specific for syphilis, for malarial conditions are not likely to be confounded with syphilitic or parasyphilitic ones, and malarial parasites will sooner or later be discovered in blood films.

Attempts have not unnaturally been made to simplify the procedure and standardize the various materials used in the test, with a view to eliminate errors. Of these simpler methods Hecht's[35] and Fleming's[36] modification of Hecht's procedure are the best known. Many, however, think that the original experiment still gives the most satisfactory results, using an extract of syphilitic liver, guinea-pig serum, washed sheep's corpuscles, and a rabbit. Nevertheless, the source of the material is probably unimportant, the animals employed being chosen for economic reasons. Wassermann's experiment is so complicated that it is only trustworthy in skilled and practised hands. Fleming's modification, on the other hand, is so simple that it requires only care to be successful.

Lipoid Substances.

Lipoids are present in all cells, vegetable as well as animal, perhaps as a sort of cell-envelope, so that when the lipoid is

linked up to an antigen the cell contents are set free. Their presence in the cell seems to be of as great importance to its vitality as the presence of protein. There are three groups of lipoids :—(1) Free from either nitrogen or phosphorus ; cholesterins, fatty-acids, lipochromes ; (2) Cerebrosides, containing nitrogen; but no phosphorus; phrenosin, kerasin; (3) Phosphatides, containing both nitrogen and phosphorus ; lecithin, sphingomyelin.

Attention was directed to lipoids by Flexnor and Noguchi's experiments with snake poison, which they found could not dissolve washed corpuscles, but could do so on the addition of serum. Lecithin was eventually shown to be the active agent, acting, perhaps, as a ferment upon the lipoids of the corpuscles, or, perhaps, so altering their surface tension as to set them free.

By the breaking down of nervous tissues lipoids are set free in the cerebro-spinal fluid and blood, but this does not of itself produce a positive Wassermann reaction ; a specific substance, probably a globulin, must be present too. The syphilitic virus induces an altered metabolism whereby large quantities of lipoids are liberated into the serum. The same lipoids, however, are present in normal serum, the difference being one of amount.

Ætiology of Parasyphilis of the Nervous System.

Esmarck and Jessen[37] in 1857 first suggested a connection between syphilis and general paralysis of the insane, and were laughed to scorn. In 1863 Eisenmann[38] and Topinard[39] propounded a similar connection between syphilis and tabes, and met with derision until Fournier[40] collected cases of locomotor ataxia, and found precedent syphilis in 93 per cent. of them. Gowers[41] published confirmatory evidence, and Erb,[42] who, in 1878, was sceptical as to any frequent connection between syphilis and tabes, afterwards took the matter up on a somewhat grand scale, and published from time to time some remarkable statistics.[43] He investigated 1,100 male tabetics of the better classes, and found that 89·45 per cent. had had syphilis, and that in only 3 per cent. was there no suspicious circumstance. That

was illuminating. Then he took 10,000 cases of diseases of all sorts (excluding only tabes) among the same class of patient, and found only 21·5 per cent. had had syphilis; 138 male tabetics of the lower classes gave 77·2 per cent. of syphilis against the 6·54 per cent. amongst 1,100 non-tabetics of the same class. He further found that 86·7 per cent. of better class tabetic women had been infected, with strong suspicion in a further 6·6 per cent.; whilst among 16 of the lower class female tabetics syphilis was proved in 11, or 68·7 per cent.[44] Minor[45] published a series of eight cases of tabes with a syphilitic history in 100 per cent. In this country, Savage[46] early insisted on the importance of syphilis in the ætiology, and Mott[47] pointed out that the pathogenesis of tabes and general paralysis was so interwoven that if syphilis was the cause of one, it must necessarily be also the cause of the other.

These figures are conclusive, and Wassermann's test only confirms them. They give a history of syphilis—often only after the closest questioning or reference to the medical attendant—in a larger proportion of instances than we get in cases where characteristic gross lesions prove antecedent syphilis in 100 per cent.

We must not forget in this connection the occurrence of syphilis ignorée. Jumon[48] estimated that 40 per cent. of syphilitics were of this class, but this figure probably includes those who persistently deny the impeachment as well as those who are truly ignorant of the infection. Then, although most cases of syphilis occulta,[49] or syphilis d'emblée, eventually betray themselves, there are probably some in whom the condition is not diagnosed.

There has been a similar accumulation of evidence with regard to general paralysis of the insane, so that the dicta, no syphilis —no tabes, no syphilis—no general paralysis, are practically universally accepted by neurologists to-day. Ferrier[50] observed that if it were possible to discover a race of people who had never had any communication with the outside world and to whom syphilis was unknown, but yet suffered from tabes or general paralysis, then, and not till then, could it be said that those diseases occur without syphilis. And Moebius,[51] too—

"The longer I reflect upon it, the more firmly I believe that tabes never originates without syphilis."

Of course, no such voluminous evidence is forthcoming with regard to primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy. These are very much rarer manifestations, and the available cases in support of my contention are but few. Not being considered parasyphilitic, more attention has been paid ætiologically to alcohol, exposure, and particularly to hard work, than to syphilis. Many reports do not mention syphilis at all, or pass it over lightly with "no history of syphilis was given," and very few cases as yet have been subjected to the Wassermann test. When, however, there are signs of tabes or general paralysis in conjunction, then syphilis is inquired after carefully and the blood is tested.

Before tabes or general paralysis were recognised as parasyphilitic the percentage of cases giving the history of syphilis was quite low, but when Fournier, Gowers, and Erb were insisting on its ætiological importance, the figures of competent observers jumped up in a surprising manner. For instance, Bernhardt's[52] rose from 21 per cent. to 60 per cent.; Oppenheim's[53] from 17 per cent. to 80 per cent.; and Rumpf's[54] from 65 per cent. to 80 per cent. The same thing occurred with general paralysis, and it might well be true of the others were more care taken in the investigation of histories and prejudice swept away. Not long ago a case of suspected amyotrophic tabes was brought before the neurological section of a certain learned society. The patient was 36 years old, and gave a positive Wassermann reaction. She had obvious tabes, and, in addition, wasting of the small muscles of the hands, particularly the left hand. The patient was a charwoman, and the section came to the conclusion that the muscular atrophy was due to her occupation, explaining the more advanced condition of the left hand as due—upon what grounds I know not—to greater pressure upon that hand in the process of scrubbing floors.

If one group of neurons can degenerate as a result of antecedent syphilis, why not another group, or any other? If both

tract degenerations can be explained as parasyphilitic in tabo-paresis, why not in tabes combined with progressive muscular atrophy? Yet we find always this seeking for a separate and distinct cause in cases where either tabes or general paralysis are combined with some other tract degeneration.

Of all the conditions of degeneration which, rightly or wrongly, have been laid to the account of syphilis by different writers, I propose to confine myself to pure instances of primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy, and combinations of these either with each other or with tabes or with general paralysis. If any of the other suggested parasyphilitic manifestations are mentioned they will only be referred to incidentally, except in the case of tabo-paresis which I shall take as the prototype of the co-incidence in one individual of two separate parasyphilitic tract degenerations.

My subject can, therefore, be divided into the following two sections, with fourteen sub-sections :—

I. Uncomplicated.

a. Primary optic atrophy.
b. Primary lateral sclerosis.
c. Progressive muscular atrophy.

II. Complicated.

d. Tabes combined with general paralysis.
e. Tabes combined with primary optic atrophy.
f. Tabes combined with primary lateral sclerosis.
g. Tabes combined with progressive muscular atrophy.
h. General paralysis combined with primary optic atrophy.
i. General paralysis combined with primary lateral sclerosis.
j. General paralysis combined with progressive muscular atrophy.
k. Primary optic atrophy combined with primary lateral sclerosis.
l. Primary optic atrophy combined with progressive muscular atrophy.

m. Primary lateral sclerosis combined with progressive muscular atrophy.

n. Cases where more than two of the symptom groups are manifested in one individual.

Some of these are comparatively common affections, others are rare, and, although I cannot cite an actual case of primary optic atrophy combined with progressive muscular atrophy, there is no theoretical reason why it should not exist.

In each of these there is a definite degeneration in brain or cord or nerve trunk which can be anticipated from a knowledge of the symptoms, and can be demonstrated post-mortem.

In a. there will be degeneration of the optic nerve alone.

In b. there will be degeneration of the upper motor neurons.

In c. there will be degeneration of the lower motor neurons.

In d. there will be degeneration of the spinal sensory neurons and of the cortical association neurons.

In e. there will be degeneration of the spinal sensory neurons and of the optic nerve neurons.

In f. there will be degeneration of the spinal sensory neurons and of the upper motor neurons.

In g. there will be degeneration of the spinal sensory neurons and of the lower motor neurons.

In h. there will be degeneration of the cortical neurons and of the optic nerve neurons.

In i. there will be degeneration of the cortical neurons and of the upper motor neurons.

In j. there will be degeneration of the cortical neurons and of the lower motor neurons.

In k. there will be degeneration of the optic nerve and of the upper motor neurons.

In l. there will be degeneration of the optic nerve and of the lower motor neurons.

In m. there will be degeneration of both the upper and lower motor neurons.

In n. there may be degenerations of any combination of these tracts.

The decay of the affected tract may be rapid, as in primary optic atrophy or general paralysis of the insane; or slow, as in tabes, primary lateral sclerosis, or progressive muscular atrophy. Norris and Oliver[55] give the average length of time for primary optic atrophy to progress to total blindness as about one year—it varies from a few months up to 15 years. General paralysis usually ends fatally in from two to three years. The other three conditions may drag on very much longer.

It has been remarked that when two parasyphilitic manifestations are present in one individual there is a tendency for the disease to progress more slowly. Benedikt,[56] as long ago as 1881, pointed out that primary optic atrophy coming on in the pre-ataxic stage of tabes exercised a favourable influence and retarded the symptoms. If this amelioration of the symptoms of one tract degeneration produced by the coincidence of a second is a reality, it would tend to show that both the degenerations were due to one cause. For if two totally distinct causes were at work there could be no reason why one should be favourably affected by the other, whereas, if one virus were, so to speak, dividing its attention between two or more tracts it would have less energy to devote to either one of them, and so each would be retarded.

Some, if not all, of these conditions may be simulated; for example, a man may have the signs and symptoms of spastic paraplegia with a history of syphilis and a positive Wassermann, and yet under the influence of antisyphilitic remedies the condition clears up, thus proving that the condition was syphilitic (*i.e.*, gummatous or due to syphilitic meningitis) and not parasyphilitic. Parasyphilis is unaffected by the antisyphilitic remedies, namely, mercury and pot. iod.; but whether it will be found that it is amenable to salvarsan injections the evidence at our disposal is at present insufficient to determine. Amongst the cases recorded later there is one, Case 19, entirely unaffected by mercury and pot. iod. which, after salvarsan injection, gave a negative Wassermann, though the reaction was positive before. The interval, however, in this case between

the injection and the change in the Wassermann reaction was so short—only two weeks—that probably it is of no importance. It might by itself be looked upon as a hopeful indication seeing that it has been considered a rule that, although parasyphilitics may not at first give a positive Wassermann, when in the progress of the case the reaction has once become positive, the patient does not afterwards give a negative one. A general distinction between a true parasyphilitic manifestation and a simulated one may be drawn thus:—

Parasyphilis.	Pseudo-Parasyphilis.
1. First symptoms not generally noticed till 8 to 15 years, or longer, after primary infection.	1. First symptoms noticed less than 8 years, and often very soon, after primary infection.
2. Average age incidence 37 years.	2. Very many patients are under 25 years of age.
3. Begins usually after all syphilitic manifestations have disappeared.	3. Tertiary or late secondary manifestations may co-exist.
4. Onset insidious as a rule, and progress not very rapid.	4. Onset often sudden, often determined by an accident as a fall or a blow, and progress rapid.
5. History of syphilis, as a rule, shows few and mild early symptoms.	5. History of syphilis often shows a severe attack.
6. Reaction to antisyphilitic remedies nil or injurious.	6. Reaction to antisyphilitic drugs marked and beneficial.

Head[57] suggests that in spinal cases where the onset is sudden, the prognosis is bad. But in parasyphilis the prognosis is bad whether the onset be sudden or slow. In syphilitic disease of the cord the prognosis depends more upon the lesion than upon the suddenness or otherwise of the attack. In thrombosis and hæmorrhage the onset is likely to be most sudden, and certainly in such the prognosis is grave, for it is difficult to see how antisyphilitic or any other remedies are going to remove thrombus or clot, at all events in time to prevent permanent damage to the nervous elements from softening or compression. But in cases where the symptoms are due to gumma or thickening of meninges, recovery is by no means rare under antisyphilitic treatment.

Wassermann's test is of great value in the differential diagnosis of parasyphilis and pseudo-parasyphilis, as the following three cases mentioned by Mott[58] indicate:—

1. A woman was admitted to an asylum as a general paralytic; she exhibited cerebro-spinal lymphocytosis, but the Wassermann was negative. Under treatment by mercurial inunction the symptoms of general paralysis cleared up.

2. A man was admitted to an asylum as a general paralytic. He improved so markedly that he would have been discharged as cured but for the fact that his cerebro-spinal fluid gave a positive Wassermann. Suddenly he began to have seizures and died. Post-mortem his brain showed lesions of general paralysis of the insane.

3. A woman with definite tabetic symptoms, but who had probably been infected less than four years before, showed intense cerebro-spinal lymphocytosis, but a negative Wassermann. The symptoms cleared up under mercurial inunction. This case shows the value of giving antisyphilitic treatment a trial in apparent parasyphilis of the nervous system with a negative Wassermann.

It is commonly said, and observation tends to confirm the view, that parasyphilitic nervous disease, as a rule, follows an apparently mild attack of syphilis. If, however, we consider the terminal nervous degeneration as really an ultimate part of the attack, it cannot in its totality be described as mild. Fournier[59] says, "If the virus expends itself in severe primary or secondary manifestations there is less tendency to parasyphilis afterwards." Mott[60] was surprised at the rarity of tertiary lesions in over 500 post-mortem examinations on general paralytics. He says further that, in spite of the well-known debauchery and sexual promiscuity of many general paralytics in the early stage of the disease, he has never seen a primary sore or secondary rash on such a patient. Krafft-Ebing[61] confirms this observation, and puts it, as well as the no syphilis—no general paralysis dictum, to a severe test by selecting nine patients suffering from general paralysis of the insane who

gave no history of syphilis and who had no physical signs of the disease about their bodies, and inoculating them by injecting the virus from a typical hard chancre. None of them developed syphilis, showing that they possessed a specific antitoxin, that they had become immune by a previous infection.

Syphilis established in a race tends to produce racial syphilisation with consequent inherited immunity; and, as a result, we see fewer cases of severe syphilis than in former generations. Yet there are more cases of tabes and general paralysis. May this not be because the population increases, and consequently there are more cases of syphilis than formerly although of a milder character, and parasyphilitic degenerations are more apt to follow mild attacks in which the virus has not spent itself riotously in the early stages?

In this connection the history of syphilis in Europe is interesting; and Dr. Norman Moore[62] has published a very learned paper on the subject just recently. It appears to have been introduced from somewhere about the end of the 15th century, and although Columbus is often credited with having imported the disease from America it is very doubtful whether he had anything to do with it, for examination of old skeletons in America has not revealed the presence of syphilitic lesions (Dr. Ales Hrdlicka); and it is known that later the disease caused great havoc amongst the Indians. And here Colonel Lambkin's[63] observations in Uganda are important as showing how intense is the reaction to syphilis of a race previously free from the disease. The same thing was observed in Greenland on the introduction of the disease there. There is a remarkable divergence of opinion with regard to Galen. One writer will confidently aver that Galen knew not syphilis, whilst another, with equal assurance, will insist that this or that passage in his writings obviously refers to syphilitic lesions. I am afraid there is a distinct tendency to exploit Galen and other ancient and mediæval writers, and that advantage is taken of our frequent inability to be sure exactly what they *are* talking about, to make them prove both sides of a question in one breath. Moore con-

cludes that no passages exist which prove ancient Latin or Greek writers to have been acquainted with syphilis. He lays stress on the absence of records of symptoms of general paralysis or tabes, but, as these parasyphilitic diseases do not appear until a race has become to some extent syphilised, it is conceivable that syphilis might have been known though parasyphilis was not. Erb,[64] on the other hand, in speaking of tabes dorsalis, says, "This disease was undoubtedly known to the most ancient physicians, and frequently enough came under their observation. Among the forms of spinal disease which are more or less definitely spoken of under the name of tabes, or phthisis ischiadica, by Hippocrates, Galen, Bonetus, and others, there were undoubtedly cases of sclerosis of the lateral columns"; but he admits that "It is hardly worth the while to trace out the reports of the older physicians on these forms of disease, for we shall nowhere find anything like an accurate characterisation of the same, or a correct distinction between the different forms."

Why do the effects of syphilitic inoculation differ so widely? Why should the disease in one patient with apparently a mild attack lead, after a period of entire quiescence, to such grave and devastating degenerations of the nervous system, whilst another, with what appears a far more severe infection, develops no nervous symptoms in after life at all? Is the difference in the spirochæte pallida, or in the patient's resistance, the defensive reaction excited in the tissues by the presence of the virus? And, if it is a question of resistance, is it the resistance of the organism as a whole or of the individual tissues? Comparison with malaria indicates the possibility of the existence of varieties of the spirochæte. Or the virus may be modified by passage through certain individuals, or the treatment of the disease may modify the virus. This last, however, is hardly likely, seeing that precisely the same ultimate nervous degeneration may overtake a man who never had any treatment with mercury at all as one who underwent a prolonged course; unless we allow that the mercurial modification of the virus occurred during passage through a previous individual. The following remark-

able instances, quoted by Mott,[65] point strongly in the direction of there being at all events a special neurotoxic variety of the spirochæte, or a special neurotoxic modification of the virus.

Two students were infected with syphilis on the same day by the same woman; both died 15 years later of general paralysis (Babinski). Two men were infected with syphilis about the same time by the same nurse; both were general paralytics 10 years afterwards (Mott). Two men acquired syphilis from the same source about the same time; 10 years later both were subjects of tabes dorsalis (Marie and Bernard). Four men were infected with syphilis by one woman, and later all suffered from either tabes or general paralysis. A fifth man who also had connection with this woman, but did not contract syphilis, did not develop either tabes or general paralysis (Erb).

Seven glassblowers were under treatment together for chancre of the lip. Ten years later five of them came under observation, of whom four were suffering from tabes or general paralysis (Brosius).

This extraordinary example is given by Morell-Lavellée:—[66]

Marthe X.—In 1870 the mistress of primus (medical student); he died in 1873 of syphilitic meningitis.

In 1871 the mistress of secundus (medical student); he married later and had healthy children, but died in 1888 from general paralysis.

In 1872 the mistress of tertius (medical student); he married later and had two healthy children, but died in 1882 from general paralysis.

Later the mistress of quartus (chemist); he died in 1890 from general paralysis.

Later the mistress of quintus (engineer); he died of folie syphilitique.

The whole five being infected with syphilis by this woman.

Yet, experiments on animals do not uphold the theory of variation in the spirochæte, or of modification of the virus so as to be specially toxic to given tissues; for there appears to be no difference in the lesions produced by inoculating apes with

virus obtained from a wide range of syphilitic lesions. And a chimpanzee inoculated with virus from a syphilised monkey (Macaque)—which does not react well to the disease, and might, therefore, be supposed likely to modify the virus by passage—shows similar and equally severe lesions as one inoculated directly from a human being. Therefore, until further light is thrown on the subject we must consider that the variations in the symptom-complex are due, not to the virus, but to the differences in the resistance of the individuals attacked, difficult as it undoubtedly is to explain the above quoted instances simply as coincidences.

Profeta's law—that a non-syphilitic child of a syphilitic mother does not acquire syphilis from the syphilitic mother who suckles it; and Colles' law—that a non-syphilitic mother does not contract syphilis when suckling her syphilitic child, whereas a wet nurse does—prove that immunity is attained by the child or mother acquiring some anti-toxic substance from the other.

In a large number of cases of congenital general paralysis, Mott[67] got histories of the mother having frequent miscarriages, and bearing typical syphilitic children, without herself acquiring the disease. In two instances the mother died of general paralysis, whilst a considerable number of the fathers succumbed to this disease. The usual conjugal history of the parents of juvenile parasyphilitics is of this type:—First a longer or shorter series of miscarriages, then children born dead, and children dying in infancy—often from hydrocephalus or meningitis, then children apparently healthy (but who later show premature nervous decay, suffering, for example, from nerve deafness, optic atrophy, tabes, or general paralysis), and finally healthy children. Indicating either that in the course of time the virus becomes attenuated, or that the resistance of the offspring increases in strength, or that both these processes are in operation together. Occasionally, however, a child with signs of inherited syphilis, or a parasyphilitic child, may be born out of its turn, as it were, after several healthy ones. Lavaditi has actually observed the spirochæte pallida within the ovum, and the pheno-

menon may be explained by the virus attacking one ovum and not another, or by this particular ovum having less resistance than previously fertilised ones.

How can the degeneration of particular groups of neurons be accounted for? Why should one parasyphilitic individual suffer from tabes, another from general paralysis, others from primary optic atrophy, primary lateral sclerosis, or progressive muscular atrophy, or others from combinations of two or more of these together? In direct syphilis of the nervous system the particular position of the lesion is probably fortuitous, there being an accidental metastasis of the organism at that spot. This cannot be the case in parasyphilis, for though a variety of symptoms may be present, yet the very constant occurrence of degeneration of the lenticular ganglion, as shown by the phenomenon of the Argyll-Robertson pupil—which is probably only met with in parasyphilis of the nervous system—shows that the incidence cannot be random and uncontrolled. Neither spirochætes nor antigens have ever been demonstrated in the cerebrospinal fluid (though inoculation of apes with that fluid is said to have been followed by the development of syphilis), but antibodies are found, or at least the unknown substance which acts the part of antibody in Wassermann's reaction, and probably in direct proportion to the neuronic decay. Nerve cells are not capable of regeneration, though their processes are; and when decay sets in, it begins in the dendrites and travels back finally to the cell and its nucleus. Most infections are short-lived and fleeting in effect, and the tissues attacked are not usually so injured as to preclude recovery sooner or later. But syphilitic infection is unfortunately prolonged; at the best severe; and the individual attacked never knows when he is at the end of his troubles; often it lasts till the end of the victim's life, and very frequently either directly or indirectly brings about his death. When such a prolonged evil influence is brought to bear on nervous tissues it leads to permanent and irreparable damage, premature decay, and death.

Bacterial infections may be important in determining the locality of parasyphilitic degeneration, by lowering the resistance of one group of neurons more than another, and they may also be the immediate factor in the onset of the disease, or produce aggravation of symptoms and acceleration of decay. Parasyphilitics not only readily fall victims to microbic infections, but are especially liable to be carried off by them, or to show increased rapidity in the progress of the disease as a result of such attack. Possibly the toxic effects of secondary bacterial infections are greatly increased by the presence of the increased quantity of lipoids in the blood consequent on the nervous decay.

Degeneration of the lenticular ganglion, which Marina[68] proved to be the cause of the phenomenon known as the Argyll-Robertson pupil, where the pupil reacts to accommodation but not to light, is the most constant physical sign of parasyphilis of the nervous system, and may be almost said to occur in no other disease. Babinski, I think, first pointed out that the phenomenon of the Argyll-Robertson pupil is not confined to tabes or general paralysis, but may be found in other post-syphilitic states.

It may well be, when, in course of time, parasyphilitic manifestations come to be more fully recognised and classified, as tubercular manifestations are classified, that degeneration of the lenticular ganglion will hold pride of place as the commonest of them all. Whether it ever occurs as the only sign is difficult to determine; patients do not seek advice for loss of light reflex. It is so commonly found with tabes, general paralysis, and primary optic atrophy as to be regarded as a symptom of all three, but in the case of tabes, at all events, it is difficult to see how degeneration of the ciliary ganglion can be in any way the result of degeneration in the posterior columns of the lower cord, and yet the Argyll-Robertson pupil is often found so early in the disease that the only other sign may be an absent tendo-Achillis-jerk. It is more easy to understand the ganglion degeneration as a concomitant of the spinal degeneration, owning a common cause with it, than as its result. Similarly,

in general paralysis with the phenomenon, both are probably separate manifestations of a primary condition. With primary optic atrophy if the pupil light reflex were not lost till after blindness had occurred, it might be argued very fairly that it was due to the non-perception of the light stimulus, but it is found before vision is lost. And so, again, we may have two results of one protean disease.

The motor nerves of the extrinsic ocular muscles are frequently affected in parasyphilis, producing ptosis and various ophthalmoplegias. These may be due to a peripheral neuritic condition, but in some cases where the paralysis does not vary, as it does in the neuritic ones, the disorder may be due to a lower neuron degeneration, a parasyphilitic progressive muscular atrophy, in fact, merely a "different manifestation of the same polymorphic disease."

If a number of the different groups and systems of neurons of which the central nervous system is made up are liable to degeneration as the result of syphilis, or even all of them, clinical experience yet shows that some of these groups are more prone to be affected than others, have less resistance than others. Individuals vary as to which set is affected first and to the greatest extent, but we shall not be far wrong if we place the groups in the following order, beginning with the least resistant: 1. Ciliary ganglion. 2. Spinal sensory protoneurons. 3. Cortical association neurons. 4. Optic nerve. 5. Pyramidal tracts. 6. Anterior cornual cells, lower motor neurons.

If organic life could be prolonged sufficiently, independently of the central nervous system, we might expect to find that every case of parasyphilitic nervous disease, in whatever tract it commenced, would progress more or less rapidly, by degeneration of neuronic community after community, to complete nervous death.

Fournier[69] apparently makes a mistake in his classification of parasyphilitic affections in including the first sub-division. For early neurasthenia after syphilitic infection is amenable to antisyphilitic remedies, and, therefore, not parasyphilitic. "But a

neurasthenia occurring in a syphilitic patient in the tertiary stage is very persistent, and always results in permanent disturbance of the nervous system" (Fournier). This form is never benefited by antisyphilitic remedies. Such cases are to be considered as early parasyphilis. For though, as a rule, parasyphilis of the nervous system does not manifest itself until 8—15 years after infection, yet cases of an undoubtedly parasyphilitic nature do sometimes show their first symptoms within even two or three years after infection. Parasyphilis, in fact, conforms to no rules. There is, however, I think, evidence to show that there is a tendency where syphilis has been acquired early in life for parasyphilis to appear late; and where the patient is more advanced in age at the time of infection for the interval until parasyphilis shows itself (the incubation period of parasyphilis we might call it) to be shortened. Erb[70] calls attention to this latter point, but the instances he gives are not of much value seeing that amongst them the lowest age at which infection occurred was 48. Still, in that case, the "incubation period" was the longest of the series, which is as follows:—

1.	Syphilis	acquired	at	48,	tabes developed	at 58.
2.	„	„		57,	„ „	66.
3.	„	„		54,	„ „	59.
4.	„	„		48,	„ „	54.
5.	„	„		55,	„ „	57.
6.	„	„		55,	„ „	58.
7.	„	„		68,	„ „	70.

The interval of time between infection and the first appearance of symptoms of parasyphilitic nervous disease varies from 3 to 30 years, though exceptional cases may be found outside these limits, as in the fifth and seventh of the above series, where the interval was only two years, and in case 9, recorded later, in which the interval was as long as 50 years. But the average is 8 to 15 years (and the average age incidence is 37); it depends on the intensity of the virus and the efficiency of the resistance. Ceteris paribus, one would expect the incubation period to be longer when the disease is acquired young, for the

younger the patient the greater will be his resistance, and the longer will he be able to hold out against the enemy. On the other hand, cerebral and spinal syphilis may occur at any period after infection, and a large number suffer before they are 25 years old.

In congenital syphilis the first manifestation of parasyphilis usually occurs about puberty, but this is by no means constant, for it may develop much earlier, as in Still's cases quoted above,[11] and it may well be that some of the cases of parasyphilis developing during the third decade, where acquired syphilis is denied and cannot be demonstrated, are due to hereditary disease. It would even seem possible that some much later cases are of the same type, for Christian Muller[71] has recorded two virgins who developed general paralysis at the end of the fourth decade, and died therefrom at the ages of 42 and 43 respectively.

Fournier considers as parasyphilitic all cases of epilepsy in which the first symptom appears after the age of 35 years. Rumpf[72] puts the same proposition less dogmatically, and says that it is rare for epilepsy occurring after the age of 30 and progressing to motor paralysis to be other than parasyphilitic.

Fournier gives the following example. A man, aged 45, who had suffered from a mild attack of syphilis 20 years previously suddenly developed grand mal which recurred at intervals for many years, with frequent attacks of petit mal interspersed. He exhibited no other symptoms. Max Nonne[73] says he has seen several such cases, and relates two where epilepsy preceded locomotor ataxia. 1. A man, aged 45, two and a half years after infection with syphilis developed epileptic attacks occurring three or four times a month. A year later primary optic atrophy appeared and lightning pains, the case progressing to one of tabes. 2. A man, aged 40, four years after infection developed epilepsy. Three years later he developed definite tabes with absent knee-jerks, typical pupils, and ataxy. Neither of these cases were affected by mercury or pot. iod.

Head[74] records a case which is probably one of parasyphilitic epilepsy. A man, aged 39, had a chancre 13 years previously,

for which he was treated with mercury for 18 months. Three years after infection he began to have attacks of minor epilepsy consisting of momentary unconsciousness. Six years later attacks of grand mal began. Each attack was preceded by hours or days of intense headache. The jaw went over to the right and unconsciousness followed. There were no abnormal signs in the nervous system; the discs were normal. Further discussion of parasyphilitic epilepsy, interesting though it is, would be outside the scope of this paper, and I will proceed to deal, seriatim, with the various sub-divisions into which I have previously tabulated my subject. It will, however, be more convenient to take them in a different order.

Tabo-Paresis as the Prototype of Combined Parasyphilitic Disease of the Nervous System.

If tabes and general paralysis are simply different manifestations of one polymorphic disease, if, in fact, they are, as is now frequently stated, really one disease, it is perfectly simple to understand the combination of symptoms which we see in cases of tabo-paresis. Though the variations are so numerous, they are governed by the varying intensity of degeneration at different points throughout the affected neuronic systems. When we consider how many different functions may be influenced more or less seriously by degeneration of the posterior columns of the cord, or of the association neurons in the cortex, we see why tabo-paresis does not conform to one type; why, indeed, it is almost impossible to find two cases precisely alike. When the two "contracting parties" are subject to almost innumerable variations, the results of their union must be more variable still. And so we find general paralytics with absent ankle-jerks, or with ataxic gait, or with perforating ulcers, or lightning pains, or loss of sexual power, or exhibiting Romberg's sign, or with two or three or all of these together indicating decay of the spinal sensory tracts in addition to cerebral degeneration.

Case 1 is an example of general paralysis of the insane with one sign of tabes. Alfred G., 50, had syphilis when 18. He gives

a positive Wassermann as well as the cerebro-spinal precipitation tests. He has well-marked mental signs and tremors of general paralysis, his pupils are unequal, irregular, and of the Argyll-Robertson type. There is slight ptosis on both sides. His gait is normal, but his knee jerks are absent.

Case 2 is a similar instance in a man who gave no history of syphilis. George C., 38, gives a positive Wassermann reaction both with serum and cerebro-spinal fluid; the latter also shows lymphocytosis and gives the precipitation tests. He has the mental symptoms, tremors, and slurred speech of general paralysis. His pupils are unequal, irregular, and of the Argyll-Robertson type. His gait is normal, but his knee-jerks are absent.

The tabetic signs may be, much more rarely, manifested in the upper extremities and not in the lower. A general paralytic may exhibit Biernacki's sign, that is, loss of sensibility to pressure on the ulnar nerve behind the elbow, or he may be anæsthetic over the distribution of the ulnar nerve; he may have loss of joint sense, not knowing where his limbs are placed without looking, or loss of muscle sense, being unable to distinguish between the weights of different objects of similar size, or he cannot with his eyes shut recognise an object placed in his hand, or his arms may be inco-ordinate in their action. On the other hand, we may have a tabetic patient who has, in addition, one or more signs of general paralysis of the insane. Perhaps he has grandiose ideas, or his speech becomes slurred, or he develops tremors of the tongue or lips, or some form of aphasia may appear. Naturally, tabo-paretics are as liable to have Argyll-Robertson pupils as patients suffering from general paralysis or tabes uncombined.

Case 3.—Here the spinal symptoms are the more marked. Henry C., 47. He admitted syphilis, but could not tell the date. Wassermann's reaction is positive and his cerebro-spinal fluid shows lymphocytosis and precipitation tests. His gait is ataxic, he exhibits Romberg's sign, he has a perforating ulcer, and his knee-jerks are absent. His pupils are unequal, irregular, and

of the Argyll-Robertson type. He further shows distinctly the mental signs of general paralysis of the insane.

Case 4 is similar, but without a history of syphilis. Frederick P., 45. Wassermann reaction and cerebro-spinal precipitation tests positive. His gait is unsteady, he shows Romberg's sign, and his knee-jerks are absent. His pupils are unequal, irregular, and of the Argyll-Robertson type. In addition he has the mental symptoms and slurred speech of general paralysis well marked, but there are no tremors. Such, then, is the picture when the two commonest manifestations of the one polymorphic disease are combined together.

Of the three conditions—primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy—the first is by far the most common. It is so frequently found in conjunction with tabes, and is so often manifested in congenital syphilitics, that although some neurologists—notably Mott—recognise it as "merely a different manifestation of the same polymorphic disease," it is surprising that it has not long ago been generally accorded a position amongst parasyphilitic manifestations. Yet, when it has occurred in combination it has simply been considered as a symptom, and when alone frequently as "optic atrophy of unknown cause."

Primary Optic Atrophy as a Parasyphilitic Manifestation either combined or alone.

a. *Primary Optic Atrophy combined with Tabes.*—Just as we saw, in speaking of tabo-paresis, that there is the utmost variety in the cases according to the exact position and extent of the neuronic degeneration, so in primary optic atrophy with tabes we may get every gradation in the intensity of tabes shown, from mere loss of tendo-Achillis-jerks to well-marked locomotor ataxia.

Norris and Oliver[75] compute that 20 per cent. of tabetics have primary optic atrophy, though Osler[76] only suggests 10 per cent. This usually develops in the pre-ataxic stage, but it may pre-

cede the first symptoms of tabes by from 2 to 20 years, and occasionally does not occur until the late stages of the latter disease.

Case 5.—Here primary optic atrophy is the first manifestation and tabes is only just commencing (Turtle[77]). A man, aged 37, had syphilis 17 years ago. His sight began to fail four years ago, and for the last four months he had shooting pains in the left leg. There was marked primary optic atrophy, most advanced in the left eye. The pupils reacted to light. There were slight Rombergism and ataxia. The tendon reflexes were normal, except that the right knee-jerk was diminished.

Case 6 is an example of primary optic atrophy with absent ankle-jerks. William H., 35, contracted syphilis 16 years ago, and gives a positive Wassermann. His sight began to fail two and a half years ago, and his left eye is now quite blind. There is primary optic atrophy in both eyes, the left more advanced. There is some ptosis. His pupils are unequal, eccentric, and of the Argyll-Robertson type. He is not ataxic, and Romberg's sign is not present, though he cannot stand on one leg with the eyes shut. His knee-jerks are present, but the ankle jerks are lost.

Case 7.—Here the tabes is more marked. As this man has no perception of light, the loss of pupil reflex to light may be ignored. Edward T., 27. No history of syphilis, but there is a question whether he is not the subject of congenital disease. He gives a positive Wassermann. He gradually lost his sight nine years ago, and is now quite blind. Primary optic atrophy was the diagnosis of the oculist who saw him at the time. Six years ago he began to suffer from lightning pains. His pupils are unequal, irregular, and of the Argyll-Robertson type. Romberg's sign is present, and the ankle-jerks are absent. (This man's wife has suffered from keratitis, but has had no miscarriages.)

Case 8 shows tabes still more advanced. Edward M., 33, acquired syphilis 14 years ago. Gives a positive Wassermann. Failing sight first symptom. There is primary optic atrophy in

both eyes, the pupils are of the Argyll-Robertson type. He suffers from vesical crises, his gait is ataxic, Romberg's sign is present, and the ankle-jerks are lost.

Case 9.—This man is interesting on account of the extreme delay in development of parasyphilis. George B., 66, had syphilis 50 years ago. He gives a positive Wassermann. Pupils unequal and fixed. Primary optic atrophy in the left eye. Paresis of the left internal rectus. Both knee and ankle-jerks are absent. There is ulnar anæsthesia, and analgesia of the calves.

b. *Primary Optic Atrophy combined with General Paralysis.*—This combination is by no means so common as the last, primary optic atrophy being present in not more than 4 per cent. of pure general paralytic patients; but it is present in a very considerable proportion of tabo-paretics and of juvenile general paralytics. Most of the latter find their way into idiot asylums, via the blind schools, as epileptic imbeciles. Here, again, we may find one case where primary optic atrophy develops in the course of typical general paralysis, and another where the optic atrophy was the first and most definite manifestation of parasyphilis, the latter being by far the commoner.

Case 10.—Mott[78] records the following example amongst others. A girl, aged 15, the subject of hereditary syphilis, was bright and intelligent up to 13. She then developed double primary optic atrophy and went quite blind. She is now mentally and physically infantile, wet, and dirty. Her knee-jerks are unobtainable owing to rigidity of the legs. The pupils do not react to light. She is suffering from progressive dementia.

Case 11.—A woman, mentioned by Head,[79] E. L., aged 23, the subject of congenital syphilis (Hutchinson's teeth, scars at angles of mouth). At six years of age she went blind from primary optic atrophy. At 20 she degenerated mentally; from being amenable and industrious she became careless and incompetent, in fact, she became socially ataxic. There is no paresis, the reflexes are normal, but her tongue is slightly tremulous.

c. *Primary Optic Atrophy alone.*—Primary optic atrophy is a disease of fairly frequent occurrence, and although it is often found in the subjects of congenital syphilis, when it is met with in the adult it is not usually looked upon as parasyphilitic, even where there is no suspicion of diabetes or other possible cause. The following cases, I think, strongly suggest its parasyphilitic character; none of them had any symptoms of neuronic degeneration beyond the optic atrophy.

Case 12.—Sidney P., 28, had syphilis 10 years ago. Gives a positive Wassermann. His pupils are normal, but he has double primary optic atrophy. All his tendon reflexes are normal.

Case 13.—James E., 49. No history of syphilis, but he gives a positive Wassermann. He has Argyll-Robertson pupils and primary optic atrophy. His deep reflexes are normal.

Case 14.—William E., 40. No history of syphilis, but he has given a positive Wassermann reaction three times. His cerebro-spinal fluid shows no lymphocytosis. He has double primary optic atrophy. His pupils are normal, though they react only sluggishly to light. All his reflexes are normal. Indican and glycuronic acid have been found in the urine, but no sugar by any test. He is a very heavy smoker. His wife has specific ophthalmic lesions, old corneal opacities, iritic adhesions, choroidal atrophy, but they may be congenital. She has one healthy child, and has had no miscarriages.

The next two are congenital syphilitic children, with no symptoms beyond primary optic atrophy.

Case 15.—Ellen M., 15. Has periosteal nodes and other evidence of hereditary disease. She gives a positive Wassermann, and her cerebro-spinal fluid shows lymphocytosis. Her pupils are dilated and fixed. She has double primary optic atrophy. All her tendon reflexes are normal.

Case 16.—Frederick H., 7. He shows evidence of hereditary syphilis, and gives a positive Wassermann. His pupils are normal, reacting sluggishly to light. He has primary optic atrophy. All his reflexes are normal.

Either of these cases may, of course, develop into juvenile tabes or general paralysis, but at present there is no indication of it. In none of these five cases of primary optic atrophy was there anything, remote or immediate, except syphilis which could be considered the cause of the condition, unless the excess of tobacco in case 14 be so regarded.

PRIMARY LATERAL SCLEROSIS AS A PARASYPHILITIC MANIFESTATION EITHER COMBINED OR ALONE.

a. *Primary Lateral Sclerosis combined with Tabes.*—Instances where signs of involvement of the lateral columns are noted in conjunction with tabes have been diagnosed as postero-lateral sclerosis or ataxic paraplegia. But the tendency has been to assume some syphilitic or traumatic thickening of meninges compressing the lateral columns to account for the paraplegia, and to refuse the combined disorder admission to the jealously-guarded parasyphilitic portals. It would seem, however, simpler to explain the precisely analogous degenerations of two neuronic groups, lying side by side in the spinal cord, as due to one primal toxic influence than to find a separate cause for each.

As with other parasyphilitic amalgamations, the intensity of the components varies within wide limits. So that cases of tabes where there are no more than one or two signs of lateral sclerosis, and of primary lateral sclerosis with very early tabes, come under the same heading as those in which both diseases are well marked. The following cases illustrate varieties of this combination.

Case 17.—In this man with primary lateral sclerosis the sensory disturbance points to the development of tabes in combination. Robert M., 53. Wassermann positive. He denied all venereal disease till told of the significance of this test; he then admitted "gonorrhœa" 25 years ago. His pupils are normal, the knee and ankle-jerks are exaggerated, ankle clonus is present, and both plantar reflexes are extensor. In addition he has loss of power, with loss of tactile and painful sensations, in both legs; there is difficulty in micturition and weak-

ness of abdominal muscles. Antisyphilitic treatment for a year was not beneficial, and injection of salvarsan gave no result.

Case 18.—Francis M., 29, had syphilis six years ago, and was treated for two years. His cerebro-spinal fluid shows lymphocytosis. His first symptom was staggering gait twelve months ago. A few days before admission he was thrown from a van and fell on his back. He was unable to walk for two days, but that passed off. On admission he had pain in the back (D 12), weakness in the legs, and difficulty in starting micturition. His pupils were unequal, but reacted. There was no anæsthesia. His gait was inco-ordinate and spastic, Romberg's sign was present. His knee-jerks were exaggerated, ankle clonus was present, and Babinski's sign was marked. There were no intention tremors nor nystagmus. Antisyphilitic remedies had no effect on his condition, although the pain in the back, which was probably due to the fall, disappeared.

Case 19.—William J., 48, had syphilis fourteen years ago. Wassermann positive, and lymphocytosis present in the cerebro-spinal fluid. Four years after infection the patient was exposed to considerable hardship in the Boer war, and his right leg became weak and "dragged." This remained much the same for three years; then he had a course of antisyphilitic treatment without improvement. Six years later he gave a negative Wassermann and was treated with Faradic baths. Twelve months after that the Wassermann reaction was positive, and he came into hospital for salvarsan treatment. The pupils are normal. Knee-jerks are absent. Plantar reflexes extensor. His gait is inco-ordinate, Romberg's sign is present, both legs are analgesic, but not anæsthetic, the right leg is rather wasted, but there are no trophic lesions. He has lightning pains. No ulnar anæsthesia. A fortnight after salvarsan injection the Wassermann reaction was negative, although it had been positive a month before. The importance to be attached to this, since the interval is so short, is doubtful.

b. *Primary Lateral Sclerosis combined with General Paralysis.*—Cases have been recorded where general paralysis has

developed in a patient already suffering from primary lateral sclerosis (Osler[80]), and certainly general paralytics may, and often do, exhibit exaggerated reflexes, whilst sometimes Babinski's sign is well marked. When that occurs there is no doubt that organic disease of the lateral columns is present.

Case 20.—Frank L., 28, had syphilis six years ago; he gives a Wassermann reaction with both serum and cerebro-spinal fluid. The latter shows lymphocytosis and gives the precipitation tests. He has the mental symptoms and tremors of general paralysis. His pupils are irregular, but react. His gait is normal; his knee-jerks are present, there is no ankle clonus, but the plantar reflexes are extensor.

Case 21.—Thomas B., 39. He gives no history of syphilis, but the Wassermann reaction is positive, and the cerebro-spinal fluid responds to the precipitation tests. He has the mental symptoms, slurred speech, and tremors of general paralysis. There is marked intention tremor in the right arm. The elbow jerks are absent, the pupils are unequal and irregular, but react. There is slight ptosis. His gait is unsteady and inco-ordinate; his knee-jerks are exaggerated, ankle clonus is present and the plantar response is extensor.

Case 22.—Here is a case recorded by Poynton,[81] in which general paralysis and primary lateral sclerosis are combined in a child, the subject of congenital disease. A girl aged 11, with Hutchinson's teeth and patches of choroiditis. She has degenerated mentally, morally, and physically for two years, becoming dirty and silly. Her tongue is tremulous. Her pupils are unequal and fixed. She is unable to walk on account of spastic paraplegia, which is more marked in the left leg than the right. The upper extremities are normal; but the deep reflexes in the legs are increased, and Babinski's sign is present in the left foot.

c. *Primary lateral Sclerosis combined with Primary optic atrophy.*—This will usually be diagnosed as disseminated sclerosis. But instances occur in which there is primary optic atrophy with spastic paraplegia, or Babinski's sign, and no other sign

of dissemination in patients past the usual age for disseminated sclerosis. These may well be really a combination of two of the rarer forms of parasyphilis and not a haphazard insular degeneration at all. Both the cases below in illustration of this amalgamation came under observation before Wassermann's reaction had assumed importance.

Case 23.—Henry C., 69, had syphilis forty years previously. His pupils were normal; there was ptosis and ophthalmoplegia of the external rectus both on the right side. His vision was blurred, and he proved to have double primary optic atrophy. Romberg's sign was present, his left knee jerk was exaggerated, the right one diminished, and Babinski's sign was present.

Case 24.—Agnes N., 41, a married woman giving a history of "vaginal trouble" some years previously. She had double primary optic atrophy. The pupils were normal, there was no nystagmus. The knee-jerks were exaggerated, ankle clonus was present, and the plantar reflexes were extensor.

d. *Primary Lateral Sclerosis alone.*—Some cases of primary lateral sclerosis give a history of syphilis, but no reliance can be placed on statistics in this condition unless Wassermann's test has been applied or a post-mortem examination made, for so many cases diagnosed as primary lateral sclerosis are found post-mortem to be secondary to cerebral tumour or other unsuspected cause. Probably every instance of uncomplicated primary sclerosis of both lateral columns is parasyphilitic, but such examples are not common.

Case 25.—Alfred D., 64, had syphilis forty years ago, and gives a positive Wassermann. Disease began a year ago with weakness of the legs. Six weeks ago paralysis of the right external rectus showed itself. His pupils are normal. His gait is spastic; Achillis jerks brisk, and Babinski's sign is present.

Case 26.—Here is a case of early primary lateral sclerosis with Argyll-Robertson pupils. James T., 43, had syphilis forty-five years ago, and now gives a positive Wassermann. His pupils are unequal, irregular, eccentric, and of the Argyll-Robertson type. There is slight ptosis. The ankle jerks are exaggerated

with a tendency to clonus. The plantar response is flexor in the right foot and unobtainable in the left. He cannot stand on one leg with the eyes shut. There is spasticity of the ankle joints with a tendency to stand always on tip-toe.

Case 27.—Winifred E., 29. A married woman who has had one miscarriage, and gives a positive Wassermann reaction. The elbow, knee, and ankle-jerks are all exaggerated, and Babinski's sign is present. Her eyes are normal, and there is no nystagmus nor any other sign of insular sclerosis.

Case 28.—Edward C., 38. No history of syphilis, but gives a positive Wassermann. His pupils are normal. The legs are spastic, knee-jerks exaggerated, ankle clonus present, plantar reflexes extensor.

Case 29.—James H., 54, had syphilis twenty-five years ago. Wassermann reaction positive. Pupils are normal. There is slight tremor of the tongue. Romberg's sign is present. His knee-jerks are increased, ankle clonus is present, and the plantar reflexes are extensor.

Case 30.—Here is an interesting case recorded by Grainger Stewart[82] as "chronic syphilitic meningitis with compression of the cord," but which I think is more probably an example of parasyphilis of the variety under consideration, for the pupils do not react to light, though they do to accommodation. There is also some sensory disturbance which may be the first sign of the supervention of tabes. A man, aged 64, had syphilis 46 years ago. Ten years ago he fell unconscious in the street, the unconsciousness lasting fifteen minutes. There was no paralysis or other after effect from this incident, and it is doubtful whether it has any connection with the ultimate condition. Five years later his legs became stiff, and he developed into an example of spastic paraplegia. The knee and ankle-jerks are exaggerated, ankle clonus is present, and the plantar response is extensor. The pupils are of the Argyll-Robertson type. Motor weakness is slight. The abdominal and epigastric reflexes are absent, and there is slight diminution of all forms of sensibility below the fourth dorsal segment. Nothing abnormal is found in the spine.

PROGRESSIVE MUSCULAR ATROPHY AS A PARASYPHILITIC MANIFESTATION EITHER COMBINED OR ALONE.

a. *Progressive Muscular Atrophy combined with Tabes.*—Quite early in this paper I referred to Dejerine's[83] estimate that 20 per cent. of tabetics exhibit some form of muscular atrophy, and to the fact that progressive muscular atrophy is becoming more and more frequently recognised in France as a parasyphilitic manifestation. Still, as a rule, the muscular atrophy is looked upon as one of the less frequent symptoms of tabes, or else it is saddled on to one or other of the etiological pack horses, usually "hard work." Kinnear Wilson,[84] however, in recording the following case, says that he considers "Tabetic muscular atrophy is neither a symptom nor a complication of tabes, but an associated condition of syphilitic origin."

Case 31.—A man, aged 47, had syphilis twenty-two years previously. He had been troubled with lightning pains for ten years, girdle pain for nine years, and wasting of the small muscles of the hands (starting in the right) for seven years. He had gastric crises, and experienced abnormal fatigue in the legs. His pupils were of the Argyll-Robertson type; the deep reflexes absent. There was extreme wasting of the thenar and hypothenar muscles, with no reaction to faradism or galvanism. The interossei were less wasted; there was no marked wasting in the arms, though they were weak. In the legs all the muscles, particularly the anterior tibial groups were small and weak. Sensation of pain was impaired in the ulnar distributions, and almost lost in the legs. There was loss of tactile sensibility over the trunk and legs. His right ankle was an incipient Charcot joint. The pathological report of this case records that there is great reduction in the cells of the anterior horns, the change being atrophic or sclerotic. What cells remain are mostly shrunken. The side where the muscular atrophy was most marked (*i.e.*, the right) shows the greater changes in the cord.

Case 32.—This is an instance of early progressive muscular atrophy in a tabetic patient. George S., 44, gives no history

of syphilis, but the Wassermann reaction is positive. His pupils are irregular and of the Argyll-Robertson type. His Achillis-jerks are absent. He has perforating ulcers, rectal crises, and anæsthesia of the bladder (he does not know when it is full). The tongue is tremulous. And there is marked weakness and wasting of the intrinsic thumb muscles of both hands.

Case 33.—Here is a case where the lower neuron degeneration is affecting the bulbar nuclei. Charles H., 36. No history of syphilis, but he gives a positive Wassermann. He has Argyll-Robertson pupils. The ankle-jerks are absent, and Romberg's sign is manifest. He has difficulty in swallowing owing to paralysis of the muscles of the soft palate. (No history of diphtheria could be elicited; the patient denied having a sore throat.)

b. *Progressive Muscular Atrophy combined with General Paralysis.*—I have met with well-marked cases of this combination. As a rule, when muscular atrophy occurs with general paralysis of the insane, it is considered as an unusually marked example of the weakness which comes on in the ordinary course of that disease.

Case 34.—Henry P., 34, had syphilis eighteen years ago, and gives a positive Wassermann and the cerebro-spinal precipitation tests. He shows the mental symptoms, tremors, and slurred speech of general paralysis. There is slight ptosis, and the pupils are unequal, irregular, and of the Argyll-Robertson type. His gait is unsteady, and his knee-jerks are absent. There is extreme wasting of all muscles, most marked in the interossei, of the right hand and the muscles of the same shoulder. All the muscles, except the interossei and lumbricales of the right hand, react to faradism and galvanism, though both are diminished, especially in the right arm and shoulder, and particularly in the right infra-spinatus. The interossei of the left hand are also affected.

Case 35.—James Q., 50, admits syphilis, but does not know date. Wassermann reaction positive, cerebro-spinal fluid gives precipitation tests and shows lymphocytosis. There are tremors

of the tongue and lips. He is very grandiose and happy. He has slight right-sided ptosis, his pupils are unequal, irregular, and fixed. His knee-jerks and all other deep reflexes are lost, as also are the cremasteric and abdominal. The interossei of both hands are wasted, and there is some motor impairment of the right arm.

c. I have not encountered a satisfactory instance of the combination of progressive muscular atrophy with primary optic atrophy.

d. *Progressive Muscular Atrophy combined with Primary Lateral Sclerosis.* Here we have the condition known as amyotrophic lateral sclerosis, or, when the degeneration extends to, or occurs at, the upper end of the cord and medulla, progressive bulbar paralysis. If the symptom-complex comprising progressive muscular atrophy, amyotrophic lateral sclerosis and progressive bulbar paralysis be considered as one primary degeneration of the motor neurons, both upper and lower, we see how cases may vary according to what point in the group is first attacked, and to what extent the disease spreads thence throughout the group. It may begin in the anterior cornual cells and confine itself to that system, giving simple progressive muscular atrophy, or it may extend to the pyramidal tracts, giving amyotrophic lateral sclerosis; and later the bulbar nuclei and the hypoglossal nerve may be involved, thus adding bulbar paralysis, which, fortunately for the victim, as a rule rapidly carries him off. But the trouble sometimes starts with bulbar paralysis, and may show evidence before the patient dies of downward spread.

In 1850 Aran[85] first described progressive muscular atrophy starting in the intrinsic muscles of the hands. In 1859 Duménil[86] noted progressive bulbar paralysis with atrophy of the hypoglossal nerve, and regarded it as a form of progressive muscular atrophy. In 1872 Charcot[87] described amyotrophic lateral sclerosis, considering it also as an extension of progressive muscular atrophy.

With regard to the etiology of these varieties of motor tract degeneration, Dana[88] and Merle[89] urge syphilis as the essential

cause, whilst Wilson[90] considers lead poisoning the commonest factor, and thinks that in some cases syphilis can be excluded. Gowers[91] thinks there is some evidence of heredity. The points in favour of parasyphilis are the age incidence, in the parasyphilitic decade—the fourth, the greater frequency with which men are attacked than women, and the fact that Argyll-Robertson pupils are sometimes associated. Cases of bulbar paralysis occurring in the seventh or eighth decade may often be due to senile changes.

Case 36.—William V., 52, does not admit syphilis, but has a positive Wassermann. He gives a four years' history of general progressive weakness and loss of control of the legs: "Feet catch in everything." He complained that his feet and legs were numb, and of cramps in the feet, legs, thighs, and hands. There is no absolute paralysis, but general paresis. His tongue is not tremulous, but his speech is altered, and his voice falsetto. The pupils are normal and react, if anything, better to light than to accommodation. Sensation is unimpaired. His knee-jerks are present, the left ankle-jerk is increased, the right absent. The plantar reflex is extensor in the left foot, unobtained in the right. The cremasteric and abdominal reflexes are present. There is well-marked wasting of both thenar eminences, and he cannot oppose the thumb and little finger. There is some wasting noticeable in the left leg.

Case 37.—William B., 50. He had syphilis thirty years ago, and has been troubled for nineteen months with weakness of the left hand, for six months with the same in the right hand, and lately with weakness of the left leg. In the left hand there is marked wasting and loss of power in all the intrinsic muscles; also in the forearm, both flexors and extensors; there is very little movement in the elbow joint, and practically none in the shoulder. The atrophy in the right hand is much less marked. Reaction of degeneration is present in all the affected muscles, and is complete in the interossei of the left hand. The legs are weak, but show no obvious wasting. His knee-jerks are exaggerated, both ankle clonus and Babinski's sign are present.

e. *Progressive Muscular Atrophy alone.*—As a parasyphilitic manifestation, progressive muscular atrophy is certainly most often met with in combination; but the fact that the knee-jerks may be lost in this condition quite early has probably led to a considerable proportion of these cases being diagnosed as locomotor ataxia with marked muscular symptoms.

Case 38 shows this liability, as his tendon reflexes are absent, although the muscular atrophy is confined, clinically, to the upper extremities. Probably some change is present in the cord lower down, some shrinkage of anterior cornual cells. Thomas F., 53, had syphilis thirty-five years ago. Wassermann reaction positive. His pupils are irregular, but react, though only very faintly, to light. The deep reflexes are lost. There is no ataxy. Wasting is marked in both upper limbs in the interossei, thenar and hypothenar eminences, the flexors and extensors of the forearms, the muscles of the upper arms and shoulders. The legs are unaffected.

Case 39.—Joseph A., 50, had syphilis thirty years ago. His pupils are unequal, but react. The right knee-jerk is absent, the left present. The thenar and hypothenar eminences are wasted, especially of the left hand, also the interossei, the flexors of arms and upper arms, the supra and infra-spinati, and the lower part of the pectoralis major. The right thigh is also much wasted.

W. Harris,[92] writing of syphilis of the spinal cord, mentions cases of chronic or sub-acute poliomyelitis due to syphilis. These, he says, are "Generally mistaken for progressive muscular atrophy." He refers to three cases, recorded by Max Nonne,[93] in which the upper extremities only were affected; and three by J. Hoffmann.[94] But Hoffmann, at all events, is one of those who recognise progressive muscular atrophy as a parasyphilitic manifestation. Harris also describes the following case of his own :—

Case 40.—A woman (age not given) with a definitely positive Wassermann, had pain in the left upper arm as the first symptom of disease, followed by paralysis of the deltoid. A year

later her right wrist dropped, and the right shoulder became paralysed. A year later there was almost complete paralysis of both deltoids, biceps, spinati, and extensors of the forearm, with reaction of degeneration. There was no sensory disturbance; all the deep reflexes, except the jaw-jerk, were absent, and the pupils reacted sluggishly to light. The Wassermann reaction and the slowly progressive nature of the complaint suggest a diagnosis of parasyphilitic progressive muscular atrophy. I cannot see why a special class should be devised for "Cases generally mistaken for progressive muscular atrophy" simply on the ground that no other cause but syphilis can be discovered.

Rose et Rendu,[95] in the interesting article already referred to, describe two cases in which no cause but syphilis could be suggested. In the first they hold that, in spite of some exaggeration of reflexes and a tendency to ankle clonus, it is a case of pure progressive muscular atrophy. But these signs should rather be held to point to some involvement of the pyramidal tracts, and as suggesting the development of the case into amyotrophic lateral sclerosis.

Case 41.—A man, aged 42, who, except for syphilis at eighteen, never had any illness. He was treated for two years, and married at 26. His wife's first pregnancy resulted in miscarriage. Some difficulty in using his hands appeared six years ago. This paresis progressed, and two years later spread to the arms successively. He has no pain, but some alteration of sensibility to cold. No urinary trouble, but loss of sexual power for the last year. The thenar and hypothenar muscles of both hands are very markedly atrophied, also the first interossei. Atrophy not so marked in the forearms, affecting the extensors and supinators only in the right, but the flexors, too, in the left. The elbow-jerks are exaggerated. The legs are unaffected, except that the reflexes are somewhat exaggerated, with a tendency to ankle clonus on both sides. Plantar response flexor. The right cremasteric reflex is absent. The pupils are of the Argyll-Robertson type, and there is an intense cerebro-spinal lymphocytosis. The second case differs from the above in that all the

deep reflexes are absent, and is a purer case of progressive muscular atrophy alone.

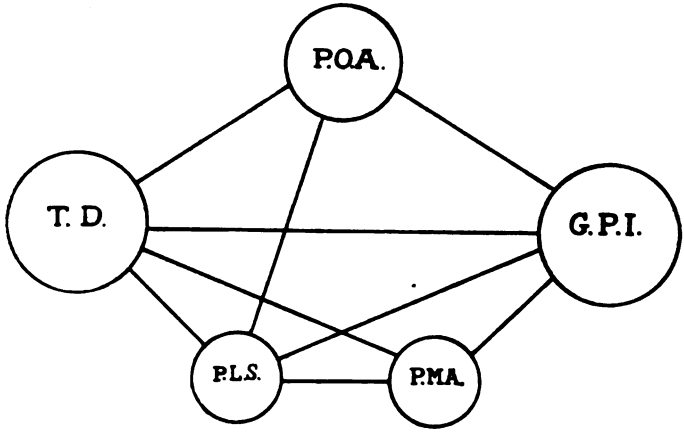
Case 42.—A man, aged 34, had syphilis at twenty-one. Three years later he had lightning pains. Six years after the infection the right hand began to get weak and waste, the thumb being first affected. Four years later the left hand followed suit, and the forearms and arms became involved. There was progressive loss of power before the atrophy appeared. The trophic changes are confined to the upper limbs, being most marked in the thumb muscles of the right hand, and the interossei of the left. The right arm is almost useless, the left not so advanced. The deep reflexes are absent in all four limbs; the superficial ones remaining. There are no abnormalities of sensation; no ataxia; Romberg's sign is not present, and there are no sphincter troubles. The pupils are unequal, and are described as "incomplete Argyll-Robertson." There is no optic atrophy. The cerebro-spinal fluid shows lymphocytosis. No benefit has accrued from anti-syphilitic treatment.

Cases where Manifestations of Degeneration in more than Two Nerve Tracts occur in One Individual.

In studying these parasyphilitic nervous diseases, one is constantly aware of a suspicion that more tracts are involved than at first sight is manifest. Some abnormality of sensation, some failure of vision, some change in moral or intellectual characteristics, some lately developed jerkiness of movement, some loss of power in the hands, or an alteration in the voice may attract the attention, and suggest that the decay is not confined to one or even two communities of neurons.

For this reason it is often very difficult to classify a case, and having classified it, one is frequently tempted to remove it to some other group. Parasyphilis of the nervous system must be considered, in fact, as one polymorphic disease with many different manifestations. The clinical picture is almost kaleidoscopic in its infinite variety. Taking only the five manifestations dealt with in this paper, the diagrammatic representation of

their combinations in pairs is well nigh confusing, and would be entirely so were the multiple conditions, now to be considered, indicated.



Yet, in a full consideration of parasyphilis of the nervous system room must probably be found for certain neurasthenias, dystrophies, non-developments, and epilepsies, with their associations.

Of multiple manifestations one would naturally expect the greater number to be based on tabo-paresis; and such is the case. I shall not describe more than one instance of each variety I have met with.

Case 43.—*Tabo-paresis with primary optic atrophy.*—John S., 50, denies syphilis, but has a scar on the glans penis. He gives a positive Wassermann both with blood and cerebro-spinal fluid; the latter further shows lymphocytosis and gives the precipitation tests. He has the mental symptoms of general paralysis, with slurred speech and tremors of the tongue, lips, and hands. His gait is unsteady, and his knee-jerks are absent. His Argyll-Robertson pupils are irregular in outline, and there is primary optic atrophy in both eyes, especially in the left.

Case 44.—*Tabo-paresis with primary lateral sclerosis.*—George C., 33. He had syphilis thirteen years ago, and gives a positive Wassermann reaction. His disorder began with a "social ataxia,"

and he now shows the mental symptoms of general paralysis; his speech is defective, his tongue tremulous, his pupils irregular and of the Argyll-Robertson type. He does not exhibit Romberg's sign, but both his knee- and ankle-jerks are absent. Finally, the plantar reflex is extensor in both feet.

Case 45. — *Tabo-paresis with progressive muscular atrophy.* George E., 45. No history of syphilis was obtained, but he gave a positive Wassermann, and his cerebro-spinal fluid gave precipitation tests. He has the mental signs of general paralysis of the insane with tremulous tongue and lips, and slurred speech. In addition, there is ptosis on the left side; his pupils are unequal, irregular, and of the Argyll-Robertson type. His gait is ataxic, and his knee-jerks are absent. There is marked progressive trophic wasting of the right thigh and calf muscles.

Case 46.—*General paralysis of the insane with amyotrophic lateral sclerosis.* — Sophie P., 43. No history of syphilis, but her cerebro-spinal fluid gave both the precipitation tests and the Wassermann reaction. She has the mental signs, tremors, and defective speech of general paralysis. Her pupils react to light and accommodation, though very slightly to the former. Her legs are inco-ordinate. The knee-jerks are present, and there is ankle clonus and extensor plantar reflex on the left side. There is great progressive muscular wasting of both arms and legs.

Case 47.—*Tabo-paresis with primary optic atrophy and primary lateral sclerosis.* — Here four out of the five nerve tracts show evidence of decay. This man had a very severe attack of syphilis, with troublesome sores all over the body. John W., 46, had syphilis twenty years ago; his wife has had numerous miscarriages. He gives a double Wassermann reaction, and his cerebro-spinal fluid also exhibits lymphocytosis and the precipitation tests. He is demented, his tongue and fingers are tremulous. He is inco-ordinate, though his gait is fair. The tendon reflexes in both legs and arms are absent. Babinski's sign is present. He has double primary optic atrophy, and his pupils are unequal, irregular, and of the Argyll-Robertson type.

Tooth and Howell[96] publish an exhaustive account of a very severe case of amyotrophic tabes from the point of view that the tabes and muscular atrophy are due to two distinct causes, though the separate and distinct cause of the latter is not clearly indicated. I bring my series of cases to an end with an abstract of this one, because it appears evident to my mind that it is an instance of parasyphilis of the nervous system exhibiting signs of degeneration of four separate nerve tracts. One of these, however, the pyramidal, was not indicated during life, or was overshadowed by the intensity of the other troubles.

Case 48.—*Tabes with primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy.*—A man, aged 35, had syphilis sixteen years before. For sixteen months there has been progressive difficulty in walking and numbness of the feet. He is mentally bright, but physically troubled with lightning pains, girdle pain, gastric crises, difficult micturition, right-sided ptosis and failing vision, especially in the left eye. Articulation lisping. Tongue tremulous, left half atrophied. Unequal Argyll-Robertson pupils. Discs distinctly suggestive of primary optic atrophy. Almost complete ophthalmoplegia of both eyes. Some paresis of lips. Hearing defective in both ears. Left masseter muscle wasted. Muscular power in arms and trunk fair, legs very weak and atrophied, the right more so than the left. Can only just stand with support, and attempts to walk only result in disordered ataxic movements. Muscle pain sense lost, joint sense defective. Analgesia over greater part of face and body, most marked in ulnar distribution and outer sides of legs. Marked inco-ordination in the arms as well as legs. All the above symptoms were progressive, and the right side of tongue, the right masseter muscle and both temporal muscles became involved in the atrophy, and deglutition became increasingly difficult.

Pathological examination showed that the third, fifth, sixth, and twelfth cranial nerves were wasted. The pia arachnoid was a little thicker over the posterior columns in the cervical and upper dorsal region. Both posterior and anterior nerve roots

were small. The anterior cornual cells were diminished in numbers, and those remaining were shrunken. The degeneration of posterior columns was typical of tabes, and there were slight degenerative changes in the lateral columns. The affected muscles showed changes identical with those of progressive muscular atrophy, though the eye muscles were very little altered.

Summary.

There is no doubt that in the vast majority of cases of tabes and general paralysis of the insane, syphilis is the essential cause. They are parasyphilitic nervous diseases.

There is evidence that at all events some cases of primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy are parasyphilitic, too. A history of syphilis or a positive Wassermann reaction, and in some instances the post-mortem examinations, point to this being so.

In tabes and general paralysis there is degeneration of certain neuronic tracts or groups; and degeneration of precisely the same character in other neuronic groups is found in primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy.

Combinations of all three with tabes or general paralysis of the insane, or with both, or amongst themselves, are so frequent that it is difficult to withstand the conclusion that their essential etiological factor is the same.

When one definite neuronic degeneration of unknown cause is found in conjunction with another similar definite neuronic degeneration known to be due to syphilis, it would seem rational to suppose that the first is also due to the same cause, more especially since it is conceded that syphilis has a great affinity for the nervous system.

If it be allowed that the neuronic degeneration of primary optic atrophy, primary lateral sclerosis, or progressive muscular atrophy when combined with that of tabes or general paralysis is due to syphilis, it cannot be denied that such degeneration, occurring uncombined, may still be parasyphilitic.

The Argyll-Robertson pupil is recognised as a "symptom" of tabes and general paralysis; in other words, it is a parasyphilitic manifestation. It is found also with primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy.

Denial of syphilis by a patient cannot be regarded as proof that he has not had the disorder. For few men care to disclose it. Often in tabes it is only after close cross-examination and promises of secrecy that the admission is obtained. In diseases not generally recognised as syphilitic or parasyphilitic such cross-examination is seldom indulged in. If the question of antecedent syphilis were carefully investigated in every case of primary optic atrophy, primary lateral sclerosis, and progressive muscular atrophy, or, better still, if every case were subjected to Wassermann's test as well, I believe that an accumulation of evidence, entirely analogous to that obtained in determining the etiology of tabes and general paralysis, would soon be at hand, and that these three diseases at present wandering in the Wilderness of Undiscovered Causes, would take their rightful place amongst the rarer manifestations of parasyphilis of the nervous system.

BIBLIOGRAPHY.

1. Fournier, Les affections parasyphilitiques, Paris, 1894, page 3.
2. Ferrier, Lumleian Lectures, Lancet, 1905, vol. I., page 951.
3. Byrom Bramwell, B.M.J., March, 1908, vol. I., page 675.
4. Mott, Syst. of Syph. (Power and Murphy), vol. IV., page 221.
5. Mott, Loc. cit. (4).
6. Osler, Princ. and Pract. of Med. (7th edit.), page 889.
7. Norris and Oliver, Syst. of Dis. of the Eye, Philadelphia, 1897, vol. IV., page 634.
8. Dejerine, Rev. de Méd., 1889, IX., page 81.
9. Holt, Dis. of Inf. and Childh., 6th edit., 1911, page 796.
10. Erb, Etiol. of Tabes, New Syd. Soc., vol. CLXX., page 79.
11. Still, Common Disorders of Childh., 1st edit., 1909, page 516.
12. Mott, Arch. of Neurol., vol. I., 1899.
13. Still, Loc. cit. (11), page 604.
14. Mott, Syst. of Syph., vol. IV., page 186.
15. Fournier, Loc. cit. (1).
16. Pousard, Thèse de Paris, 1893.
17. Lannois et G. Lévy, Echo méd. de Lyon, Avril, 1900.

18. Léri, Sem. méd., 1903, page 266.
19. Camus, Thèse de Lyon, 1905.
20. Lannois et Poret, Rev. de Méd., 1906, XXVI., page 567.
21. van Gehuchten, Névraxe, 1907, IX., page 329.
22. Merle, Rev. neurol., 1909, XVII., page 877.
23. Kinneir Wilson, Proc. Roy. Soc. Med., 1909, Neurol., page 20.
24. Rose et Rendu, Sem. méd., 1910, XXX., page 217.
25. Raymond, Sem. méd., 1893, XIII., page 56.
1908, XXXI., page 289.
26. Noguchi, Journ. Exper. Med., 1909, vol. XI., No. 1.
27. Pfeiffer, Centr. f. Bakt., etc., vol. XIX., page 385, and Deutsch. Med. Wochenschr., 1896, Nos. 7 and 8.
28. Bordet et Gengou, Ann. de l'Inst. Pasteur, Paris, 1901, XV., page 289.
29. Wassermann, Deutsch. Med. Wochenschr., 1906, XXXII., 745; Berl. klin. Wochenschr., 1907, Nos. 50 and 51; Wien. klin. Wochenschr., 1908, XXI., 388; and (F. Plant) Die Wassermannsche Serodiagnostik der Syph. in ihrer Anwendung auf die Psychiatrie, 1909.
30. Plant, Serodiagnostik der Syph, Zentralbl. f. Nerv. u. Psych., 1908, XXXI., page 289.
31. Lavaditi et Yamanouchi, Soc. de Biol., Comptes rendus, t. LXII., 1907, page 740; t. LXIV., 1908, page 349; Bull. Soc. de Path. Exot., t. 1., No. 1.
32. Lavaditi et Marie, Ann. de l'Inst. Pasteur, Paris, 1907, XXI., 138.
33. Porges, Münch. Med. Wochenschr., 1907, No. 51.
34. Landsteiner, R. Muller, und Potzl, Wien. klin. Wochenschr., 1907, No. 50.
35. Hecht, Wien. klin. Wochenschr., 1908, page 1742.
36. Fleming, Proc. Roy. Soc. Med., 1909, Clin., page 220.
37. Esmarck und Jessen, Syph. u. Geistesstörung, Allg. Zeitschrift. f. Psych., 1857, Bd. CXLV.
38. Eisenmann, Die Bewegangsataxie, Wien., 1863.
39. Topinard, De l'ataxie locomotrice, Paris, 1864.
40. Fournier, De l'ataxie locom. d'origine syph., Paris, 1882.
41. Gowers, Syph. and Locom. Atax., Lancet, 1881, vol. I., page 94.
42. Erb, Zeimssen's Cycl. of Pract. of Med., London, vol. XIII. Handbuch der Krankheitendes Rückenarks, eds. 1 and 2.
43. Erb, Centralbl. f. d. med. Wissenschaften, 1881, Nos. 11 and 12. Berl. klin. Wochenschr., 1883, No. 32; ibid., 1891, No. 29; ibid., 1896, ibid., 1904; Syph. und Tabes, pages 9, 40, 62, and 86; Neurol. Centralbl., 1892, No. 6.
44. v. Mott, Syst. of Syph., vol. IV., page 218.
45. Minor, Arch. de Neurol., XVII., 1889.
46. Savage, Tuke's Dict. of Psychol. Med., 1892.
47. Mott, Arch. de Neurol., vol. II., 1903.
48. Jumon, Etude sur les syphilis ignorées, Paris, 1880.
49. Fleiner, Ueber syph. occulta, Deutsch. Arch. f. klin. Med., Bd. 48, 1891; J. E. Lane, Syphilis d'emblée, Lancet, June 15th, 1912.
50. Ferrier, Loc. cit. (2).
51. Moebius, Ueber die Tabes.
52. Bernhardt, Zur Aetiol der Tabes, Neurol. Centralbl., 1890, IX., page 710.

53. Oppenheim, Neue Beiträge z. Pathol. der Tabes. Arch. f. Psychiat., XX., page 131.
54. Rumpf, Ein Beiträge z. Tabes-syphilisfrage, Berl. klin. Wochenschr., 1881, XVIII., page 521.
55. Norris and Oliver, Loc. cit. (7).
56. Benedikt, Wien. Medizin Presse, 1881, XXII., pages 1, 40, 101, 135.
57. Head, Proc. Roy. Soc. Med., 1910, Neurol., page 55.
58. Mott, Proc. Roy. Soc. Med., 1910, Neurol., page 41.
59. Fournier, Loc. cit. (1).
60. Mott, Loc. cit. (58).
61. Krafft-Ebing, Discussion on G.P.I., Internat. Cong., Moscow. Ref. Neurol. Centralbl., 1897, XVI., page 871.
62. Moore, Lancet, June 15th, 1912.
63. Lambkin, Syst. of Syph., vol. II., page 339 et seq.
64. Erb, Loc. cit. (42).
65. Mott, Loc. cit. (14), pages 246-7.
66. Morell-Lavellée, Gaz. des hôpit., 1892, LXV., page 303.
67. Mott, Loc. cit. (14), page 419.
68. Marina, Annali di Neurol., 1901, vol. XIX., page 209.
69. Fournier, Loc. cit. (1).
70. Erb, Loc. cit. (10).
71. v. Mott, Loc. cit. (14), page 234.
72. Rumpf, Die Syph. Erkrankangen des Nervensystems, Wiesbaden. 1887.
73. Max Nonne, Syph. des Nervensystems, 1909.
74. Head, Loc. cit. (57), page 52.
75. Norris and Oliver, Loc. cit. (7).
76. Osler, Loc. cit. (6).
77. Turtle, Proc. Roy. Soc. Med., 1909, Neurol., page 104.
78. Mott, Loc. cit. (14), page 422.
79. Head, Loc. cit. (57), page 33.
80. Osler, Loc. cit. (6), page 910.
81. Poynton, Proc. Roy. Soc. Med., 1909, Clin., page 11.
82. Grainger Stewart, Proc. Roy. Soc. Med., 1910, Neurol., page 33.
83. Dejerine, Loc. cit. (8).
84. Kinneir Wilson, Loc. cit. (23).
85. Aran, Arch. gén. de méd., Paris, 1850, XXIV., page 172.
86. Duménil, Gaz. hebd. de méd., Paris, 1859, VI., page 390.
87. Charcot, Arch. de Physiol., 1871-2, page 509 (reported by Gombault).
88. Dana, Journ. Nerv. and Ment. Dis., N.Y., 1906, XXXIII., p. 81.
89. Merle, Loc. cit. (22).
90. Kinneir Wilson, Rev. Neurol. and Psychiat., Edin., 1907, page 441.
91. Gowers, Dis. of Nerv. Syst., 1899, I., pages 489-533.
92. W. Harris, Syst. of Med. (Allbutt and Rolleston), 1910, VII., page 730.
93. Max Nonne, Syph. und Nervensystem, 2nd edit.
94. Hoffmann, Neurol. Centralbl., Leipzig, 1909, XXVIII., page 1074.
95. Rose et Rendu, Loc. cit. (24).
96. Tooth and Howell, Proc. Roy. Soc. Med., 1912, Neurol., page 81.

LIST OF BOOKS BY GUY'S MEN IN THE WILLS LIBRARY, GUY'S HOSPITAL.

WITH AUTOGRAPH INSCRIPTIONS, NOTES, ETC.

COMPILED BY

WILLIAM WALE, F.R.Hist.S.,

Wills Librarian; Member of the Bibliographical Society.

THIS is a list of books in the Wills Library written by Guy's men, which have been recently collected together and placed in two special bookcases near the entrance of the library. By reference to the Surgeon-General of America's Index Catalogue it will be seen that there are many other books by Guy's men not included in this list, and it is hoped that there may be friends who, having copies of some of these missing volumes, will be willing to fill up the gaps by presenting them to the library of their medical Alma Mater, and thus help to make the monument of nobler proportions. The term "Guy's men" has been used in its widest sense, and includes those who have at any time been connected with the Hospital, either as students or in an official capacity, such as lecturers, demonstrators, etc.

A. I. signifies autograph inscription.

"Many discoveries in medicine will for ever be associated with the name of Guy's."—Sir Samuel Wilks, Guy's Hosp. Rep., 1877, xxii., 259.

The remarkable original work done at Guy's Hospital is probably a subject for the pathologist and the clinician, but it may be worth while in passing to glance at these discoveries from the bibliographic angle in their historic sequence. . . . It was here that Sir Astley Cooper did some of the earliest work in experimental surgery after the time of

John Hunter (1836): here Addison, Golding Bird, and Gull first tried the effects of static electricity in disease (1837); here Pavy did his first experimental work in diabetes (1853); here John Cooper Forster performed the first gastrostomies in England (1858—9); and here occurred what might be regarded as an event in the history of obstetrics, the first findings of albuminous urine in cases of puerperal eclampsia by Lever in 1843, the most important outcome of Bright's teaching in albuminuria. But the special achievement of the men of Guy's was in the department of original descriptions of disease.—Dr. Fielding H. Garrison, Editor of *Index Medicus;* "Richard Bright's Travels in Lower Hungary: a Physician's Holiday."—The Johns Hopkins Hospital Bulletin, June, 1912.

Though Guy's Hospital is by no means the oldest of the metropolitan hospitals (it was founded in 1725), it has attained a world-wide reputation, and during an existence of little more than a century and a half has had connected with it many of the foremost men of science whose names are imperishably written in the annals of medicine.—*British Medical Journal,* November 26th, 1892.

In so large a School as Guy's, it is evident that it must have sent out into the world a great many students who afterwards rose to celebrity and held offices and appointments elsewhere. It would be a pleasant task, therefore, to look around and select all those men of eminence in every part of the world who might at one time have been styled Guy's men; but it would be beyond the scope of this work to attempt, even were it possible, to go on such a voyage of discovery. We should have liked, however, to have enumerated all those who have at any time been connected with our own School, even for a short period; but unfortunately our records do not allow us to do even this, and therefore we must be content to mention the names of one or two men known to fame. —Wilks' and Bettany's Biographical History of Guy's Hospital.

THOMAS ADDISON (1793—1860),

M.D. Edin., F.R.C.P. Lond., 1838,

Assistant Physician to Guy's, 1824; Physician, 1837; President of the Royal Medical and Chirurgical Society, 1849.

For many years he was the leading light of Guy's, so that every Guy's man during the thirty or forty years of his teaching was a disciple of Addison, holding his name in the greatest reverence and regarding his authority as the best guide in the practice of the profession.—Wilks and Bettany's History of Guy's Hospital.

In his hospital practice he soon became distinguished for his remarkable zeal in the investigation of disease both by observation of cases during life and by post-mortem examinations. He thus acquired a brilliant reputation as a clinical teacher, and contributed perhaps more than any of his colleagues to the fame which Guy's Hospital attained as a School of Medicine during his connection with it.—Dr. J. F. Payne, Dict. Nat. Biog.

With John Morgan. An essay on the operation of poisonous agents upon the living body. Illus., 8vo., Lond., 1829.

The first serious investigation in England into the phenomena of general poisoning.—Wilks and Bettany.

Observations on the disorders of females connected with uterine irritation. 8vo., Lond., 1830.

On the constitutional and local effects of disease of the suprarenal capsules. Col. plates, fol., Lond., 1855.

A. I.—*The Physical Society of Guy's Hospital, from the Author, Augt.* 16/55.

Die Erkrankungen der Nebennieren und ihre Folgen (1855). Zum ersten Male in deutscher Übersetzung herausgegeben und eingeleitet von Erich Ebstein. 8vo., Leipzig, 1912.

A. I.—*Der Bibliothek des Guy's Hospital in London vom Übersetzer Dr. Erich Ebstein.*

Addison produced in 1855 the work by which he is, and always will be, best known, though less valued by his own pupils and immediate successors than his earlier works. In this, the "Essay on Disease of the Suprarenal Capsules," he announced a discovery of remarkable originality, viz., that these organs, not previously known to be the seat of any definite disease, were, in certain cases, affected in such a way as to produce a fatal malady, with well-marked symptoms, including a remarkable discoloration of the skin, and now known as Addison's disease.— Dr. J. F. Payne, Dict. Nat. Biog.

"Ce médecin est le Docteur Addison, le collaborateur de Bright, le doyen des professeurs du Guy's Hospital à Londres, et depuis longtemps connu parmi nous par les travaux dont il a enrichi la science. Je propose donc d'appeler *Maladie d'Addison* cette singulière cachexie spécialement characterisée par un discoloration ou plutôt par un coloration particulière, par *la teinte bronzée* que prennent les téguments et qui a valu à la maladie la denomination de *bronze disease*, sous laquelle le docteur Addison l'a designée."—Dr. Armand Trousseau.

A collection of the published writings of the late Thomas Addison. Edited, with introductory prefaces to several of the papers, by Dr. Wilks and Dr. Daldy. New Syd. Soc., *Port.*, 8vo., Lond., 1868.

See also Bright and Addison's Elements of the practice of medicine, 1839.

ARTHUR AIKIN (1773—1854).

With Alfred Swaine Taylor. Syllabus of a course of chemical lectures delivered at Guy's Hospital. (Interleaved with notes.) 8vo., Lond., 1839.

RICHARD WILLIAM ALLEN,
M.A., M.D., B.S. Lond.,
Editor of *The Journal of Vaccine Therapy*, 1912.

The opsonic method of treatment. 8vo., Lond., 1907

Vaccine therapy, its theory and practice. 3rd edit., 8vo., Lond., 1910.

See also Guy's Hospital—Gull Students' Reprints.

WILLIAM ALLEN (1770—1843),
F.L.S., 1801; F.R.S., 1807.
Lecturer on Chemistry at Guy's, 1802—1826.

With John Bostock, F.R.S., and Arthur Aikin. Syllabus of a course of chemical lectures delivered at Guy's Hospital. Frontis., 8vo., Lond., 1822. (Some pages missing at end.)

See also Babington and Allen's Syllabus of a course of chemical lectures read at Guy's Hospital, 1802; and 1816 (*with* Marcet).

SAMUEL ASHWELL (1798—1852?),
Lecturer on Midwifery at Guy's, 1834.

A practical treatise on parturition, comprising the attendant circumstances and diseases of the pregnant states. To which are appended two papers, the one containing some remarks on abdominal surgery, the other on transfusion. Presented by Dr. Blundell. Plates, 8vo., Lond., 1828.

A practical treatise on the diseases peculiar to women, illustrated by cases derived from hospital and private practice. 8vo., Lond., 1844.

A. I.—*Library of Guy's Hospital with the best wishes of the Author.*

Lectures on midwifery. MS., 4to.

BENJAMIN GUY BABINGTON (1794—1866),

M.D. Camb., 1830; F.R.C.P., 1831; F.R.S.;

Physician at Guy's, 1840—1858; President of the Pathological Society, 1853—54; Founder and first President of the Epidemiological Society; President of the Royal Medical and Chirurgical Society, 1861.

Dr. Babington was the son of Dr. Wm. Babington, and born in Guy's Hospital, when his father was Resident Apothecary there. He is the sole instance of a son succeeding his father in the Presidency of the Royal Medical and Chirurgical Society.

Translator from the German of J. F. C. Hecker's Epidemics of the Middle Ages. Syd. Soc., 8vo., Lond., 1844.

WILLIAM BABINGTON (1756--1833),

M.D. Aber., 1795; Hon. M.D. Dub., 1831; F.R.S., 1828; Elected F.R.C.P. by special grace, 1827;

Apothecary, 1871, and Physician, 1795—1811, to Guy's; President of the Royal Medical and Chirurgical Society, 1817; One of the Founders and, in 1822, President of the Geological Society.

Lectures on materia medica, delivered at Guy's Hospital, 1796. MS., 4to.

Lectures on therapeutics, delivered at Guy's Hospital, 1796. MS., 4to.

Lectures on chemistry, delivered at Guy's Hospital, 1797. MS.

Has the following written on the title-page: "Martin Tupper's, Guy's Hospital, Southwark, London."

A new system of mineralogy, in the form of catalogue, after the manner of Baron Born's systematic catalogue of the collection of fossils of Mlle. Eléonore de Raab. 4to., Lond., 1799.

Lectures on chemistry. MS., 4to., no date.

Lectures on phosphorus and analysis of mineral waters. MS., 4to., no date.

With William Allen, F.R.S. A syllabus of a course of chemical lectures read at Guy's Hospital. 8vo., Lond., 1802.

Interleaved copy with notes. A. I.—"George Hickman, St. Thomas's and Guy's Hospitals, 1806."

With James Curry. Outlines of a course of lectures on the practice of medicine, as delivered in the Medical School of Guy's Hospital. Frontis., 8vo., Lond., 1802—06.

Interleaved copy with notes. A. I.—"G. Hickman, No. 19, St. Thomas's Street, Boro', 1806."

With Alexander Marcet, F.R.S., and William Allen, F.R.S. A syllabus of a course of chemical lectures read at Guy's Hos-Hospital. Plate, 8vo., Lond., 1816.

Interleaved copy with frontispiece of the Chemical Theatre, Guy's Hospital. It contains the following inscription written by the late Sir Thomas Stevenson: "This appears to be an annotated copy in the handwriting of Dr. Babington or Dr. Marcet. It was shown in 1886 to Mr. Cornelius Hanbury of Plough Court, nephew of Mr. Allen, who said the handwriting was not that of Mr. Allen. In 1888 the Lecture Theatre was much as in 1816.—Thomas Stevenson."

With James Perry. Extracts from lectures on the practice of medicine, delivered at Guy's Hospital between January 1st and June 1st, 1817. MS., 8vo.

Contains printed label with the following: "A.D. 1899.—Presented to the Library of Guy's Hospital, by H. W., a son of Joshua Waddington, F.R.C.S., the writer of these notes."

CHARLES BADER.

Ophthalmic Assistant Surgeon and Lecturer on Diseases of the Eye at Guy's Hospital.

The natural and morbid changes of the human eye, and their treatment. 8vo., Lond., 1868.

Formerly belonged to Mr. John Hilton.

Plates illustrating the natural and morbid changes of the human eye Plates, 8vo., Lond., 1868.

WILLIAM HENRY BAINBRIGGE.

The Droitwich salt springs: their medicinal action and curative properties. 3rd edit., enlarged and revised, 8vo., Droitwich and Lond., 1881.

A. I.—*From a sincere well-wisher of dear old Guy's. The Author, Sep.* 22, 1881.

GEORGE HILARO BARLOW (1806—1866),
M.A., 1832; M.D. Camb., 1841; F.R.C.P., 1842;
Physician to Guy's, 1843—1866; Goulstonian Lecturer, R.C.P., 1844.

A manual of the practice of medicine. 8vo., Lond., 1856.

Ditto. 2nd edit., 1861.

A. I.—*From the Author.*

SAMUEL BAYFIELD.

A treatise on practical cupping: comprising an historical relation of the operation through ancient and modern times, with a copious and minute description of the several methods of performing it. Plates, 8vo., Lond., 1823.

FRANK EVERS BEDDARD,
M.A. Oxon.; F.R.S., 1892;
Formerly Lecturer on Biology at Guy's.

Mammalia. Cambridge Natural History, vol. 10. Illus., 8vo., Lond. and New York, 1902.

THOMAS BELL (1792—1880),
F.R.S., 1828;
Secretary of the Royal Society, 1848—1853; Lecturer on Dentistry and Zoology at Guy's; Professor of Zoology at King's College, 1836—1862; President of the Linnæan Society, 1853—1861; President of the Ray Society, 1844—1859.

The anatomy, physiology, and diseases of the teeth. Plates, 8vo., Lond. and Edin., 1829.

Formerly Mr. John Hilton's copy, with his bookplate.

"Bell had the great merit of applying the general rules of surgery to the art of dentistry."—Sir John Erichsen.

GEORGE THOMAS BETTANY,
M.A., B.Sc.;
Lecturer on Botany, Guy's Hospital.

With W. K. Parker. The morphology of the skull. Illus., 8vo., Lond., 1877.

Joint author of Wilks and Bettany's Biographical History of Guy's Hospital, 1892.

GOLDING BIRD (1814—1854),
M.A., M.D. St. Andrews; F.R.C.P., 1844; F.R.S.;
Lecturer on Natural Philosophy at Guy's, 1836—53; Assistant Physician to Guy's, 1843—1853; Lecturer on Materia Medica, R.C.P., 1847.

When barely twenty-one he (G. B.) went up for examination at Apothecaries' Hall; but the Court of Examiners, in consideration of the reputation he had already attained, declined to examine him, and gave him at once the licence to practise, with the 'honours of the Court,' on January 21st, 1836."—Dr. J. F. Payne, Dict. Nat. Biog.

Urinary deposits, their diagnosis, pathology, and therapeutical indications. 8vo., Lond., 1844.

A. I.—*Library of Guy's Hospital. From the Author,* 1844.

Ditto. 2nd edit., 1846.

Elements of natural philosophy; being an experimental introduction to the study of the physical sciences. 3rd edit., revised and enlarged, illus., 8vo., Lond., 1848.

Ditto. 4th edit., 1854, with Charles Brooke, F.R.S.

Lectures on the influence of researches in organic chemistry on therapeutics, especially in relation to the depuration of the blood. 8vo., Lond., 1848.

Lectures on electricity and galvanism, in their physiological and therapeutical relations, delivered at the Royal College of Physicians. Revised and extended, illus., 8vo., Lond., 1849.

JOHN BIRKETT,
F.R.C.S., 1844;
Jacksonian Prizeman, 1848; Surgeon and Lecturer on Surgery at Guy's.

The diseases of the breast and their treatment (Jacksonian Prize for 1848). Col. plates, 8vo., Lond., 1850.

Translator of von Behr's Handbook of human anatomy, general, special, and topographical. From the original German of Dr. Alfred von Behr, and adapted to the use of the English student by John Birkett. 8vo., Lond., 1846.

JAMES BLUNDELL (1790—1878),
M.D. Edin., 1813; F.R.C.P. Lond., 1838;
Lecturer on Physiology and Midwifery at Guy's.

The principles and practice of obstetricy. To which are added notes and illustrations by Thomas Castle, illus., 8vo., Lond., 1834.

Lectures on the principles and practice of midwifery. Ed. by Charles Severn. Sm. 8vo., Lond., 1839.

The principles and practice of obstetric medicine. Carefully revised and corrected, with numerous additions and notes by Alexander Cooper Lee and Nathaniel Rogers, 8vo., Lond., 1840.

The above contains a remarkable dedication for a medical work, to the Duke of Wellington, by the publisher, who was Joseph Butler, of 4, St. Thomas's Street. It runs as follows: "To Arthur, Duke of Wellington, 'the hero of a hundred fights'; whose transcendent military talents are the boast of the present, and will be the wonder of the future: who has augmented the splendid catalogue of British victories on foreign shores; and to Cressy and Agincourt has added Waterloo: who conquered the great Conqueror of the age; who delivered Europe from military despotism, and Ireland from bigotry still more despotic; this volume is dedicated, with sentiments of profound admiration for his matchless talents, his dauntless fortitude, and his unfaltering decision of character, by the publisher."

See also Haighton and Blundell's A syllabus of the lectures on midwifery, 1821.

Dr. Blundell died worth £350,000.

JOHN BOSTOCK (1773—1846),
M.D. Edin.; F.R.S.;
Vice-President, Royal Society, 1832; Lecturer on Chemistry at Guy's; President of the Geological Society, 1826.

"To Bostock belongs the credit of giving the first complete description of the disease" (hay fever).—Dr. Norman Moore, Dict. Nat. Biog.

An essay on respiration. Parts I. and II. Plate, 8vo., Liverpool and Lond., 1804.

An elementary system of physiology. 3 vols., 8vo., Lond., 1824—1830.

ARTHUR EDWIN BOYCOTT,
M.A., B.Sc., B.Ch., M.D. Oxon.;

Lecturer in Pathology at Guy's, 1907—1912; Milroy Lecturer, R.C.P., 1911; Professor of Pathology, University of Manchester, 1913.

The Milroy Lectures on ankylostoma infection. Delivered before the Royal College of Physicians of London on March 2nd, 7th, and 9th, 1911. 8vo., Lond., 1911.

See also Guy's Hospital—Gull Lecturers' Reprints.

RICHARD BRIGHT (1789—1858),
M.D. Edin., 1812; F.R.S., 1821; F.R.C.P., 1832;

Goulstonian Lecturer, R.C.P., 1833; Lumleian Lecturer, R.C.P., 1837; Physician Extraordinary to Queen Victoria, 1837; President of the Royal Medical and Chirurgical Society, 1837—38.

"There has been no English physician—perhaps it may be said none of any country—since the time of Harvey who has effected so great a revolution in our habits of thought and methods of investigating morbid phenomena and tracing the etiology of disease as the late Dr. Richard Bright."—Dr. G. H. Barlow.

"Bright could not theorise, and fortunately gave us no doctrines and no 'views,' but he could see, and we are struck with astonishment at his powers of observation, as he photographed pictures of disease for the study of posterity. . . . I might also allude, in connection with Bright's marvellous power of observation, to the fact that he was one of the first who described acute yellow atrophy of the liver, pigmentation of the brain in miasmatic melanæmia, condensation of the lung in hooping-cough, unilateral convulsion without loss of consciousness in local cerebral diseases. He was also the first, I believe, who noted the bruit in chorea."—Sir Samuel Wilks, Guy's Hosp. Rep., 1877, xxii., 261—262.

"The lantern of Aristotle is too small to do more than transmit one ray of the brilliancy of that luminary of the ancient world, and the fame of Archimedes would have remained firm and unshaken without being attached to time by his screw; but these ancient examples show that the custom of affixing the names of discoverers to natural objects began in the earliest days of science. It was confirmed by the anatomists of the Renaissance and their successors, who are commemorated in the heart and in the brain and throughout the human frame, and has since been used by physicists and chemists and the followers of the system of scientific nomenclature begun by Linnæus. In later times this method of commemoration of discoverers has been extended to the names of diseases. Three physicians connected with Guy's Hospital have received this honour, of whom the most famous and widely known is Bright.

Bright's disease is a term used all over the world, and unlikely to fall out of use since it is applied to a group of morbid conditions, of which if some are detached and proved to be distinct others will remain."—Dr. Norman Moore, The Royal Medical and Chirurgical Society of London Centenary, 1805—1905.

"In his (Bright's) intellectual character the first feature that strikes us is a certain simplicity. Beyond most observers he succeeded in viewing objects without prejudice. Not putting forward any theories himself, he was not biased by any of the prevailing systems of medicine. Next, he had a remarkable tact, which appeared to be exercised unconsciously, of picking out the important facts in any subject, and, perhaps half unconsciously also, of combining them together so as to explain each other."—Dr. J. F. Payne, Dict. Nat. Biog.

Reports of medical cases, selected with a view of illustrating the symptoms and cure of diseases by a reference to morbid anatomy. 2 vols. Vol. 2 in two parts. Col. plates, 4to., Lond., 1827—31.

"The truth and importance of his researches were soon generally recognised. In a short time Morbus Brightii, or Bright's Disease, was a familiar appellation over the whole of Europe, and will doubtless preserve the memory of Bright so long as the disease is known by a separate name. Next to Laennec's discoveries in chest diseases, this of Bright's is perhaps the most important special discovery made in medicine in the first half of the nineteenth century. . . . Both volumes (Reports of Medical Cases) are illustrated with admirable plates, and taken together form one of the most important contributions to morbid anatomy ever made in this country by one person."—Dr. J. F. Payne, Dict. Nat. Biog.

Clinical memoirs on abdominal tumours and intumescence. Reprinted from Guy's Hospital Reports, edited by G. Hilaro Barlow, illus., 8vo., Lond., 1860.

With Dr. Thomas Addison. Elements of the practice of medicine. Vol. I. (No more published.) 8vo., Lond., 1839.

HERBERT ORPE BROOKHOUSE, M.D., B.S. Lond.

With Harold Chapple. "Ortocure": or, "don't-you-haf-anything-to-do-with-it-Yes." Illus., 8vo., Lond., 1908.

With Gilbert W. Dryland. "The Dean's Dilemma": or, "the Socialist, the sister, and the statue." Illus., 8vo., Lond., 1909.

See also Edridge and Brookhouse.

JOHN HENRY BRYANT,

M.D., B.S. Lond.; F.R.C.P. Lond., 1901.

Papers (collected reprints). 8vo., Lond., 1893—1905.

THOMAS BRYANT,

F.R.C.S., 1853; Hon. M.Ch., R.U.I., 1887; Hon. M.D. Dub., 1892;

Lettsomian Lecturer, 1863; Surgeon to Guy's, 1871—1888; President of the Medical Society of London, 1872; President of the Hunterian Society, 1873; President of the Clinical Society, 1885; President of the Royal College of Surgeons, 1890—1893; President of the Royal Medical and Chirurgical Society, 1898—1899; late Surgeon Extraordinary to Queen Victoria; Surgeon in Ordinary to King Edward VII., 1901—1910.

On the diseases and injuries of the joints. Clinical and pathological observations. 8vo., Lond., 1859.

Clinical surgery (in 7 parts). 8vo., Lond., 1860.

The surgical diseases of children, being the Lettsomian lectures, 1863. 8vo., Lond., 1863.

On ovariotomy (Clinical surgery, part 7). 8vo., Lond., 1867.

The practice of surgery, a manual. Illus., 8vo., Lond., 1872.

A manual for the practice of surgery. 3rd edit., revised and enlarged, 2 vols., illus., 8vo., Lond., 1879.

Ditto, 4th edit., 1884

Harveian Lectures on the mode of death from acute intestinal strangulation and chronic intestinal obstruction. 8vo., Lond., 1885.

The diseases of the breast. Col. plates, 8vo., Lond., etc., 1887.

Hunterian Lectures on tension, as met with in surgical practice. Inflammation of bone. And on cranial and intracranial injuries. Delivered before the Royal College of Surgeons, England, June, 1888. 8vo., Lond., 1888.

The Bradshaw Lecture on colotomy, lumbar and iliac, with special reference to the choice of operation. 8vo., Lond., 1890.

The Hunterian Oration, delivered in the presence of their Royal Highnesses the Prince of Wales and Duke of York, on the centenary of Hunter's death, February 14th, 1893. 8vo., Lond , 1893.

On villous growths and the common affections of the rectum Illus., 8vo., Lond., 1899.

FREDERIC FRANCIS BURGHARD,
M.S., M.D. Lond.; F.R.C.S.;
Surgeon to King's College Hospital, 1897.

A system of operative surgery, by various authors. Edited by F. F. Burghard, 4 vols., illus., 8vo., Lond., 1909.

With Sir Watson Cheyne. A manual of surgical treatment. 6 parts in 7 vols., illus., 8vo., Lond. and Bombay, 1899—1903.

ALFRED St. CLAIR BUXTON,
F.R.C.S. Edin.

Vision and vision testing. With practical tests. Illus., 8vo., Lond., 1887.

A. I.—*Presented by the author to the library of Guy's Hospital as a slight token of the happy hours spent there by him, March*, 1887.

THOMAS CALLAWAY, Jun.,
F.R.C.S. (Hon.), 1843; F.R.C.S. (by Exam.), 1849;
Assistant Surgeon to Guy's.

A dissertation upon dislocations and fractures of the clavicle and shoulder-joint. Being the Jacksonian Prize essay for 1846. Plates, 8vo., Lond., 1849.

A. I.—*Alfd. S. Taylor, Esq., F.R.S., with the Author's respects.*

ROBERT EDMUND CARRINGTON (1853—1887).

M.D. Lond., 1879;

Assistant Physician, and Physician for Cutaneous Diseases, at Guy's Hospital, 1883; Examiner in Anatomy, R.C.P.

A manual of dissections of the human body. Illus., 8vo., Lond.. 1881.

Ditto. 2nd edit., revised and enlarged, by W. Arbuthnot Lane, illus., 8vo., Lond., 1888.

Notes on pathology. A handbook for the post-mortem room. Edited, revised, and amplified by H. Evelyn Crook and Guy Mackeson, 8vo., Lond., 1892.

HAROLD CHAPPLE,

M.A., M.B., M.C. Camb.; F.R.C.S. Eng.

See Brookhouse and Chapple.

HENRY JAMES CHOLMELEY (1777—1837).

M.A., 1803, M.D., 1807, Oxon.; F.R.C.P. Lond., 1810;

Physician to Guy's, 1811—1837.

Outlines of a course of lectures on the practice of medicine, delivered in the Medical School of Guy's Hospital. Frontis., 8vo., Lond., 1820.

Interleaved copy, with a few notes. The frontispiece consists of an engraving of the front entrance of Guy's Hospital, with the following notice in copperplate underneath: "Six months pupils may have the privilege of becoming 12 months pupils by paying six guineas within two months after their first entry."

JOHN COOKE (1756—1838).

M.D. Leyden; F.R.C.P. Lond., 1807;

Physician to the London Hospital, 1784—1807; President of the Royal Medical and Chirurgical Society, 1821; Censor, R.C.P., 1811 and 1820: Croonian Lecturer, R.C.P., 1819, 1820. and 1821; Harveian Orator, R.C.P., 1828.

A treatise on nervous diseases, Vol. I. On apoplexy, including apoplexia hydrocephalica, or water in the head (Croonian Lectures of 1819. 8vo., Lond., 1820.

The library does not contain Vol. 2.

"Sets forth all then known on diseases of the nervous system, a book worth reading and comparing with such a modern treatise as that of Sir William Gowers by those who desire to know how much progress medicine has made in the last hundred years."—Dr. Norman Moore, The Royal Medical and Chirurgical Society of London Centenary, 1805—1905.

Sir ASTLEY PASTON COOPER (1768—1841), Baronet, 1821; F.R.S., 1802;

Lecturer on Anatomy, 1793—96, and on Comparative Anatomy, 1813—15, at the Royal College of Surgeons; Surgeon to Guy's, 1800—25; President of the Royal Medical and Chirurgical Society of London, 1819—20; President of the Royal College of Surgeons, 1827, and 1836; Sergeant-Surgeon to King William IV., 1828; Vice-President of the Royal Society, 1830.

"From the period of his appointment to Guy's until the moment of his latest breath, he was everything and all to the suffering and afflicted. His name was a host, but his presence brought confidence and comfort; and I have often observed that on an operating day, should anything occur of an untoward character in the theatre, the moment Sir Astley entered, and the instrument was in his hands, every difficulty was overcome, and safety generally ensured."—Dr. William Roots.

"I can never forget the enthusiasm with which he entered upon the performance of any duty calculated to abridge human suffering. . . . He was the idol of the Borough School. The pupils followed him in troops; and like to Linnæus, who has been described as proceeding upon his botanical excursions accompanied by hundreds of students, so may Sir Astley be depicted traversing the wards of the hospital with an equal number of pupils listening with almost breathless anxiety to catch the observations which fell from his lips. But on the days of operation this feeling was wound up to the highest pitch."—Dr. T. J. Pettigrew, Medical Portrait Gallery.

"Sir A. Cooper was a good anatomist, but never was a good operator where delicacy was required. He felt too much before he began ever to make a perfect operator. Quickness of perception was his forte, for he saw the nature of disease in an instant, and often gave offence by pouncing at once upon his opinion. The same faculty made his prognosis good. He was a good anatomist of morbid as well as of natural structure. He had an excellent and useful memory. In imagination he was vivid. His principle in practice was never to suffer any who consulted

him to quit him without giving them satisfaction on the nature and proper treatment of their case. My own success depended upon my zeal and industry, but for this I take no credit, as it was given to me from above."—Sir Astley Cooper's estimate of himself, found amongst his private papers after his death.

On hernia. Part I. The anatomy and surgical treatment of inguinal and congenital hernia. Part II. The anatomy and surgical treatment of crural and umbilical hernia. Plates, folio, Lond., 1804—07.

The illustrations to this work were so expensive that Sir A. Cooper lost a thousand pounds by it when every copy had been sold. Mr. Bettany, in the Dict. Nat. Biog., mentions the interesting fact that Sir Astley Cooper "suffered from hernia early in life, but was able to keep himself perfectly free from derangement by his own method of treatment."

The anatomy and surgical treatment of abdominal hernia. In two parts, 2nd edit., by C. Aston Key, plates, folio, Lond., 1827.

A. I.—*To John Hilton, Esq., as a mark of regards and esteem from his obliged friend and servant John Allen, Dulwich College, 25th February*, 1837.

Lectures on anatomy and the principal operations of surgery, delivered at the Theatre, St. Thomas's Hospital, between January 1st and June 1st, 1816. MS., 3 vols., 4to.

A printed label is affixed in each volume with the following: "A.D. 1899. Presented to the Library of Guy's Hospital, by H. W., a son of Joshua Waddington, F.R.C.S., the writer of these notes."

Outlines of the lectures on surgery, at St. Thomas's and Guy's Hospitals. 8vo., Lond., 1821.

Interleaved copy with a few notes.

A treatise on dislocations, and on fractures of the joints. Plates, 4to., Lond., 1822.

Ditto, 3rd edit., 1824.

Ditto, 4th edit., 1824.

Ditto, 6th edit., 1829.

A I.—*From Bransby B. Cooper to Henry Marshall Hughes*, 1828.

Ditto. A new edition much enlarged. Edited by Bransby B. Cooper, F.R.S., illus., 8vo., Lond., 1842.

The lectures of Sir Astley Cooper, Baronet, F.R.S., on the principles and practice of surgery; with additional notes and cases by Frederick Tyrrell. 3 vols., 8vo., Lond., 1824—27.

Formerly the property of Mr. Arthur E. Durham, with his characteristic autograph on the back of each title-page.

The principles and practice of surgery. Edited by Alexander Lee. 3 vols., plates, 8vo., Lond., 1836—43.

Illustrations of the diseases of the breast. In two parts. Part I. (non-malignant). Col. plates, 4to., Lond., 1829.

Observations on the structure and diseases of the testis. Col. plates, 4to., Lond., 1830.

The anatomy of the thymus gland. Col. plates, 4to., Lond., 1832.

A. I.—*To the Pupils of Guy's Hospital, with the best wishes of the Author.*

On the anatomy of the breast. With atlas. 2 vols., col. plates, folio, Lond., 1840.

A. I. in Vol. I.—*To my young friends, the Students of Guy's Hospital, this work is presented by their real friend, Astley Cooper, February 18th,* 1840.

With Benjamin Travers, F.R.S. Surgical essays. In two parts. Plates, 8vo., Lond., 1818—19.

MS. Lectures on surgery, delivered in the Theatre, St. Thomas's Hospital. Vol. I. (only), 8vo., no date.

MS. Lectures on surgery, given at St. Thomas's Hospital. Vols. I., II., and IV., 8vo., no date. The third volume is not in the library.

MS. Surgical Lectures. 2 vols., 8vo., no date.

"William Compson, October 1st, 1817—8," written on the fly-leaf, who, no doubt, was the writer of these lectures.

A great portion of his (Sir Astley Cooper's) practice was really medical, and in this department his treatment was very simple. "Give me," he would say, "opium, tartarised antimony, sulphate of magnesia, calomel, and bark, and I would ask for little else."—G. T. Bettany, Dict. Nat. Biog.

See also Bransby B. Cooper's Life of Sir Astley Cooper, Bart. 2 vols., 1843.

BRANSBY BLAKE COOPER (1792—1853),
F.R.S., 1828; F.R.C.S., 1843;
Surgeon to Guy's, 1825—1853.

A treatise on ligaments. Plates, 4to., Lond., 1825.

A. I.—*Charles Aston Key, Esqre., with the kindest regards of the Author.*

Ditto, 2nd edit., 1827.

A. I.—*To John Graves, from his sincere friend, Bransby B. Cooper.*

Surgical essays: the result of clinical observations made at Guy's Hospital. Col. plates, 8vo., Lond., 1833.

Lectures on anatomy. 4 vols., plates, 8vo., Lond., 1835.

The life of Sir Astley Cooper, Bart., interspersed with sketches from his note-books of distinguished contemporary characters. In two volumes. Port., 8vo., Lond., 1843.

Lectures on the principles and practice of surgery. 8vo., Lond., 1851.

MAURICE CRAIG,
M.A., M.D. Camb.; F.R.C.P. Lond.;

Physician for Mental Diseases, Lecturer in Mental Diseases, and Demonstrator in Psychology at Guy's Hospital.

Psychological medicine. A manual on mental diseases. Plates, 8vo., Lond., 1905.

Ditto, 2nd edit., 1912.

HERBERT EVELYN CROOK,
M.D., B.S. Lond.; F.R.C.S.;

Joint Editor of R. E. Carrington's Notes on pathology, 1892.

JAMES CURRY (—1819),
M.D. Edin., 1784;
Physician to Guy's, 1802—1819.

See W. Babington and Curry's Outlines of a course of lectures on the practice of medicine, 1802—06.

WILLIAM RADFORD DAKIN,
M.D., B.S.; F.R.C.P. Lond.;

Obstetric Physician and Lecturer on Midwifery at St. George's Hospital; President of the Obstetrical Society of London, 1889—1890.

A handbook of midwifery. Illus., 8vo., Lond., 1897.

JOHN THOMPSON DICKSON,

The science and practice of medicine in relation to mind, the pathology of nerve centres, and the jurisprudence of insanity. Being a course of lectures delivered in Guy's Hospital. Illus., 8vo., Lond., 1874.

ERNEST BLAIR DOWSETT,
Assistant Dental Surgeon to Guy's.

Dental surgery notes. 8vo., Lond., 1909.

HERBERT EDWARD DURHAM,
M.A., M.B., B.C., Sc.D. Camb.;

John Lucas Walker Student, Cambridge University; Gull Research Student in Pathology, Guy's Hospital.

See Guy's Hospital—Gull Students' Reprints, Vol. I.

RAY EDRIDGE.

With H. O. Brookhouse. "The spoils of victory": or, a very pleasant line of thought. Illus., 8vo., Lond., 1909.

WILLIAM ENGLAND,
M.D. Edin., 1829;

Observations on the functional disorders of the kidneys, which give rise to the formation of urinary calculi; with remarks on their frequency in the county of Norfolk. 8vo., Lond. and Norwich (1830).

A. I.—*To the Physical Society of Guy's Hospital, with the Author's best wishes for its prosperity.*

JOHN WILLIAM HENRY EYRE,
M.D., M.S. Dur.; F.R.S. Edin.;

Director of the Bacteriological Department, Guy's Hospital; Chairman of the working party of the Royal Society Commission on Mediterranean Fever at Malta, 1906; Erasmus Wilson Lecturer, Royal College of Surgeons, 1908; Milroy Lecturer, Royal College of Physicians, 1908; Hunterian Professor, Royal College of Surgeons, 1911—12.

The elements of bacteriological technique. A laboratory guide for the medical, dental, and technical student. Illus., Philad. and Lond., 1902.

With W. C. Bosanquet. Serums, vaccines, and toxins, in treatment and diagnosis. 2nd edit., thoroughly revised, 8vo., Lond., etc., 1909.

Translator of Angelo Celli's Malaria according to the new researches. Translated from the 2nd Italian edition, illus., 8vo., Lond., etc., 1900.

Translator of Achille Monti's The fundamental data of modern pathology. New Syd. Soc., 8vo., Lond., 1900.

CHARLES HERBERT FAGGE,
M.S. Lond.; F.R.C.S. Eng.;
Assistant Surgeon to Guy's.

"The Pocket Gray," or anatomist's vade-mecum, by the late Edward Cotterell. 5th edition, revised and edited by C. H. Fagge, 8vo., Lond., 1901.

The pocket anatomy (formerly "The Pocket Gray"). 6th edit., 8vo., Lond., 1908.

CHARLES HILTON FAGGE (1838—1883),
M.D. Lond., 1862; F.R.C.P. Lond., 1870;
Physician and Lecturer on Pathology at Guy's.

The principles and practice of medicine. 2 vols., 8vo., Lond., 1886.

With P. H. Pye-Smith. Ditto, 2nd edit., 1888.

Ditto, 3rd edit., 1891.

Ditto, 4th edit., 1901—02.

See also Guy's Hospital Museum. Catalogue of the Models, etc., 1876.

JOHN FAWCETT,
M.D., B.S. Lond.; F.R.C.P., F.R.C.S. Lond.;
Physician to Guy's Hospital; Dean of the Medical and Dental Schools, Guy's Hospital, 1901—03.

See Guy's Hospital—Beaney Scholars' Reprints.

Also Guy's Hospital Museum—Descriptive catalogue of the Pathological specimens. 3rd edit., 1894—1910.

HORACE FINNEMORE,
B.Sc. Lond.; F.I.C.;
Pharmacist to and Teacher of Pharmacy at Guy's Hospital; Member of the Board of Examiners of the Pharmaceutical Society of Great Britain.

Pharmacy and materia medica for nurses. Founded on the lectures given to the nurses at Guy's Hospital. 8vo., Lond., 1913.

JOHN COOPER FORSTER (1823—1886),
M.B. Lond., 1847; F.R.C.S. Eng., 1849;
Physician to Guy's, 1870—1880.

The surgical diseases of children. Col. plates, 8vo., Lond., 1860.

A. I. — *With the Author's kind regards. Presented to the Library for the use of the students.*

DAVID FORSYTH.
M.D., D.Sc. Lond.; F.R.C.P. Lond.;

Children in health and disease: a study of child-life. Frontis., 8vo., Lond., 1909.

JOHN MATTHEW FORTESCUE-BRICKDALE,
M.A., B.Ch., M.D. Oxon.;
Clinical Lecturer and Director, Public Health Laboratory, University of Bristol.

A practical guide to the newer remedies. 8vo., Bristol and Lond., 1910.

HENRY ISAAC FOTHERBY,
L.S.A., 1846; M.D. Lond., 1867;
Physician to Metropolitan Free Hospital.

Scientific associations, their rise, progress, and influence, with a history of the Hunterian Society. An oration delivered on the fiftieth anniversary of the above Institution, February 10th, 1869. 8vo., Lond., 1869.

CLAUD FRANCIS FOTHERGILL,
B.A., M.B., B.C. Camb.

Blood examination and its value in tropical disease. With a preface by Sir Ronald Ross, F.R.S., C.B. 8vo., Lond., 1907.

JOSEPH FOX,
Dentist to Guy's Hospital.

The natural history of the human teeth. Plates, 4to., Lond., etc., 1803.

The history and treatment of the diseases of the teeth, the gums, and the alveolar processes, with the operations which they respectively require. To which are added, observations on other diseases of the mouth, and on the mode of fixing artificial teeth. Plates, 4to., Lond., etc., 1806. Bound with the previous work.

"Joseph Fox was probably the first dentist in England who wrote a scientific work on the teeth and gave lectures at a medical school."—Wilks and Bettany's History of Guy's Hospital.

FRANCIS EDWARD FREMANTLE,
M.A., M.Ch. Oxon.; F.R.C.S. Eng.; F.R.C.P. Lond.;
D.P.H.; R.C.P.S.
Medical Officer of Health and School Medical Officer, Hertford County Council; Ex-President, Home Counties Branch, Society

of Medical Officers of Health; Edward Jenner Lecturer, St. George's Hospital Medical School, 1909—11; late House Physician, Guy's Hospital; Civil Surgeon, Field Force, South African War; Assistant Secretary, War Office Commission for Re-Organization of Army Medical Service; Plague Medical Officer, Punjab District, 1903—04; Special Correspondent of *Lancet*, Russo-Japanese War, 1904; Editor of *Guy's Hospital Gazette*, 1896.

Impressions of a doctor in khaki. Illus., 8vo., Lond., 1901.

A traveller's study of health and empire. Illus., 8vo., Lond., (1911).

A. I.—*To Wills Library, Guy's Hospital Medical School, from an old Guy's man, the Author, Francis Fremantle. March*, 1912.

HERBERT STANLEY FRENCH,

M.A., M.D., B.Ch. Oxon.; F.R.C.P. Lond.; L.S.A.

University Scholar, Guy's Hospital, 1898; Beaney Scholar, 1901; Gillson Scholar, Society of Apothecaries, 1902—03; Radcliffe Travelling Fellow, Oxford, 1902—04; Goulstonian Lecturer, Royal College of Physicians, 1908; Honorary Wills Librarian, Guy's Hospital, 1909; Assistant Physician and Pathologist to Guy's Hospital, 1906.

Medical laboratory methods and tests. Illus., 8vo., Lond., 1904.

Ditto, 2nd edit., col. plates, 8vo., Lond., 1908.

Ditto, 3rd edit., 1912.

On the influence of pregnancy upon certain medical diseases, and of certain medical diseases upon pregnancy. Goulstonian Lectures, 1908. 8vo., (Lond., 1908).

An index of differential diagnosis. By various writers. Edited by Herbert French. Col. plates, etc., 8vo., Bristol, Lond., and New York, 1912.

See also Guy's Hospital—Beaney Scholars' Reprints.

Sir ALFRED FRIPP, Kt.,
K.C.V.O., C.B.; M.B., M.S. Lond.; F.R.C.S. Eng.;
Surgeon in Ordinary to the King and the Duke of Connaught: late Surgeon in Ordinary to King Edward VII.; Surgeon to, and Lecturer upon Surgery at, Guy's Hospital.

With A. R. Thompson. Human anatomy for art students. With drawings by Innes Fripp, and an appendix on comparative anatomy by Harry Dixon. Plates, etc., 8vo., Lond., 1911.

ALFRED LEWIS GALABIN (1843—1913),
M.A., M.D. Camb.; F.R.C.P. Lond., 1878;

Lecturer on Clinical Midwifery in 1874, Obstetric Assistant Physician in 1875, and Lecturer on Diseases of Women in 1876, at Guy's Hospital; Obstetric Physician to Guy's, 1882—1903; Lecturer on Midwifery at Guy's, 1887—1903; Consulting Obstetric Physician to Guy's, 1903—1913; President of the Obstetrical Society of London, 1889—1890; President of the Hunterian Society, 1889—1890. Dr. Galabin graduated at Cambridge in 1866 as a double-first in classics and mathematics, being seventeenth wrangler, and eighth in the classical tripos.

The student's guide to the diseases of women. Illus., 8vo., Lond., 1879.

Ditto, 3rd edit., 1884.

Diseases of women. 4th edit., 1887.

Ditto, 5th edit., 1893.

Ditto, 6th edit., 1903.

A manual of midwifery. Illus., 8vo., Lond., 1886.

Ditto, 4th edit., 1897.

Ditto, 5th edit., 1900.

Ditto, 6th edit., 1904.

HASTINGS GILFORD,
F.R.C.S. Eng.

The disorders of post-natal growth and development. Illus., 1a. 8vo., Lond., 1911.

KENNETH WELDON GOADBY,

Erasmus Wilson Lecturer, Royal College of Surgeons, 1907; Hunterian Professor, Royal College of Surgeons, 1911.

The mycology of the mouth. A text-book of oral bacteria. Illus., 8vo., Lond., etc., 1903.

CUTHBERT HILTON GOLDING-BIRD,

B.A., M.B. Lond.; F.R.C.S. Eng.

An account of the United Hospitals' Club from its foundation, February 14th, 1828, to its diamond jubilee, February 13th, 1903. 8vo., Lond., 1904.

Mr. Golding-Bird on page 11 of his book draws attention to a slight, but certainly excusable, mistake that was made many years ago, which was nothing less than the canonization of Thomas Guy. It occurs in an entry of an old register of Deadman's Place Meeting House, where it is recorded that in 1758 "Mrs. Draper is buried from St. Guy's Hospital." It might also be mentioned that there is another reference to "St. Guy's" recorded on page 115 of Wilks and Bettany's "History." This occurs in an "Essay on Delirium Tremens" by Thomas Sutton, M.D., in 1813, where he refers to "Dr. William Saunders, late physician to St. Guy's, and for many years lecturer on medicine at that hospital." Although Mr. Golding-Bird has pointed out this error of the canonization of Guy, he himself falls into a rather similar one, in the paragraph immediately preceding, by conferring knighthood on the Founder of the Hospital. The paragraph referred to states: "Guy's Hospital owes its foundation and endowment to the charity of Sir Thomas Guy, Knight." In a recent number of the *Gazette*, November 23rd, 1912, Mr. Wingent mentions that he has discovered yet another reference to "St. Guy's Hospital," and this one is made by no less a personage than a Prime Minister of England, viz., Sir Robert Peel, who, in a letter to Queen Victoria, dated September 15th, 1845, describes the Rev. F. D. Maurice as "Chaplain of *St. Guy's* Hospital." It seems very evident from these allusions that Thomas Guy is enshrined in an odour of sanctity, and although the ceremony of beatification has not been performed at Rome on his behalf, there are a goodly number of persons in whose hearts he has received canonization.

EDWARD WILBERFORCE GOODALL,

M.D., B.S. Lond.

With J. W. Washbourne. A manual of infectious diseases. Illus., 8vo., Lond., 1896.

Ditto, 2nd edition, revised and enlarged, by E. W. Goodall. Plates, etc., 8vo., Lond., 1908.

EDWIN GOODALL,
M.D., B.S. Lond.; F.R.C.P. Lond.;
Medical Superintendent, Cardiff Mental Hospital.

See Savage and Goodall's Insanity and allied neuroses, 1907.

Sir JAMES FREDERIC GOODHART, Baronet,
M.D., C.M., LL.D. Aber; F.R.C.P. Lond.:
Physician to Guy's, 1886–99; President of the Harveian Society, 1898.

The students' guide to diseases of children. 8vo., Lond., 1885.

The diseases of children. 4th edit., 8vo., Lond., 1891.

Ditto, 6th edit., with the assistance of George Frederic Still. 8vo., Lond., 1899.

Ditto, 8th edit. Edited by G. F. Still. 8vo., Lond., 1905.

Ditto, 9th edit. Edited by G. F. Still. Illus., 8vo., Lond., 1910.

On common neuroses, or the neurotic element in disease and its rational treatment. Three lectures delivered before the Harveian Society of London, November–December, 1891. 2nd edit., 8vo., Lond., 1894.

The passing of morbid anatomy. The Harveian Oration for 1912, delivered at the Royal College of Physicians on St. Luke's Day, October 18th, 1912. 8vo., Lond., 1912.

Sir WILLIAM WITHEY GULL, Bart. (1816–1890),
M.D. Lond., 1846; F.R.C.P. Lond., 1848; F.R.S., 1858; D.C.L. Oxon., 1868; LL.D. Camb., 1880; LL.D. Edin., 1884;
Lecturer on Physiology and Comparative Anatomy at Guy's, 1846–56; Goulstonian Lecturer, 1849; Hunterian Orator, 1861; Harveian Orator, 1870; Physician to Guy's, 1858–68; President of the Clinical Society, 1871–72; Physician to the Prince of Wales, 1871; Physician in Ordinary to Queen Victoria, 1887–90.

A collection of the published writings of William Withey Gull, Bart., M.D., F.R.S. Edited by Theodore Dyke Acland, M.D. 2 vols., New Syd. Soc.

Medical papers—Memoir and Addresses. Port., 8vo., Lond., 1894—96.

Sir W. Gull left personalty worth over £344,000 besides landed estates, a fortune unprecedented in the history of medicine.

Hommage à notre Fondateur.

THOMAS GUY (1645—1724),

Freeman of the Stationers' Company, 1668; Freeman of the City of London, 1668; set up as a bookseller in a corner house at the junction of Cornhill and Lombard Street, 1668; founded an almshouse for women at Tamworth, 1678, enlarged for men, 1692; one of the Oxford University printers, 1679—92; M.P. for Tamworth, 1695—1707; built Tamworth Town Hall, 1701; a Governor of St. Thomas's Hospital, 1704; built and furnished three wards in St. Thomas's Hospital, 1707; sole founder of Guy's Hospital in his lifetime, erected at a cost of £18,793, with an endowment of £200,000, to "receive and entertain therein four hundred poor persons, or upwards, labouring under any distempers, infirmities or disorders, thought capable of relief by physic or surgery."

"Mr. Thomas Guy, our incomparable benefactor."—From the Resolution passed by Tamworth Corporation, July 21st, 1693.

"Deeds rather than words" was the characteristic of Thomas Guy.—Wilks and Bettany's History.

He established this asylum for that stage of languor and disease to which the charity of others had not reached; he provided a retreat for hopeless insanity, and rivalled the endowment of kings.—From Guy's Epitaph.

A copy of the last will and testament of Thomas Guy, Esq. 8vo., Lond., 1732. Presented to the Library by Mr. R. M. Wingent.

A copy of the last will and testament of Thomas Guy, Esq. 8vo., Lond., 1732 (modern reprint).

Published by Guy. Death's vision represented in a philosophical, sacred poem. 4to., Lond., printed for Thomas Guy at the Oxford Arms in Lumber Street, 1709.

A.I.—"Presented to Guy's Hospital by Joshua W. Butterworth, F.S.A., law publisher, one of the Governors of this Hospital, at a General Court held on the 14th November, 1888. This book being, by its imprint, an illustration that the illustrious Founder was a *publisher*, as well as a bookseller."

Four years after the above was written by Mr. Butterworth, Wilks and Bettany's History of Guy's Hospital was published. After what must have entailed a great amount of research on the part of the authors, they were able to record the titles of a large number of books published by Guy. These may be found on pages 10—16, and a list of Oxford University Bibles and Prayer Books on pages 34 and 35. Probably the only book Guy published relating to a medical subject was Dr. John Freind's Emmenologia. Wilks and Bettany in their History mention that "an entry in the Copyright Registry of the Stationers' Company, dated March 24th, 1710—11, records the whole copyright of 'Emmenologia' as belonging to Thomas Guy."

GUY'S HOSPITAL.

Guyoscope, The, vols. 1—2. Illus., 4to. and 8vo., Lond., 1897—1898. Vol. 1, new series, illus., 8vo., Lond., 1905—06. No more published.

Guy's Hospital, 1724—1902: a tribute to its Founder and a record of its work. Illus., obl. 8vo., Lond., 1903.

Guy's Hospital—Album of photographs of Guy's men, including—

C. Bader.
James Bankart.
G. H. Barlow.
John Birkett.
Thomas Bryant.
Edward Cock.
Arthur Durham.
C. Hilton Fagge.
J. Cooper Forster.
"Old" Golding, *Janitor.*
Sir W. W. Gull.
S. O. Habershon.
J. Braxton Hicks.
Monson Hills, *Cupper.*
John Hilton.
James Hinton.
Walter Moxon.
W. Odling.
Henry Oldham.
F. W. Pavy.
Alfred Poland.
G. Owen Rees.
J. C. Steele, *Superintendent.*
James Stocker, *Apothecary.*
Alfred S. Taylor.
Thomas Turner, *Treasurer.*
Sir Samuel Wilks.

Presented to the Library by Dr. Astley Vavasour Clarke, of Leicester, in 1907. 4to.

Guy's Hospital—Album of photographic views of Guy's Hospital. Oblong folio, no date.

Guy's Hospital—Beaney Scholars' Reprints. 8vo., Lond., etc. 1894—1904. Containing papers by the following:—John Fawcett, 1893—97; John Beresford Leathes, 1897—99; Herbert French, 1901—04.

Guy's Hospital—Clinical cases and lectures taken by Joshua Waddington at Guy's Hospital, between December 1st, 1816, and May 1st, 1817 (MS.).

Contains printed label pasted on inside cover with the following: "A.D., 1899. Presented to the Library of Guy's Hospital, by H. W., a son of Joshua Waddington, F.R.C.S., the writer of these notes.

Guy's Hospital Gazette, 1st series, vols. 1—6, 4to., Lond., 1872—75.

Guy's Hospital Gazette, new series, vols. 1—3, 4to., Lond., 1876—78.

Guy's Hospital Gazette, new series, vols. 1— , 4to., Lond., 1887—

The following note, by Dr. F. Taylor, is written in the first volume of the *Gazette*: "This, the original series of the *Guy's Hospital Gazette*, was edited and issued as his own venture, by Thomas Cattell Jones, a second years' student. It was continued by him for some time; but was eventually taken over by the President of the Pupils' Physical Society. Frederick Taylor, April 4th, 1912."

The following have been editors or joint-editors of *Guy's Hospital Gazette* since 1896: F. E. Fremantle, H. L. Eason, L. E. Stamm, D. G. Greenfield, J. M. Brydone, R. Edridge, F. W. Morton-Palmer, H. C. Cameron, C. L. Leipoldt, M. E. Ball, L. Mandel, A. Neville Cox, E. G. Schlesinger, J. A. Ryle, F. G. Scott, and G. T. Mullally.

Guy's Hospital—Gordon Lecturers' Reprints, vol. 1. 8vo. and fol., Lond., 1902. Contains papers by:—E. W. Ainley Walker; A. E. Boycott; F. A. Bainbridge.

Guy's Hospital—Gull Students' Reprints, vol. 1. 8vo. and 4to., Lond., 1891. Contains papers by:—F. G. Hopkins; H. E. Durham; Alfred Salter; E. I. Spriggs; R. W. Allen.

Guy's Hospital Medical School Calendar, 1900—01— 8vo., Lond., 1901—01

Guy's Hospital Museum—A catalogue of the models of diseases of the skin in the Museum of Guy's Hospital, arranged according to the classification of Willan and Bateman. By S. O. Habershon. 8vo., Lond., 1854.

Guy's Hospital Museum—Catalogue of the models of diseases of the skin in the Museum of Guy's Hospital. By C. Hilton Fagge. 8vo., Lond., 1876.

Guy's Hospital Museum—Catalogue of the preparations in the Anatomical Museum of Guy's Hospital, arranged and edited by Thomas Hodgkin. 8vo., Lond., 1829.

Guy's Hospital Museum—Pathological catalogue of the Museum of Guy's Hospital—Organs of digestion. Enlarged and revised from the original catalogue of Dr. Hodgkin, by S. O. Habershon. 8vo., Lond., 1857.

Guy's Hospital Museum—Catalogue of the pathological preparations in the Museum of Guy's Hospital. Revised and edited by Samuel Wilks, 2 vols. 8vo., Lond., 1863.

Vol. 1, Part 1. Diseases of the bones, joints, etc.

Part 2. Diseases of the heart and circulatory system.

Part 3. Diseases of the nervous system, integument, and senses.

Part 4. Diseases of the vocal and respiratory organs.

Vol. 2, Part 5. Diseases of the organs of digestion.

Part 6. Diseases of the urinary, and male genital organs, and peritoneum. Appendix.

Formerly Mr. John Hilton's copy, with his autograph.

Guy's Hospital Museum—Descriptive catalogue of the pathological specimens contained in the Museum of Guy's Hospital. 3rd edition, by Lauriston E. Shaw, Sir E. Cooper Perry, and John Fawcett. 3 vols., 8vo., Lond., 1894—1910.

Vol. 1. Morbid conditions of the respiratory organs and alimentary tract.

Vol. 2. Morbid conditions of the liver, pancreas, spleen, suprarenal bodies, urinary organs, and male genitalia.

Vol. 3. Morbid conditions of the pericardium, heart aorta, pulmonary artery, arteries and veins, spinal cord, brain, and nerves.

Guy's Hospital Museum—Catalogue of the preparations of comparative anatomy in the Museum of Guy's Hospital. By P. H. Pye-Smith.

Guy's Hospital—Pharmacopœia in usum valetudinarii a Thoma Guy, Armigero fundati, ad normam recensitæ editionis Pharmacopœiæ Collegii Regalis Medicorum Londinensis, reformata. Sm. 8vo., Lond., 1791, 1st edition.

Guy's Hospital—Pharmacopœia in usum nosocomii à Thoma Guy, Armigero, A.D. MDCCXXI fundati. Sm. 8vo., Lond., 1826.

Interleaved copy.

Guy's Hospital—Pharmacopœia in usum nosocomii a Thoma Guy, Armigero, A.D. MDCCXXI fundati. Sm. 8vo., Lond., 1837.

Guy's Hospital Pharmacopœia—Formulæ used at Guy's Hospital, in addition to those in the British Pharmacopœia, 1868. 8vo., Lond., 1868.

Guy's Hospital Pharmacopœia—Formulæ used at Guy's Hospital, in addition to those in the British Pharmacopœia. Compiled by a Committee of the Staff. Sm. 4to., Lond., 1879.

Guy's Hospital Pharmacopœia—The Pharmacopœia of Guy's Hospital. Compiled by a Committee of the Staff. Sm. 8vo., Lond., 1891.

Guy's Hospital Pharmacopœia—The Pharmacopœia of Guy's Hospital. Compiled by a Committee of the Staff. 8vo., Lond., 1899, 9th edition.

Guy's Hospital.—Physical Society. A catalogue of books in the library of the Physical Society, Guy's Hospital. 4to., Lond.

Consists of the printed copy of 1823 with MS. additions.

Guy's Hospital.—Physical Society. Laws of the Physical Society, 1775. Folio, MS., 1775.

Guy's Hospital.—Physical Society. Transactions. MS. Vols. for 1775—83; 1794—98; 1804—12; 1813—20; 1835—40. 4to. and folio, 1775—1840.

The Physical Society of Guy's Hospital was founded in 1771, and is, therefore, as old as the Medical Society of London, or even older according to some accounts. Its members were composed for the most part of the officers of the two hospitals, together with the general practitioners in their neighbourhood, although numerous other medical men of celebrity joined the Society. . . . It was at one time the most flourishing institution of the kind in London; but owing to the formation of other societies in more fashionable parts of the town, it began to wane, and finally ceased to exist in 1852.—Wilks and Bettany's Biographical History of Guy's Hospital.

"Instituted in the year 1773" appears on the title-page of the first five volumes of "Memoirs of the Medical Society of London," published during 1787—1799. The Physical Society of Guy's, instituted in the year 1771, can therefore claim priority of this Society, with respect to foundation, by about two years.—W. W.

Guy's Hospital. — Physiological Laboratory, Guy's Hospital Medical School (University of London). Collected Papers, 1900—1904. 8vo., and la. 8vo., Lond., 1900—

Guy's Hospital.—Physiological Society's Papers (MS.). Vols. for 1900—01 to 1910—11. Folio, 1900—01.

Guy's Hospital.—Pupils' Physical Society. Transactions. MS. Volumes for 1830—32; 1836—43; 1857—70; 1870—95. 4to., 1830—95.

Guy's Hospital Reports.—1st series, vols. 1—7, 8vo., Lond., 1836—42. 2nd series, vols. 1—9, 8vo., Lond., 1843—54. 3rd series, vol. 1— 8vo., Lond., 1855—

The well-known Guy's Hospital Reports started into existence in the year 1836, and their publication has continued up to the present time with great success. . . . Other hospitals soon attempted to follow the example, but for some reasons not very obvious failed after the issue

of two or three volumes to continue the good work. Of late they have been more successful. Nothing, perhaps, has done more to establish the reputation of Guy's Hospital abroad than these Reports. They may be found in the best libraries in Europe and in America, and have been well perused by many of the leading men on the Continent.—Sir Samuel Wilks, Biographical History of Guy's Hospital.

The following is a complete list of those who have been editors or joint-editors of the Reports: G. H. Barlow, J. P. Babington, Edward Cock, E. L. Birkett, J. H. Browne, A. Poland, S. Wilks, C. H. Fagge, A. E. Durham, H. G. Howse, F. Taylor, N. Davies-Colley, W. Hale White, W. H. A. Jacobson, E. C. Perry, J. H. Bryant, F. J. Steward, and Herbert French.

Guy's Hospital.—Statistical Tables of the patients treated in Guy's Hospital, 1854—78. By John Charles Steele, Superintendent.

Guy's Hospital.—Treasurer's Reports. 2 vols., 1887—92, 1893—95. 8vo., Lond., 1887—95.

Guy's Hospital.—Ward prayers, 8vo., Lond., 1906.

SAMUEL OSBORNE HABERSHON (1825—1889).

M.D. Lond., 1851; F.R.C.P. Lond., 1856; Physician to Guy's, 1866—80; Lecturer on Materia Medica, 1856—73; and Medicine, 1873—77; Vice-President of the Royal College of Physicians, 1887; Lumleian Lecturer, 1876; Harveian Orator, 1883; President of the Medical Society of London, 1873.

Habershon "was the first in England to propose the operation of gastrostomy for stricture of the œsophagus, which Cooper Forster performed on a patient of Habershon's in 1858."—G. T. Bettany, Dict. Nat. Biog.

Pathological and practical observations on diseases of the alimentary canal, œsophagus, stomach, cæcum, and intestines. Plates, 8vo., London., 1857.

Pathological and practical observations on diseases of the abdomen, comprising those of the stomach, and other parts of the alimentary canal, œsophagus, cæcum, intestines, and peritoneum. 2nd edit., plates, 8vo., Lond., 1862.

Ditto, 3rd edit., 1878.

Ditto, 4th edit., 1888.

On the injurious effects of mercury in the treatment of disease. 8vo., Lond., 1860.

On diseases of the stomach, the varieties of dyspepsia, their diagnosis and treatment. 8vo., Lond., 1866.

Ditto, 3rd edit., 1879.

The Lettsomian Lectures delivered at the Medical Society of London, 1872, on the pathology and treatment of some diseases of the liver. 8vo., Lond., 1872.

On the pathology of the pneumogastric nerve, being the Lumleian Lectures delivered at the Royal College of Physicians of London, 1876. 8vo., Lond., 1877.

The advancement of science by experimental research. The Harveian Oration delivered at the Royal College of Physicians, June 27th, 1883. 8vo., Lond., 1883.

See also Guy's Hospital Museum. A catalogue, etc., 1854.

See also Guy's Hospital Museum. Pathological catalogue, etc., 1857.

JOHN HAIGHTON (1755—1823), M.D., F.R.S.;

Lecturer on Physiology and Midwifery to St. Thomas's and Guy's, 1788; Silver Medallist of the London Medical Society for paper on "Deafness," 1790.

(MS.) Lectures on the physiology of the human body, delivered at Guy's Hospital, 1796. 4to.

Formerly the property of Martin Tupper, with his autograph on title-page. A. L.—"Bought at the Cobham sale, Gravesend, Feb., 1909. Presented to the Wills Library of Guy's Hospital by F. W. C., April 22nd, 1909." This book contains the bookplate of the giver, Dr. F. Wm. Cock, M.D., F.S.A., in which a fine bird of the Galliformes Order figures prominently, with the appropriate motto underneath, "Whilst I live I'll crow."

A syllabus of the lectures on midwifery delivered at Guy's Hospital and at Dr. Haighton's theatre, in St. Saviour's Church-Yard, Southwark. 8vo., Lond., re-printed in the year 1803.

Contains the following letter:

38, Harrington Road,
Preston, Brighton,
May 15th, 1906.

Dear Dr. Galabin,

This ancient Syllabus of lectures delivered at Guy's which was *re*-printed 103 years ago may be of interest to you as a curiosity, or perhaps they may like it for the library at the Hospital. Anyhow, it seems a pity for it to become destroyed or utterly lost. It was formerly my great uncle's (Harry Blaker), who was surgeon to the Prince Regent when he frequented Brighton. At that time of day he (the Prince) suffered from boils on the nape of the neck, and in consequence wore high collars and mufflers round his neck. This was the origin of that cumbrous fashion which lasted long after the Prince's (King's) death.

With compts.,
I remain,
Very faithfully yours,
Thos. F. J. Blaker
(Guy's 1873 to 1877).

P.S.—The Prince left my uncle and placed himself under the care of Sir Astley Cooper in town as I understand the matter.

(MS.) Lectures on midwifery and diseases of women and children. Delivered at the Theatre, Guy's Hospital, between the 1st of November, 1816, and the 1st of March, 1817. 4to.

Printed label affixed in volume with the following: "A.D. 1899. Presented to the Library of Guy's Hospital by H. W., a son of Joshua Waddington, F.R.C.S., the writer of these notes."

With William Blundell. A syllabus of the lectures on midwifery delivered at Guy's Hospital. 8vo., Lond., 1821.

Interleaved copy, with MS. notes. Formerly Mr. John Hilton's, with his bookplate.

WILLIAM SAMPSON HANDLEY,

M.S., M.D. Lond.: F.R.C.S. Eng.:

Assistant Surgeon to Middlesex Hospital; Astley Cooper Prizeman, 1904; Hunterian Professor of Surgery and Pathology, R.C.S., 1905—1910.

Cancer of the breast and its operative treatment. Illus., 8vo., Lond., 1906.

ARTHUR FREDERICK HERTZ,

M.A., M.D., B.Ch. Oxon.; F.R.C.P. Lond.:

Radcliffe Travelling Fellow, University of Oxford, 1905; Radcliffe Prizeman for Medical Research, 1909; Goulstonian Lecturer, R.C.P., 1911; Assistant Physician and Physician for Nervous Diseases and to Electrical Department, Guy's Hospital.

Constipation and allied disorders. Illus., 8vo., Lond., 1909.

La constipation et les troubles intestinaux qui s'y rattachent. Traduction Française par A. E. E. Reboul. Avec une préface du Dr. J.-Ch. Roux. Illus., 8vo., Paris, 1912.

The Goulstonian Lectures. On the sensibility of the alimentary canal. Delivered at the Royal College of Physicians on March 14th, 16th, and 21st, 1911. 8vo., Lond., 1911.

CHARLES HIGGENS,

F.R.C.S. Eng.;

Senior Consulting Ophthalmic Surgeon, Guy's Hospital; Formerly Ophthalmic Surgeon and Lecturer on Ophthalmology, Guy's Hospital.

Hints on ophthalmic out-patient practice. 2nd edit., 8vo., Lond., 1879.

A manual of ophthalmic practice. Illus., 8vo., Lond., 1888.

Ditto, 2nd edition, revised and edited by Arthur W. Ormond. Illus., 8vo., Lond., 1903.

Mr. Higgens was the ophthalmic surgeon who attended General Booth in his last illness. An interesting account of this is given in Mr. Higgen's article entitled "A note on a case of double cataract: the case of General Booth," in the *Lancet*, October 19th, 1912, pages 1073—1074.

MONSON HILLS (1792—1853),

Cupper to Guy's Hospital.

A treatise on the operation of cupping. 2nd edition, enlarged. Plates, sm. 8vo., Lond., 1839.

JOHN HILTON (1804—1878),
F.R.C.S. Eng., 1843; F.R.S.;

Surgeon to Guy's Hospital, 1849—70; President of the Medical Society of London, 1859; Professor of Human Anatomy and Surgery, R.C.S., 1860—62; President of the Royal College of Surgeons, 1867; President of the Pathological Society, 1871—72; Surgeon Extraordinary to Queen Victoria.

Notes on some of the developmental and functional relations of certain portions of the cranium. Selected by Frederick William Pavy from the lectures on anatomy delivered at Guy's Hospital by John Hilton. Plates, 8vo., Lond., 1855.

On the influence of mechanical and physiological rest in the treatment of accidents and surgical diseases, and the diagnostic value of pain. Illus., 8vo., Lond., 1863.

"Bacon says there are books to skim over, books to read parts of, and books to absorb: Hilton's book on 'Rest and Pain' is one to absorb."—Prof. John Chiene, Address in Surgery before the British Medical Association, 1891.

Rest and pain: a course of lectures on the influence of mechanical and physiological rest in the treatment of accidents and surgical diseases, and the diagnostic value of pain. 3rd edition. Edited by W. H. A. Jacobson. Illus., 8vo., Lond., 1880.

Ditto, 4th edit., illus., 8vo., Lond., 1887.

JAMES HINTON (1822—1875),
Aural Surgeon to Guy's, 1863.

Man and his dwelling-place. An essay towards the interpretation of nature. 2nd edit., 8vo., Lond., 1861.

Life in nature. Illus., 8vo., Lond., 1862.

The place of the physician, being the introductory lecture at Guy's Hospital, October, 1873. With other essays. 8vo., Lond., 1874.

Atlas of the membrana tympani, with descriptive text. Being illustrations of diseases of the ear. Col. plates, 8vo., Lond., 1874.

Part writer of Joseph Toynbee's The diseases of the ear; their nature, diagnosis, and treatment. With a supplement by James Hinton. Illus., 8vo., Lond., 1868.

Also Translator of (1) von Troltsch's The surgical diseases of the ear. New Syd. Soc., 8vo., Lond., 1874.

(2) Helmholtz's The mechanism of the ossicles and the membrana tympani. New Syd. Soc., 8vo., Lond., 1874.

THOMAS HODGKIN (1798—1866).

M.D. Edin., 1823; M.R.C.P., 1825;

In 1836 offered Fellowship of R.C.P., but declined it; Curator of Museum and Demonstrator of Morbid Anatomy, Guy's Hospital, 1825.

Dr. Hodgkin declined the Fellowship of the Royal College of Physicians, refusing, as did his friends Sir James Clark and Dr. Arnott, to accept an honour which he thought involved an invidious distinction.

"It will be my constant aim—whether I may be fortunate enough to reach the mark or not—to co-operate with those who are strenuously endeavouring to render the School of Guy's Hospital the first medical school in the kingdom."—Dr. Hodgkin.

"Everyone who cares to look upon Guy's as his Alma Mater cannot but feel a debt of gratitude to Hodgkin for the important share he took in endeavouring to constitute it a scientific school of medicine. It may be safely asserted that there has been no writer on pathology in this country before his time who had grasped the subject in so philosophic a spirit, for although Baillie, Hooper, and others had already written good descriptions of morbid appearances, Hodgkin was the first to follow in the footsteps of the great Bichat by discussing the diseases and changes according to the various tissues of the body."—Sir Samuel Wilks, Bart., F.R.S., Biographical History of Guy's Hospital.

De absorbendi functione. 8vo., Edin., 1823.

Thesis for M.D. Edin. A. I.—"The Physical Society of Guy's Hospital, with the author's best wishes."

Lectures on the morbid anatomy of the serous and mucous membranes. In two volumes, 8vo., Lond., 1836—40: Vol. I. On the serous membranes; and as appended subjects, parasitical animals, malignant adventitious structures, and the indications afforded by colour. Vol. II., part i. On the mucous membranes.

A. I.—*To Dr. Hughes, with the author's kind regards.*

"In 1836 Hodgkin's 'Lectures on the morbid anatomy of the serous and mucous membranes' was published in two volumes, and it established his reputation as a member of the distinguished school of morbid anatomists connected with Guy's Hospital."—Dr. Norman Moore, Dict. Nat. Biog.

The means of promoting and preserving health. 2nd edit., with additions. 8vo., Lond., 1841.

A. I.—*James Mash, with the author's kind regards.*

Joint Translator, etc., of W. F. Edward's Influence of physical agents on life. Translated from the French by Dr. Hodgkin and Dr. Fisher. To which are added in the appendix, some observations on electricity by Dr. Edwards, M. Pouillet, and Luke Howard, F.R.S.; on absorption and the uses of the spleen by Dr. Hodgkin; on the microscopic characters of the animal tissues and fluids by J. J. Lister, F.R.S., and Dr. Hodgkin; and some notes to the work of Dr. Edwards. 8vo., Lond., 1832.

See also Guy's Hospital Museum.—Catalogue of the preparations, etc., 1829.

The following book, which contains a brief account of two visits of Sir Moses Montefiore to Dr. Hodgkin's grave at Jaffa, is also in the library.

M. Auerbach and S. Salant's An open letter addressed to Sir Moses Montefiore, Bart., on the day of his arrival in the holy city of Jerusalem . . together with a narrative of a forty days' sojourn in the Holy Land. 2nd edit., 8vo., Lond., 1877.

A. I. on title-page—"To Dr. Samuel Wilks, M.D., etc., etc., etc., with Sir Moses Montefiore's best regards."

A. I. by Sir Samuel Wilks: "This book was given to me by Sir Moses Montefiore on account of the great interest I had taken in Dr. Hodgkin, and which was fully set forth in the letter which accompanied it.—Samuel Wilks.

"I present this book given to me by Sir Moses Montefiore, to the Library of Guy's Hospital as a memento of Dr. Hodgkin, and whose name appears in it at p. 60 and 68.

"It should be placed with the books already in the Library, as it will form a kind of appendix describing the latter part of his life, whilst those written by himself are associated with Guy's and give a good history of his work when he was Curator of the Museum and Lecturer on Pathological Anatomy.

"These are the two volumes—entitled the 'Anatomical Museum at Guy's Hospital by Dr. Hodgkin,' 1829, and two volumes entitled 'Hodg-

kin's Lectures on the Morbid Anatomy of the Serous and Mucous Membranes,' in two volumes, 1836; vol. i. On the Serous Membranes, vol. ii. On the Mucous Membranes.

Hampstead, **Samuel Wilks.**
Jan. 7th, 1910."

This book contains photographs of Dr. Hodgkin and Sir Moses Montefiore pasted on inside cover and fly leaf.

WILLIAM TIFFIN ILIFF, Junior,
M.D. Lond., 1856.

Book of cuttings of articles contributed to medical journals, 1845—53. Folio.

WALTER HAMILTON ACLAND JACOBSON,
M.A., M.B., M.Ch. Oxon.; F.R.C.S. Eng.;
Assistant Surgeon to Guy's 1876—1901; Surgeon to Guy's, 1901—1905.

The operations of surgery, intended especially for the use of those recently appointed on a hospital staff. Illus., 8vo., Lond., 1889.

Ditto, 2nd. edit., 1891.

Ditto, 3rd edit., 1897.

With F. J. Steward. 4th edit., 2 vols., 1902.

With R. P. Rowlands. 5th edit., 2 vols., 1907.

The diseases of the male organs of generation. Illus., 8vo., Lond., 1893.

Also editor of 2nd, 3rd, 4th, and 5th editions of Hilton's "Lectures on Rest and Pain."

Old Guy's most famous son.

JOHN KEATS (1795—1821),
Poet;
Student at Guy's Hospital, 1815—17; L.S.A., 1816.

Oh, weep for Adonais—he is dead!
Like a pale flower by some sad maiden cherish'd
And fed with true-love tears, instead of dew.
The soul of Adonais, like a star,
Beacons from the abode where the Eternal are.—
Percy Bysshe Shelley.

"There is a noise
Among immortals when a God gives sign,
With hushing finger, how he means to load
His tongue with the full weight of utterless thought,
With thunder, and with music, and with pomp."

"By Keats's soul, the man who never stepped
In gradual progress like another man,
But, turning grandly on his central self,
Ensphered himself in twenty perfect years
And died, not young,—(the life of a long life,
Distilled to a mere drop, falling like a tear
Upon the world's cold cheek to make it burn
For ever)."
Elizabeth Barrett Browning: *Aurora Leigh.*

O sweetest lips since those of Mitylene!
O poet-painter of our English Land!
Thy name was writ in water—it shall stand:
And tears like mine will keep thy memory green,
As Isabella did her Basil-tree.
Oscar Wilde, *The Grave of Keats.*

He dwelt with the bright gods of elder time,
On earth and in their cloudy haunts above,
He loved them; and in recompense sublime,
The gods, alas! gave him their fatal love.
William Watson.

Meek child of earth! thou wilt not shame
The sweet, dead poet's holy name;
The God of music gave thee birth,
Called from the crimson-spotted earth,
Where, sobbing his young life away,
His own fair Hyacinthus lay.
Oliver Wendell Holmes, *After a Lecture on Keats.*

The complete poetical works of John Keats. Edited with an introduction and textual notes by H. Buxton Forman, C.B. Oxford edition. Port., 8vo., Lond., etc., 1910.

CHARLES ASTON KEY (1793—1849).

F.R.C.S., 1845; F.R.S.;

Surgeon to Guy's, 1824—49; Surgeon to Prince Albert, 1847.

"To be seen to perfection you must follow him to the Hospital. Napoleon's eye was never lighted with purer ambitious pleasure when, at the head of a brilliant cortége, he dashed on his gallant charger into the centre of the square of the Tuileries, to review his devoted veterans' after some glorious campaign, than Key when he steps along the wards with his head erect, with his numerous suite or staff of admiring students. He marches along as if he felt himself every inch a king. There is an air of conceit, a conviction of self-importance, an arrogant pretension of optimism which would be regarded by his equals as an assumption,—we will call it usurpation,—but is regarded and recognised as his right by his subjects. Every look is here law, every diagnosis infallibility, every prognosis life or death. Doubt is treason, scepticism infidelity. If an error now and then occurs, it is one of nature's freaks. She departed from one of her fixed laws; she might be wrong, but her interpreter, her prophet, Key, never. Every word he utters is manna, molten gold. Observations the most trivial become aphorisms. He is, indeed, the epicycle of his own uncle; they revolve round like satellites around the sun."—***Medical Times***, quoted in Wilks and Bettany's Biographical History of Guy's Hospital.

A memoir on the advantages and practicability of dividing the stricture in strangulated hernia on the outside of the sac. Col. plates, 8vo., Lond., 1833.

A. I. *From the author to T. W. King, Esq.*

See also Sir Astley Cooper's Anatomy and surgical treatment of abdominal hernia. 2nd edition, edited by C. Aston Key, 1827.

WALTER AUBREY KIDD,

M.D., B.S. Lond., F.R.S. Edin.

Use inheritance, illustrated by the direction of hair on the bodies of animals. Illus., 8vo., Lond., 1901.

The direction of hair in animals and man. Illus., 8vo., Lond., 1903.

Sir WILLIAM ARBUTHNOT LANE, Bart.,
M.S., M.B. Lond.; F.R.C.S. Eng.;
Surgeon to Guy's.

Cleft palate. Treatment of simple fractures by operation. Diseases of joints. Antrectomy. Hernia, etc. Illus., 8vo., Lond., 1897.

Cleft palate and adenoids. Treatment of simple fractures by operation. Diseases of joints. Operative treatment of cancer. Acquired deformities. Antrectomy. Hernia, etc. 2nd edition, illus., 8vo., Lond., 1900.

Cleft palate and hare lip. 2nd edition, illus., 4to., Lond., 1908.

The operative treatment of fractures. Illus., 4to., Lond., 1905.

The operative treatment of chronic constipation. Illus., 4to., Lond., 1909.

See also R. E. Carrington's A manual of dissections of the human body. 2nd edition, revised and enlarged by W. A. Lane, 1888.

JOHN BERESFORD LEATHES,
M.A., M.B., B.Ch. Oxon.; F.R.C.S. Eng.; F.R.S. Canada;
F.R.S.

Beaney Scholar, Guy's Hospital, 1897—99; Professor of Pathological Chemistry, University of Toronto, 1909.

Problems in animal metabolism. 8vo., Lond., 1906.

See also Guy's Hospital—Beaney Scholars' Reprints.

JOHN CHARLES WEAVER LEVER (1811—),
M.D. Giessen;

President of the Hunterian Society; Physician-Accoucheur and Lecturer on Midwifery, Guy's Hospital.

A practical treatise on organic diseases of the uterus: being the prize essay to which the Medical Society of London awarded the Fothergillian gold medal for 1843. 8vo., Lond., 1843.

RICHARD CLEMENT LUCAS,
M.B., B.S. Lond.; F.R.C.S. Eng.;
Vice-President of the Royal College of Surgeons; President of the Hunterian Society; Surgeon to Guy's Hospital, 1888—1906.

The Bradshaw Lecture on some points in heredity. Delivered before the Royal College of Surgeons of England on December 6th, 1911. Illus., 8vo., Lond., 1912.

ALEXANDER JOHN GASPARD MARCET (1770—1822),
M.D. Edin., 1797; F.R.S., 1815;
Physician to Guy's Hospital, 1804—19; Lecturer on Chemistry at Guy's Hospital, 1807—09.

An essay on the chemical history and medical treatment of calculous disorders. Col. plates, 8vo., Lond., 1817.

See also Babington, Marcet, and Allen's Syllabus of a course of chemical lectures read at Guy's Hospital, 1816.

CHARLES FREDERICK MAUNDER,
F.R.C.S. Eng., 1857;
Lettsomian Lecturer, 1875; Surgeon and Lecturer on Clinical Surgery, London Hospital.

Tumour of lateral portions of the lower jaw removed without external wound. Illus., 8vo., Lond., 1874.

Surgery of the arteries. Lettsomian Lectures of the Medical Society of London, 1875. Illus., 8vo., Lond., 1875.

Translator of Philippe Ricord's Lectures on chancre. Published by M. Fournier, with notes and cases; and translated from the French by C. F. Maunder. With remarks on perineal section of stricture of the urethra by the translator. 8vo., Lond., 1859.

A. I.—*To the Students of Guy's Hospital, with the Translator's best wishes.*

FREDERICK DENISON MAURICE (1805—1872),
M.A. Oxon.;

Chaplain at Guy's Hospital, 1836—46; Founder of the Working Men's College, and Queen's College for Women, London; Professor of Moral Philosophy, Cambridge University, 1866.

"I like Guy's increasingly."—F. D. Maurice, letter to Rev. R. C. Trench, 1836.

"My whole married life is bound up with Guy's, and there is nothing about it which is not in some way associated with her."—F. D. Maurice in 1846.

The life of Frederick Denison Maurice, chiefly told in his own letters. Edited by his son, Frederick Maurice. 2 vols., ports., 8vo., Lond., 1884.

A. I.—*Presented to the Students' Library of Guy's Hospital by one of the Governors, whose interest in medical students dates from the chaplaincy of F. D. M.*

ALFRED ERNEST MAYLARD,
M.B., B.S. Lond.;
Surgeon to Victoria Infirmary, Glasgow.

The students' handbook of the surgery of the alimentary canal, being an abridged and amended edition of the author's treatise on the same subject. Illus., 8vo., Lond., 1900.

EDWARD PIGOTT MINETT,
M.D. Brux., 1907; D.P.H. Camb., 1907; D.T.M. and Hy., 1909;
Assistant Government Bacteriologist, British Guiana.

Differential diagnosis of bacteria and practical bacteriology. Sm. 8vo., Lond., 1909.

ROBERT OSWALD MOON,
M.A., M.D., B.Ch. Oxon.; F.R.C.P. Lond.

The relation of medicine to philosophy. 8vo., Lond., etc., 1909.

JOHN MORGAN (1797—1847),
Surgeon to Guy's Hospital, 1824.

Lectures on diseases of the eye. 2nd edition, carefully revised and enlarged, with notes by John F. France. Col. plates, 8vo., Lond., 1848.

Sir HENRY MORRIS, Baronet,
M.A., M.B. Lond.; F.R.C.S. Eng.;
President of the Royal College of Surgeons, 1906—09; President of the Royal Society of Medicine, 1910—12; Consulting Surgeon and Emeritus Lecturer on Surgery, Middlesex Hospital; Cavendish Lecturer, 1893; Hunterian Lecturer, 1899; Bradshaw Lecturer, 1903; Sir Mitchell Banks Memorial Lecturer, 1908; Hunterian Orator, 1909.

The anatomy of the joints of man. Col. plates, 8vo., Lond., 1879.

Surgical diseases of the kidney. Illus., 8vo., Lond., etc., 1885.

A treatise on human anatomy by various authors. Edited by Henry Morris. Illus., la. 8vo., Lond., 1893.

Morris's human anatomy: a complete systematic treatise by English and American authors. Edited by Henry Morris and J. Playfair McMurrich. 4th edition, revised and enlarged. Col. and other illus., la. 8vo., Lond., 1907.

Injuries and diseases of the genital and urinary organs. Illus., 8vo., Lond., etc., 1895.

Surgical diseases of the kidney and ureter, including injuries, malformations, and misplacements, 2 vols. Illus., 8vo., Lond., etc., 1901.

WALTER MOXON (1836—1886),
M.D. Lond., 1864; F.R.C.P. Lond., 1868;
Physician to Guy's Hospital, 1873; Lecturer on Medicine at Guy's Hospital, 1872; Croonian Lecturer, 1881.

Pilocereus senilis, and other papers. 8vo., Lond., 1887.

See also Wilks and Moxon's Lectures on pathological anatomy. 2nd edit., 1875.

Sir SHIRLEY FORSTER MURPHY, Kt.,
F.R.C.S. Eng.;

Consulting Medical Officer of Health, Administrative County of London: Ex-President of the Epidemiological Society; Ex-President of Society of Medical Officers of Health.

See Stevenson and Murphy's Treatise on hygiene and public health. 3 vols., 1892—94.

Museum—*see* Guy's Hospital Museum.

WILLIAM ODLING,
M.B. Lond., 1851; F.R.C.P. Lond., 1859; F.R.S.;

Professor of Practical Chemistry and of Natural Philosophy at Guy's Hospital; Fullerian Professor of Chemistry, Royal Institute; Waynflete Professor of Chemistry, Oxford University, since 1872.

A course of practical chemistry, arranged for the use of medical students. 8vo., Lond., 1854.

Ditto, 5th edit., 1876.

A manual of chemistry, descriptive and theoretical, part I. 8vo., Lond., 1861.

Outlines of chemistry, or brief notes of chemical facts. 8vo., Lond., 1870.

Miss M. N. OXFORD,
Sometime Sister at Guy's Hospital.

A handbook of nursing. 8vo., Lond., 1900.

ARTHUR WILLIAM ORMOND,
F.R.C.S. Eng.;
Ophthalmic Surgeon to Guy's Hospital.

See Higgens's Manual of ophthalmic practice. 2nd edition, revised and edited by Arthur W. Ormond, 1903.

FREDERICK WILLIAM PAVY (1829—1911),

M.D. Lond., 1853; F.R.C.P. Lond., 1860; Censor, 1883—84; Senior Censor, 1891—92; F.R.S.; LL.D. Glas., 1888; Lecturer on Comparative Anatomy, Guy's Hospital; Lecturer on the Principles and Practice of Medicine, Guy's Hospital; Physician to Guy's Hospital, 1871—90; Lettsomian Lecturer, 1859; Goulstonian Lecturer, R.C.P., 1862 and 1863; Croonian Lecturer, 1878 and 1894; Harveian Orator, 1886; President of the Pathological Society, 1893—94; President of the Royal Medical and Chirurgical Society, 1900—01.

What was exactly the scientific position of this distinguished man? In the first place, his labours form a connecting link between the past and the present. He was the last survivor of a line of distinguished physician-chemists who in this country during the earlier and middle years of the last century, did much to lay the foundations and advance the study of metabolic disorders. He was a worthy successor of Prout, Alexander Marcet, Bence Jones, and Golding Bird, and a contemporary of William Marcet, Roberts, Garrod, and Thudichum; but he also ranks as a pioneer among the chemical pathologists of the modern school, who, in the light of a far more ample chemical science, and by methods far more exact and adequate, are carrying on, in Europe and America, the work which those men initiated, and are expanding it in all directions. A pupil of Claude Bernard, Pavy realised to the fullest extent the truth that all advances in the study of disease must rest upon investigations into the normal processes of the body, and his memory is reverenced by physiologists, pathologists, and physicians alike. We associate with his name the recognition of postural albuminuria, and the theory that the living gastric wall owes its immunity from auto-digestion to the alkalinity of the blood; but it is chiefly in connexion with the study of carbohydrate metabolism that his name and work will be remembered. He was the founder of the modern study of diabetes. His labours in that field covered a period of no less than half a century. His first public delivery on the subject dates from 1860, and he dealt with it with undiminished vigour in a course of lectures delivered before the Royal College of Physicians of London in 1908. Thus, even in old age he showed that the keenness of his interest in the subject which he had made his own suffered no abatement.—*The Lancet*, 1911, ii., pp. 979—10.

Pavy's work is, perhaps, the most remarkable instance of steady persistence in one relatively limited line of inquiry, and illustrates not only the extraordinary tenacity of purpose of the inquirer, but also, and in no less degree the evasiveness and the almost complete insolubility of some problems of life and disease. Pavy devoted little short of 60 years of his life to this particular problem, for he was working with Bernard in the summer of 1853, and he was still continuing his laboratory experiments up to the time of the autumn holiday just preceding his death in

1911. There was remarkable sagacity, not devoid of satire, in Gull's comment upon Pavy. What sin, said he, has Pavy committed, or his fathers before him, that he should be condemned to spend his whole life seeking for the cure of an incurable disease? And Pavy, still researching, survived Gull by some one-and-twenty years.—Dr. Frederick Taylor: In Memoriam: Frederick William Pavy, Guy's Hospital Reports, 1912, lxvi., 9—10.

The recollection of Dr. Pavy spending time, thought, and money in trying to unravel the problems of physiology and pathology up till his death, happily postponed till the age of 82, will always be a help to those who feel daunted and discouraged by the immensity and complexity of the riddles they are attempting to solve. None of us can tell what will be the fate of his most cherished views, but this at least we know, that for all time he will be an outstanding ornament to British medicine, for it is doubtful whether the physician ever lived who sought truth for so long or so whole-heartedly as did Dr. Pavy.—Dr. W. Hale White, *British Medical Journal*, 1911, ii., 778.

Researches on the nature and treatment of diabetes. 8vo., Lond., 1862.

Ditto, 2nd edition, revised and enlarged. 8vo., Lond., 1869.

A treatise on the function of digestion; its disorders, and their treatment. 8vo., Lond., 1867.

A treatise on food and dietetics, physiologically and therapeutically considered. 8vo., Lond., 1874.

The Harveian Oration, delivered at the Royal College of Physicians, October 18th, 1886. 8vo., Lond., 1886.

The physiology of the carbohydrates; their application as food and relation to diabetes. Illus., 8vo., Lond., 1894.

The physiology of the carbohydrates. An epicriticism. 8vo., Lond., 1895.

On carbohydrate metabolism, with an appendix on the assimilation of carbohydrate into proteid and fat, followed by the fundamental principles, and the treatment, of diabetes dialectically discussed. Illus., 8vo., Lond., 1906.

MARCUS SEYMOUR PEMBREY,
M.A., M.D., B.Ch. Oxon.;

Radcliffe Travelling Fellow, Oxford University, 1890; Lecturer in Physiology, Guy's Hospital; Examiner in Physiology, University of London.

Practical physiology. Edited by M. S. Pembrey, 3rd edition. Illus., 8vo., Lond., 1910.

Sir EDWIN COOPER PERRY, Kt.,
M.A., M.D. Camb.; F.R.C.P. Lond.;

Superintendent of Guy's Hospital; Physician, and Physician to Skin Department, Guy's Hospital; Lecturer on Medicine and Dermatology, Guy's Hospital Medical School; Dean of the Medical School, Guy's Hospital, 1888—93.

See Guy's Hospital Museum, Descriptive Catalogue of the pathological specimens contained in the Museum of Guy's Hospital. 3rd edit., 1894—1910.

Pharmacopœias.—*See* Guy's Hospital Pharmacopœias.

Physical Society.—*See* Guy's Hospital—Physical Society.

Physiological Laboratory, Collected papers.—*See* Guy's Hospital—Physiological Laboratory, etc.

Physiological Society's Papers (MS.).—*See* Guy's Hospital—Physiological Society's Papers.

ALFRED POLAND (1821—1872),
F.R.C.S. Eng., 1847;

Surgeon to Guy's Hospital, 1861—72; Ophthalmic Surgeon and Lecturer on Ophthalmic Surgery at Guy's Hospital; Fothergillian Medallist, 1853; Jacksonian Prizeman, 1857.

Tables used in the course of lectures on the theory and practice of surgery, delivered at Guy's Hospital, during the session 1853—54. 4to., Lond., 1854.

JOHN POLAND,
F.R.C.S. Eng.;

Late Senior Surgeon to the City Orthopædic Hospital; President of the Hunterian Society, 1906; Master of the Worshipful Company of Skinners, 1902—03.

Traumatic separation of the epiphyses. Illus., la. 8vo., Lond., 1898.

Skiagraphic atlas showing the development of the bones of the wrist and hand. Illus., la. 8vo., Lond., 1898.

CHARLES EDWARD POLLOCK,

With L. W. Harrison. Gonococcal infections. 8vo., Lond., 1912.

HENRY F. A. PRATT,

M.D. St. Andrews, 1854.

The genealogy of creation, newly translated from the unpointed Hebrew text of the book of Genesis; showing the general scientific accuracy of the cosmogony of Moses and the philosophy of creation. 8vo., Lond., 1861.

Pupils' Physical Society. Transactions (MS.).—*See* Guy's Hospital—Pupils' Physical Society, etc.

PHILIP HENRY PYE-SMITH,

B.A., M.D. Lond.; F.R.C.P. Lond.; F.R.S.; Physician to Guy's Hospital, 1883; Lumleian Lecturer, R.C.P., 1892; Harveian Orator, 1893; Vice-Chancellor of the University of London, 1903—05; President of the Pathological Society, 1906—07.

Syllabus of a course of lectures on physiology, delivered at Guy's Hospital. Illus., 8vo., Lond., 1885.

An introduction to the study of diseases of the skin. 8vo., Lond., 1893.

The Lumleian Lectures on certain points in the ætiology of disease, delivered before the Royal College of Physicians, 1892. To which is added the Harveian Oration, delivered before the College in 1893. With a biographical notice of Harvey and an appendix of statistical tables. 8vo., Lond., 1895.

See also C. H. Fagge and P. H. Pye-Smith's The principles and practice of medicine. 2nd edit., 1888; 3rd edit., 1891; 4th edit., 1901—02.

See also Guy's Hospital Museum—Catalogue of the preparations of comparative anatomy in the Museum of Guy's Hospital. By P. H. Pye-Smith, 1874.

THOMAS RADFORD (1793—1881).

M.D. Heidelberg, 1839; F.R.C.P. Edin., 1839; F.R.C.S. Eng., 1852.

Observations on the Cæsarian section and on other obstetric operations. With an appendix of cases. 8vo., Manchester, 1865.

Cases of laceration of the uterus, with remarks. (From volume 8 of the Trans. of the Obstetrical Society of London.) Plates, 8vo., Lond., 1867.

ALFRED THEODORE RAKE.

M.B., B.S. Lond.; F.R.C.S. Eng.

Translator of C. Schimmelbusch's The aseptic treatment of wounds. With a preface by Prof. Bergmann. Translated from the 2nd German edition by A. T. Rake. Illus., 8vo., Lond., 1894.

GEORGE OWEN REES (1813—1889).

M.D. Glas., 1836; F.R.S., 1843; F.R.C.P., 1844; Goulstonian Lecturer, R.C.P., 1845; First Lettsomian Lecturer, 1850—51; Physician and Lecturer on the Practice of Medicine, Guy's Hospital, 1856—73; Croonian Lecturer, R.C.P., 1856—58; Censor, R.C.P., 1859, and Senior Censor, 1863; Harveian Orator, R.C.P., 1869.

On the analysis of the blood and urine, in health and disease. With directions for the analysis of urinary calculi. 8vo., Lond., 1836.

Ditto, and on the treatment of urinary diseases. 2nd edition. 8vo., Lond., 1845.

He deserves to be known in medical history as one of the first men to turn his attention to the chemistry of the urine. — Surgeon-Captain W. W. Webb, Dict. Nat. Biog.

On the nature and treatment of diseases of the kidney connected with albuminous urine (Morbus Brightii). 8vo., Lond., 1850.

On calculous disease and its consequences: being the Croonian Lectures for the year 1856. 8vo., Lond., 1856.

A. I.—*Library, Guy's Hospital, from the Author.*

GEORGE ERNEST RICHMOND,
B.A., M.D., B.S., B.Sc. Lond.; D.P.H. Camb.

An essay upon disease, its cause and prevention. 8vo., Lond., 1907.

BENJAMIN RIDGE,
F.R.C.S. Eng., 1854.

Glossology: or the additional means of diagnosis of disease to be derived from indications and appearances of the tongue. Read before the Senior Physical Society of Guy's Hospital, 4th November, 1843. Plates, 8vo., Lond., 1844.

A. I.—*The Library of Guy's Hospital. From the Author.*

GEORGE ROBINSON,
L.S.A., 1841; M.D. St. Andrew, 1844.

An inquiry into the nature and pathology of granular disease of the kidney, and its mode of action in producing albuminous urine. 8vo., Lond., 1842.

ROBERT PUGH ROWLANDS,
M.S. Lond.; F.R.C.S. Eng.;

Assistant Surgeon and Surgeon-in-charge of Orthopædic Department, Guy's Hospital.

See Jacobson and Rowlands' The operations of surgery. 5th edit., 2 vols., 1907.

JAMES RYMER,

Note book for dental students (dental anatomy and physiology). Sm. 8vo., Lond., 1888.

ALFRED SALTER,
M.D., B.S. Lond.; D.P.H.; R.C.P.S. Eng.;

See Guy's Hospital—Gull Students' Reprints.

SAMUEL JAMES AUGUSTUS SALTER (1825—1897),
M.B. Lond., 1849; L.S.A., 1847; F.R.S.:
Dental Surgeon to Guy's Hospital.

Dental pathology and surgery. Illus., 8vo., Lond., 1874.

WILLIAM SAUNDERS (1743—1817).
M.D. Edin., 1765; F.R.C.P. Lond., 1790; F.R.S., 1793; Censor, R.C.P., 1791, 1798, 1805, and 1813; Physician to Guy's Hospital, 1770—1802; Goulstonian Lecturer, R.C.P., 1792; Harveian Orator, R.C.P., 1796; Physician to George, Prince Regent, 1807; First President of the Medical and Chirurgical Society, 1805.

A treatise on the chemical history and medical powers of some of the most celebrated mineral waters; with practical remarks on the aqueous regimen. To which are added observations on the use of cold and warm bathing. 8vo., Lond., 1800.

Ditto, 2nd edition, enlarged, 1805.

An important matter in which Dr. Saunders was concerned after leaving Guy's was the foundation of the Royal Medical and Chirurgical Society. On May 22nd, 1805, an inaugural meeting was held at Freemason's Tavern, Dr. Saunders in the chair, at which it was determined to establish a society comprehending the several branches of the medical profession in London, to receive professional communications, and to form a library. The first meeting of the society, when constituted, was held in Gray's Inn, in December, 1805, but it was not till 1809 that the first volume of its well-known "Transactions" was published. Among the members of the first council were: Dr. Saunders, President; Charles Rochement Aikin, Secretary; Dr. William Babington, Vice-President; Astley Cooper, Vice-President; Dr. James Curry; Dr. Alexander Marcet, Foreign Secretary; with others, including many of the best names in medicine and surgery at that time. The names quoted show the influential part Guy's men played in establishing the society. In 1812 the society became "Royal" by obtaining a charter from the Prince Regent.*—Wilks and Bettany's Biographical History of Guy's Hospital.

It is interesting to note that Guy's Hospital continued to supply its full quota of men as Presidents of the Royal Medical and Chirurgical Society. Of the fifty Presidents who occupied the chair of this Society from its foundation in 1805 to 1907, when, with its amalgamation with a number of other societies, it became the Royal Society of Medicine, nine were Guy's men. These were: W. Saunders, 1805; W. Babington, 1817; Astley Cooper, 1819; John Cooke, 1821; Richard Bright, 1837; Thomas Addison, 1849; B. G. Babington, 1861; Thomas Bryant, 1898; and F. W. Pavy, 1900. This number was not exceeded by any other hospital, the only one to equal it being St. Bartholomew's, who also provided nine.

* This statement is incorrect. The petition for a charter was refused in 1812. The charter was granted in 1834 by King William IV.

Sir GEORGE HENRY SAVAGE, Kt.,

M.D. Lond., 1867; L.S.A., 1872; F.R.C.P. Lond., 1885; Consulting Physician for and Lecturer on Mental Diseases, Guy's Hospital; Examiner in Mental Pathology, University of London; President of the Neurological Society, 1897; First President of the Section of Psychiatry of the Royal Society of Medicine, 1912; Lumleian Lecturer, R.C.P., 1907; Harveian Orator, R.C.P., 1909; Past President of the Medico-Psychological Association; Late Senior Physician and Superintendent of Bethlem Royal Hospital; Late Editor of "The Journal of Mental Science."

Insanity and allied neuroses: practical and clinical. Illus., 8vo., Lond., etc., 1884.

With E. Goodall. Ditto, new and enlarged edition. 1907.

The Harveian Oration on experimental psychology and hypnotism. 8vo., Lond., 1909.

J. SEFTON SEWILL.

Joint Editor of Henry Sewill's Dental surgery: including special anatomy and pathology. 4th edition. Edited by W. J. England and J. Sefton Sewill. Illus., 8vo., Lond., 1901.

SAMUEL SHARP (1700—1778).

Freeman of Barber-Surgeons' Company, 1731; Obtained Diploma, 1732; Surgeon to Guy's Hospital, 1733—57; F.R.S. and Member of Paris Royal Society, 1749.

Samuel Sharp was the first great surgeon appointed to Guy's.—Wilks and Bettany's Biographical History of Guy's Hospital.

It must be noted, to the credit of Guy's, that one of the earliest public courses of anatomy and surgery was given by Samuel Sharp.—Id. ibid.

A treatise on the operations of surgery, with a description and representation of the instruments used in performing them. To which is prefix'd an introduction on the nature and treatment of wounds, abscesses, and ulcers. Plates, 8vo., Lond., 1739.

A critical enquiry into the present state of surgery. 8vo., Lond., 1750.

Ditto, 4th edition, 1761.

LAURISTON ELGIE SHAW,
M.D. Lond.; F.R.C.P. Lond.;

Dean of the Medical School, Guy's Hospital, 1893—1901; Honorary Wills Librarian, 1903—09; Physician and Lecturer on Medicine, Guy's Hospital.

See Guy's Hospital Museum. Descriptive catalogue of the pathological specimens contained in the Museum of Guy's Hospital. 3rd edition, 3 vols., 1894—1910.

EDWARD WARREN HINE SHENTON,
Senior Surgical Radiographer to Guy's Hospital.

Disease in bone, and its detection by the X-rays. Illus., 8vo., Lond., 1911.

Sir JAMES EDWARD SMITH, Kt. (1759—1828),
Lecturer on Botany at Guy's Hospital; Founder of the Linnæan Society, 1788.

Lectures on botany, delivered in the Theatre, Guy's Hospital (MS.), 1797. 4to.

A. I.—*Martin Tupper's, Guy's Square, London.*

Flora Britannica, 3 vols. 8vo., Lond., 1800—04.

An introduction to physiological and systematical botany. 8vo., Lond., 1807.

Lachesis Lapponica, or a tour in Lapland, now first published from the original manuscript journal of the celebrated Linnæus by James Edward Smith, M.D., F.R.S. 2 vols., 8vo., Lond., 1811.

A grammar of botany, illustrative of artificial, as well as natural classification, with an explanation of Jussieu's system. Col. plates, 8vo., Lond., 1821.

EDMUND IVENS SPRIGGS,
M.D. Lond., 1898; F.R.C.P. Lond., 1905;
Senior Assistant Physician, Lecturer on Pharmacology, and Dean of the Medical School, St. George's Hospital; Gull and Beaney Research Student, Guy's Hospital; Oliver-Sharpey Lecturer, R.C.P., 1906.

The Oliver-Sharpey Lectures on the bearing of metabolism experiments upon the treatment of some diseases. Delivered before the Royal College of Physicians of London on April 3rd and 5th, 1906. 8vo., Lond., 1906.

See also Guy's Hospital—Gull Students' Reprints, vol. I.

ERNEST HENRY STARLING,
M.D. Lond., 1890, B.S., 1888; M.D. (hon. causâ) Breslau; F.R.C.P. Lond., 1897; F.R.S., 1899;
Jodrell Professor of Physiology, University College, London; Demonstrator of Physiology, Guy's Hospital, 1889; Joint Lecturer on Physiology, Guy's Hospital, 1890—99; Arris and Gale Lecturer, R.C.S., 1894, 1896, and 1897; Croonian Lecturer, Royal Society, 1904; Croonian Lecturer, R.C.P., 1905; Baly Medallist, 1907; Herter Lecturer, New York, 1908.

Elements of human physiology. 5th edition. Illus., 8vo., Lond., 1902.

Ditto, 6th edit., 1904.

Ditto, 7th edit., 1905.

Ditto, 8th edit., 1908.

Principles of human physiology. Illus., 8vo., Lond., 1912.

Mercers' Company Lectures on recent advances in the physiology of digestion, delivered in the Michaelmas Term, 1905, in the Physiological Department of University College, London. 2nd impression. Illus., 8vo., Lond., 1906.

JOHN CHARLES STEELE (1821–1892),
M.D. Glas., 1848;
Superintendent of Guy's Hospital, 1853–92; Howard Medallist, Statistical Society, 1876.

See Guy's Hospital—Statistical tables of the patients treated in Guy's Hospital, 1854—78.

Dr. Steele was the first at Guy's Hospital to bear the title of Superintendent, his predecessor, Mr. Browell, being called a Steward.

THOMAS GEORGE STEVENS.
M.D., B.S. Lond.; F.R.C.S. Eng.

Obstetric Surgeon, St. Mary's Hospital; Surgeon (Gynæcol.) Hospital for Women, Soho Square; Physician, In-Patients, Queen Charlotte's Lying-In Hospital; Examiner, Central Midwives Board; Ex-President Æsculapian Society.

Diseases of Women. Illus., 8vo., Lond., (1912).

Sir THOMAS STEVENSON, Kt. (1838—1908),
M.D. Lond., 1864; F.R.C.P. Lond., 1871;

Lecturer on Chemistry at Guy's Hospital, 1870—98; Lecturer in Forensic Medicine at Guy's Hospital, 1878—1908; Senior Scientific Analyst to the Home Office, 1881—1908; President of the Society of Medical Officers of Health; President of the Society of Public Analysts; President of the Institute of Chemistry.

For many years . . the acknowledged representative and head of the science of medical jurisprudence in this country, whilst his reputation abroad was equalled, if at all, by few and excelled by none. Stevenson has left his "footprints on the sands of time," and his name will go down to posterity with those of Orfila, Casper, Tardieu, and Brouardel.—*The Lancet.*

Spirit-gravities with tables. 8vo., Lond., 1880.

A treatise on alcohol, with tables of spirit-gravities. 2nd edit. 8vo., Lond., 1888.

With Sir Shirley Murphy. A treatise on hygiene and public health. 3 vols. Illus., la. 8vo., Lond., 1892—94.

See also A. S. Taylor's Manual of medical jurisprudence. 11th edition. Edited by Sir T. Stevenson, 1886. 12th edit., 1891.

FRANCIS JAMES STEWARD,
M.S., M.B. Lond.; F.R.C.S. Eng.;

Surgeon to Guy's Hospital, 1912; Examiner in Surgery, I.M.S.; Formerly Assistant Surgeon and Surgeon-in-charge of Throat Department, Guy's Hospital.

See Jacobson and Steward's Operations of Surgery. 4th edit., 2 vols., 1902.

GEORGE FREDERICK STILL,
M.A., M.D., B.C. Camb.; F.R.C.P. Lond.;
Murchison Scholar, R.C.P., Lond., 1894; Goulstonian Lecturer, R.C.P., 1902; Physician for Diseases of Children, and Professor of Diseases of Children, King's College Hospital.

Common disorders and diseases of childhood. 2nd edit. Illus., 8vo., Lond., 1912.

See also Goodhart and Still's The diseases of children, 6th edit., 1899: 8th edit., 1905: 9th edit., 1912.

FREDERICK OTTO STOHR,
M.B., B.Ch. Oxon., 1899;
Medical Officer of Geodetic Survey, N.E. Rhodesia, and Union Minière du Haut Katanga.

La maladie du sommeil au Katanga. Illus., 8vo., Lond., 1912.

ALFRED SWAINE TAYLOR (1806—1880),
L.S.A., 1828; M.D. St. Andrew (Hon.), 1852; F.R.C.P. Lond., 1853; F.R.S., 1845;
First Professor of Medical Jurisprudence at Guy's Hospital, 1831—77; Lecturer on Chemistry at Guy's Hospital, 1832—70; Editor of the "London Medical Gazette," 1844—51; Swiney Prizeman, 1859.

The greatest medical jurist whom this country has produced, and, perhaps, the greatest in Europe. Dr. Taylor did much to create the department of medico-juridical science in which he became so distinguished; for, when he first began to lecture on medical jurisprudence at Guy's Hospital, at the age of twenty-five, his was the first English course on this subject, and many leading members of the bar, and even judges, attended his teaching. . . Lacking, on the one hand, the high chemical attainments of Orfila, and, on the other, the extended antiquarian and scientific research of Caspar, he may yet be considered by anyone who fairly reviews the history of medical jurisprudence and the place of Alfred Swaine Taylor in its progress, to have contributed more than either to the establishment of this subject as a coherent whole; to have laid down the limits within which scientific data are available for the guidance and application of legal principle; and to have combined the whole mass of legal precedents, of judicial rulings, of anatomical and chemical data, into a code of instruction which stands quite unsurpassed in the literature of any country.—***British Medical Journal,*** June 12th. 1880.

The elements of medical jurisprudence. Vol. I. 8vo., Lond., 1836.

On poisons, in relation to medical jurisprudence and medicine. 8vo., Lond., 1848.

A. I.—*The Library of Guy's Hospital from the Author.*

Ditto, 2nd edit., 1859.

Ditto, 3rd edit., illus., 1875.

Medical jurisprudence. 5th edit., 8vo., Lond., 1854.

A manual of medical jurisprudence. 9th edit., 8vo., Lond., 1874.

Ditto, 11th edit., edited by Thomas Stevenson, 1886.

Ditto, 12th edit., edited by Thomas Stevenson, 1891.

The principles and practice of medical jurisprudence. 8vo., Lond., 1865.

Ditto, 4th edit., edited by T. Stevenson, 2 vols., 1894.

Ditto, 6th edit., edited, revised, and brought up to date by Fred. J. Smith, 2 vols., 1910.

Syllabus of a course of lectures on chemistry delivered at Guy's Hospital. 8vo., Lond., 1857.

Interleaved copy.

With W. T. Brande. Chemistry. 8vo., Lond., etc., 1863.

Ditto, 2nd American edition, thoroughly revised, la. 8vo., Philad., 1867.

Part author of Thomas Wicksteed's Preliminary report upon the sewage, drainage, and supply of water for the Borough of Leicester. And a report upon the analysis of the sewage water, and the water of the streams in the neighbourhood of Leicester by Arthur Aikin, Esq., and Dr. Alfred Swaine Taylor. 8vo., Lond., 1850.

Joint editor of Neil Arnott's Elements of physics or natural philosophy. 7th edition, edited by Alexander Bain and Alfred Swaine Taylor. Illus., 8vo., Lond., 1876.

See also Aikin and Taylor's Syllabus of a course of chemical lectures delivered at Guy's Hospital, 1839.

FREDERICK TAYLOR,

M.D. Lond., 1870; F.R.C.P. Lond., 1879;

Physician to Guy's Hospital, 1885—1907; Dean of the Medical School, Guy's Hospital, 1874—88; President of the Clinical Society, 1903—04; Examiner in Medicine, University of Cambridge; Harveian Orator, R.C.P., 1907.

A manual of the practice of medicine. 8vo., Lond., 1890.
Ditto, 2nd edit., 1891.
Ditto, 3rd edit., illus., 1893.
Ditto, 4th edit., illus., 1895.
Ditto, 5th edit., illus., 1898.
Ditto, 6th edit., illus., 1901.
Ditto, 7th edit., illus., 1904.
Ditto, 8th edit., illus., 1908.
Ditto, 9th edit., illus., 1911.

The need of research in medicine. The Harveian Oration delivered before the Royal College of Physicians of London on St. Luke's Day, 1907. 8vo., Lond., 1908.

THOMAS PRIDGIN TEALE (1801—1868),

F.R.C.S. Eng., 1843; M.D., T.C.D. (Hon. Causâ), 1867; F.R.S., 1862;

Surgeon to the Leeds General Infirmary, 1833—64.

A treatise on neuralgic diseases dependent upon irritation of the spinal marrow and ganglia of the sympathetic nerve. 8vo., Lond., 1829.

On amputation by a long and a short rectangular flap. Illus., 8vo., Lond., 1858.

ARTHUR RALPH THOMPSON,

M.B., Ch.M. Viet.; F.R.C.S. Eng.;

Surgeon-in-charge of Genito-Urinary Department, Guy's Hospital: Hunterian Professor, R.C.S., 1908.

See Fripp and Thompson's Human anatomy for art students, 1911.

ROBERT JOHN THORNTON (1768—1837),
M.D. Camb., 1797;
Lecturer on Botany at Guy's Hospital.

Facts decisive in favour of the cow-pock: including an account of the inoculation of the village of Lowther. 8vo., Lond., 1802. Bound up with Haygarth's Prevention of infectious fevers, 1801.

A new family herbal: or popular account of the natures and properties of the various plants used in medicine, diet, and the arts. The plants drawn from nature by Henderson, and engraved on wood by Thomas Bewick. Illus., 8vo., Lond., 1810.

A. I.—*Presented by Samuel Ashwell, M.D., to the Physical Society, Oct.*, 1837. With Dr. Ashwell's bookplate.

An easy introduction to the science of botany. Plates. 8vo., Lond., 1823.

MS. note on flyleaf: "This book contains a singular discussion as to whether it is decent to teach girls botany seeing that plants have sexual organs and functions."

BENJAMIN TRAVERS (1783—1858),
F.R.S., 1813;

Demonstrator of Anatomy at Guy's Hospital, 1809; Surgeon to the East India Company, 1809; Surgeon to the London Eye Infirmary, 1810; Surgeon to St. Thomas's Hospital, 1815; President of the Royal Medical and Chirurgical Society, 1827; President of the Hunterian Society, 1827; Hunterian Orator, 1838; President of the Royal College of Surgeons in 1847 and 1856—57; Surgeon Extraordinary to Queen Victoria; Surgeon in Ordinary to Prince Albert.

An inquiry into the process of nature in repairing injuries of the intestines, illustrating the treatment of penetrating wounds, and strangulated hernia. 8vo., Lond., etc., 1812.

A synopsis of the diseases of the eye, and their treatment: to which are prefixed a short anatomical description and a sketch of the physiology of that organ. Col. plates. 8vo., Lond., 1820.

Ditto, 2nd edit., 1821.

Ditto, 3rd edit., 1824.

An inquiry concerning that disturbed state of the vital functions usually denominated constitutional irritation. 8vo., Lond., 1826.

A further inquiry concerning constitutional irritation, and the pathology of the nervous system. 8vo., Lond., 1835.

See also Cooper and Travers' Surgical essays, 1818—19.

ALFRED HERBERT TUBBY,

M.S. Lond., 1890; F.R.C.S. Eng., 1887;

Surgeon, and in charge of Orthopædic Department, Lecturer on Clinical Surgery and Orthopædic Surgery, Westminster Hospital; Surgeon to the Royal National Orthopædic Hospital.

Deformities. A treatise on orthopædic surgery. Illus., 8vo., Lond., 1896.

Deformities, including diseases of the bones and joints. 2nd edition, 2 vols. Illus., 8vo., Lond., 1912.

Appendicitis. 8vo., Lond., 1900.

With Robert Jones. Modern methods in the surgery of paralyses. With special reference to muscle-grafting, tendon transplantation and arthrodesis. Illus., 8vo., Lond., etc., 1903.

PHILIP TURNER,

M.S., B.Sc. Lond., F.R.C.S. Eng.;

Assistant Surgeon to Guy's, 1908—

Pocket osteology. 8vo., Lond., 1908.

FREDERIC GEORGE VICARS,

M.D. Brux.

In a house of pain. 8vo., Lond., etc., 1894.

Frederic Vynon, *nom de plume* of F. G. Vicars.

JOHN WADE (1864–1912),
D.Sc. Lond., 1902; F.I.C., F.C.S.;
Lecturer on Chemistry, Guy's Hospital, 1900; University Reader in Chemistry, 1912.

Introduction to the study of organic chemistry. Illus., 8vo., Lond., 1898.

Ditto. new and enlarged edition. With an appendix containing supplementary practical detail. Illus., 8vo., Lond., 1911.

WILLIAM WALE,
F.R.Hist.S.; Wills Librarian.

Bibliography of the published writings of Sir Samuel Wilks, Bart., M.D., LL.D., F.R.S., F.R.C.P. Reprinted from *Guy's Hospital Gazette*, November 25th, 1911, with additions. Port., 8vo., Lond., 1911.

What great men have said about great men. A dictionary of quotations. 8vo., Lond., 1902.

ERNEST WILLIAM AINLEY WALKER,
M.A., D.M., B.Ch. Oxon.;
Radcliffe Travelling Fellow, Oxford, 1899—1902; Gordon Lecturer in Experimental Pathology and Director of the Pathological Department, Guy's Hospital, 1902; Gillson Research Scholar in Pathology of the Society of Apothecaries of London, 1905; University Lecturer in Pathology, Oxford.

The general pathology of inflammation, infection, being the Gordon Lectures (Guy's Hospital) for 1902. 8vo., Lond., 1904.

JOSEPH WARNER (1717—1801),
F.R.S., 1750;
Surgeon to Guy's Hospital, 1746–80; Master of the Company of Surgeons, 1773, 1780, and 1784.

An account of the testicles, their common coverings and coats; and the diseases to which they are liable, with the method of treating them. 2nd edition, with additions. 8vo., Lond., 1779.

Bound up with Warner's Human Eye.

A description of the human eye and its adjacent parts, together with their principal diseases, and the methods proposed for relieving them. 2nd edition, revised and corrected. Plates, 8vo., Lond., 1775.

Cases in surgery; with introductions, operations, and remarks. 4th edit., with considerable additions. Plates, 8vo., Lond., 1784.

JOHN WICHENFORD WASHBOURNE, C.M.G. (1863-1902), M.D., B.S. Lond.; F.R.C.P. Lond.; F.R.C.S. Eng.; Assistant Physician and Joint Lecturer on Physiology, Guy's Hospital, 1890; Lecturer on Bacteriology, Guy's Hospital; Consulting Physician to Her Majesty's Army in South Africa.

See Goodall and Washbourne's A manual of infectious diseases, 1896. 2nd edit., 1908.

Sir HERMANN WEBER, Kt.,
M.D. Bonn., 1848; F.R.C.P. Lond., 1859;
Consulting Physician, German Hospital, London.

With F. P. Weber. Climatotherapy and balneotherapy. The climates and mineral water health resorts (spas) of Europe and North Africa, including the general principles of climatotherapy and balneotherapy, and hints as to the employment of various physical and dietetic methods. 8vo., Lond., 1907.

WILLIAM JOHN WICKHAM (1798—1864),
L.S.A., 1816; F.R.C.S. (Hon.), 1843;

A practical treatise on diseases of the joints. Plates, 8vo., Winchester and Lond., 1833.

WILLIAM HALE WHITE,
M.D. Lond., 1880; F.R.C.P. Lond., 1888; M.D. (Hon.), Dub., 1909;
Senior Physician and Lecturer on Medicine, Guy's Hospital; Croonian Lecturer, R.C.P., 1897.

A text-book of general therapeutics. Illus., 8vo., Lond., etc., 1889.

Materia medica, pharmacy, pharmacology, and therapeutics. 8vo., Lond., 1892.

Ditto, 2nd edit., 1897.

Ditto, 3rd edit., 1898.

Ditto, 4th edit., 1899.

Ditto, 7th edit., 1902.

Ditto, 8th edit., 1903.

Ditto, 10th edit., 1907.

Ditto, 11th edit., 1909.

Ditto, 12th edit., 1911.

The means by which the temperature of the body is maintanied in health and disease, being the Croonian Lectures, 1897. 8vo., Lond., 1897.

Text-book of pharmacology and therapeutics. La. 8vo., Edin. and Lond., 1901.

Common affections of the liver. 8vo., Lond., 1908.

Sir SAMUEL WILKS, Bart. (1824—1911).
M.D. Lond., 1850; F.R.C.P. Lond., 1856; F.R.S., 1870;
LL.D. Edin., 1884;

Physician to the Surrey Infirmary, 1853; Curator of Museum, Guy's Hospital, 1856—65; Assistant Physician to Guy's Hospital, 1856; Examiner in the Practice of Medicine, University of London, 1866—70; Physician to Guy's and Lecturer on Medicine, 1867; Examiner in Medicine, R.C.S., 1868—75; Physician to the Duke and Duchess of Connaught, 1879; President of the Pathological Society, 1881—82; President of the Pathological Section, International Medical Congress, London, 1881; President of the Neurological Society, 1887; Member of the Senate of the University of London, 1887—1900; Member of the General Medical Council, 1887—1896; President of the Royal College of Physicians, 1896—99; Physician Extraordinary to Queen Victoria, 1897; Moxon Gold Medallist, 1897.

The "Grand Old Man of Medicine."—*The Lancet*, 1907.

When Death came as a friend to Samuel Wilks he took with him a great man, one of those rare blessings to the human race who, besides commanding our admiration for their intellect, compel our affectionate reverence for their influence on us. He did not preach, but after talking with him you left him with a fresh incentive to work for its own sake and feeling that never would you be a party to any humbug, intellectual or other. It was his enthusiasm for scientific progress, his constant interest in the investigations of others, his encouragement to beginners, his honesty, his hearty contempt of dishonesty, and the way in which he exemplified that medicine was at its highest, not when it was a means of livelihood, but when it was an intellectual pursuit—it was all these points that made him loved by those who fell under his spell, that made young men determine to do their best. . . Not until the end came did the light of his mind cease to burn as bright, steady, beautiful, and clear as ever. To him, indeed, was the hackneyed phrase, "grand old man." applicable; he was a man who would have been a glory to any profession, and happily for us he chose that of medicine."—Dr. Hale White, *The Lancet*, November 18th, 1911.

Throughout his long connexion with Guy's he was the beloved physician —all men worshipped him in a special and peculiar way. They saw in him the great physician, but more—the representative of the best in the old hospital. . . Lovable, but strong, sympathetic, but with little feeling for mere sentiment, he always lived and worked regardless of what others might say about his work. No one having to do with him could fail at once to see his wholehearted affection for Guy's and its interests. The Guy's Hospital Reports contained, he believed, the history of recent medicine and its advances better than any other publication.—Sir George Savage, *British Medical Journal*, November 18th, 1911.

The death of Sir Samuel Wilks, Bart., may be said to be the close of a chapter in the life-history of a great teaching school of medicine. As a noble follower of scientific truth, from the beginning of his career to the end, it would be difficult to find his equal in any hospital of the United Kingdom. . . That Dr. Wilks was a great offshoot of the stem of Guy's Hospital none will dispute; Dr. Addison, Dr. Bright, Dr. Hodgkin, Dr. Babington, Dr. Gull, Dr. Moxon, Dr. Hilton Fagge were all of the same stem, but they differed only in their mental pattern. A few of these physicians suffered from worldly success, and of the rest at least Guy's men can always say that Sir Samuel Wilks never coined money, because he spoke the truth, the whole truth, and nothing but the truth to his patients. As a consultant, Guy's men always feared that Dr. Wilks might come out with the truth too bluntly; thus he was not a fashionable physician of his day. . . The greatest scientific follower of truth that the hospital of Guy's can chronicle.—Dr. J. F. Briscoe, *British Medical Journal*, November 18th, 1911.

Lectures on pathological anatomy, delivered at Guy's Hospital during the summer sessions of 1857, 1858. 8vo., Lond., 1859.

Lectures on the specific fevers and diseases of the chest. Reprinted from the *Guy's Hospital Gazette* for the years 1873 and 1874. 8vo., Lond., 1874.

With Walter Moxon. Ditto, 2nd edit., 1875.

Ditto, 3rd edit., thoroughly revised by Samuel Wilks, 1889.

He stands out as the one of all others in England who first set up pathology on its proper bedrock, by the careful record of his daily work in the post-mortem room of Guy's Hospital. His published lectures on morbid anatomy should thus ever remain a commanding monument to his memory, as we hope they will also ever be the pride of his school. For although that which a man sows is not that body that shall be, and even the morbid anatomy of one generation is not exactly that of the next, let the almost complete disappearance of pyæmia and even infective disease, as Wilks saw them in his day, bear witness. The book is so carefully descriptive of the appearances of disease in all its aspects, as seen after death, that it must ever remain full of information for those who appeal to it.—From a tribute by a friend and colleague, *The Lancet*, November 18th, 1911.

Lectures on diseases of the nervous system, delivered at Guy's Hospital. 8vo., Lond., 1878.

Ditto, 2nd edit., 1883.

The Harveian Oration delivered at the Royal College of Physicians, June 26th, 1879. 8vo., Lond., 1879.

With G. T. Bettany. A biographical history of Guy's Hospital. Illus., 8vo., Lond., etc., 1892.

A memoir by Sir Samuel Wilks, Bart., M.D., LL.D., F.R.S., on the new discoveries or new observations made during the time he was a teacher at Guy's Hospital. Port., 8vo., Lond., 1911.

In perusing this volume we are impressed with the number of learned societies, in the foundation of which many of those whose lives are here portrayed have been more or less directly concerned. When the Royal Medical and Chirurgical Society was inaugurated, Dr. Saunders was the first President, and four other Guy's men had seats in the Council. The Pathological Society was founded by Dr. Edward Bentley, of Guy's; the Geological Society by Dr. William Babington: and the first President of the Epidemiological Society was Dr. Babington, Jun. Similarly, Dr. Hodgkin was instrumental in founding the Aborigines and Ethnological Societies.—*British Medical Journal*, November 26th, 1892. review of Wilks and Bettany's Biographical History of Guy's Hospital.

See also Guy's Hospital Museum—Catalogue of the pathological preparations, etc., 1863.

See also Wale's Bibliography of the published writings of Sir Samuel Wilks, Bart., 1911.

JOSEPH WILLIAMS (1815–1882),
M.D. Edin., 1839; L.S.A., 1836.

Treatise on the ear, including its anatomy, physiology, and pathology. 8vo., Lond., etc., 1840.

A. I.—*Benjamin Harrison, Esq., with the Author's respectful compliments.*

LEONARD CHARLES WOOLDRIDGE (1857—1889),
D.Sc. Lond., 1881; M.D. Lond., 1886;
G. H. Lewes Student, 1882; Grocers' Research Scholar, 1884–88; Assistant Physician and Joint Lecturer on Physiology, Guy's Hospital, 1887; Examiner in Physiology, R.C.P. Lond.; Arris and Gale Lecturer, R.C.S., 1885 and 1886; Croonian Lecturer, Royal Society, 1886.

He had a true touch of the rare gift of genius, and promised, had he lived, not only to have been a bright ornament to Guy's school, but one of the pioneers in our profession who might have ranked with Mayow or with Cohnheim, both prematurely cut off, but not so prematurely as he. *Sed Deo aliter visum est.*

He was taken in the fulness of his powers with no sad forewarning of decay; and we must be thankful that he did as much as he did. Few men have enjoyed more or succeeded better, and few have better deserved to succeed and to enjoy. He used the talents entrusted to him with rare fidelity, and his brief and brilliant career shows once more that much may be accomplished in few years.

And in short measures life may perfect be.

Dr. P. H. Pye-Smith, Obituary—L. C. Wooldridge.

On the chemistry of the blood and other scientific papers. Arranged by Victor Horsley and Ernest Starling. Plates, 8vo., Lond., 1893.

Translator of R. Heidenhain's Hypnotism or animal magnetism. Physiological observations. Translated from the 4th German edition by L. C. Wooldridge. With a preface by G. J. Romanes. 2nd edition, 8vo., Lond., 1888.

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, OR OBTAINED

OTHER DISTINCTIONS, DURING THE YEAR 1912.

University of London.

July.

Examination for the Degree of Doctor of Medicine.

Branch IV.—*Midwifery and Diseases of Women.*

H. B. Foster, M.B. | G. B. S. Maxted, B.S. (University Medal).

December.

Branch I.—*Medicine.*

H. F. Renton, B.S.

Branch IV.—*Midwifery.*

G. Dunderdale, B.S. | T. Evans, B.S. | R. Stout, B.S.

Third (M.B., B.S.) Examination for Medical Degrees.

May.

Pass.

M. M. Adams
B. Blackwood
J. H. Campain
A. F. W. Denning
E. L. R. Norton
B. R. Parmiter

R. D. Passey.

October.

Pass.

C. Aldis
T. P. Cole
G. E. Genge-Andrews
W. S. George
P. J. Monaghan
J. F. G. Richards

J. L. Stewart

Supplementary Pass List.

May.

Group I.—*Medicine.*

W. Matthews.

Group II.—*Surgery and Midwifery.*

C. Aldis | T. P. Cole

Supplementary Pass List.

October.

Group I.—*Medicine.*

A. L. FitzMaurice
J. M. Joly
G. S. Miller
W. Robinson

Second Examination for Medical Degrees.

March.

Part I.

Organic and Applied Chemistry.

E. Biddle
G. B. Dowling
D. H. A. Galbraith
A. F. G. Guinness
V. R. Hirsch
J. G. Jones
*F. A. Knott
J. Kyle
H. J. Leviseur
V. E. Lloyd
P. G. McEvedy
A. L. Stokes
H. S. Wacher
M. J. T. Wallis

**Awarded a mark of distinction.*

Part II.

Anatomy, Physiology, and Pharmacology.

G. D. Eccles
S. S. B. Harrison
W. M. Lansdale
J. T. Mackenzie
A. N. Minns
J. York Moore
A. L. Punch
M. Scott
P. D. Scott
A. G. Simmins
L. Muir-Smith
J. F. H. Stallman
J. Stephenson
L. B. Stringer
S. Wilson

July.

Part I.

Organic and Applied Chemistry.

E. S. Bowes
O. St. L. Campion
A. W. Cocking
J. W. H. Grice
*L. S. Debenham
R. L. Portway
M. B. M. Tweed
W. J. Vance
S. Vidot

**Awarded a mark of distinction.*

Part II.

Anatomy, Physiology, and Pharmacology.

C. H. Edwards
K. N. Purkiss
C. S. Lane Roberts

First Examination for Medical Degrees.

July.

E. De Robillarde
L. Gill
J. C. C. Howe
*†R. G. Meyer

**Distinguished in Organic Chemistry. †Awarded a mark of distinction in General Biology.*

December.

G. N. Anderson
‡H. F. G. Berncastle
A. B. Bond
C. G. Coombs
G. W. Elkington
G. H. FitzGerald
G. W. Heckels
J. Joffe
S. T. Wong
G. E. Kidman
J. O. R. Montocchio
E. S. Phillips
K. R. Traill

‡Distinguished in Biology.

University of Oxford.

Degree of D.M.

A. R. Wilson, B.M., B.Ch.

Degree of B.M.

R. A. Fawcus | R. C. Ozanne
R. Reynell, M.R.C.S., L.R.C.P.

Second Examination for B.M. Degree.

July.

Pathology.

F. A. Hampton | J. F. Venables

First Examination for B.M. Degree

Human Anatomy and Physiology.

R. J. W. A. Cushing. | O. G. Parry-Jones
F. L. G. Scott | D. M. P. Whitcombe

University of Cambridge.

Examination for the Degree of M.C.

J. G. Saner

Degree of Doctor in Medicine.

H. B. Carlill.

Degrees of M.B., B.C.

H. W. Barber | H. T. Depree
A. M. Bodkin | A. E. Rayner

Degree of B.C.

J. M. Jarvie | A. C. Jepson

Third Examination for Medical and Surgical Degrees.

Easter Term.

Part II.

Surgery, Midwifery and Medicine.

A. H. Birks | J. M. Jarvie
J. B. Hance | R. S. Kennedy
C. Worster-Drought.

Second Examination for Medical and Surgical Degrees.

Part I.

Human Anatomy and Physiology.

J. H. Parry.

Third Examination for Medical and Surgical Degrees.
(Old Regulations).
Michaelmas Term.
Part II.
Surgery, Midwifery and Medicine.

F. S. Adams
W. T. Chaning-Pearce
A. C. Clifford
W. C. D. Maile
A. S. Seabrooke
C. Warner
A. M. Zamora

Second Examination for Medical and Surgical Degrees.
(New Regulations).
Part II.
Pharmacology and General Pathology.
E. S. Taylor.

University of Durham.

Degree of M.B.
H. L. James.

Degree of B.S.

R. C. H. Francis | H. L. James

Third Examination for the Degree of Bachelor of Medicine.
Materia Medica, Pharmacology, Public Health, Medical Jurisprudence, Pathology and Elementary Bacteriology.

R. P. Ninnis | C. R. Smith

Second Examination for the Degree of Bachelor of Medicine.
Anatomy and Physiology.

J. F. Carter Braine
Jap-Ah-Chit, M.R.C.S., L.R.C.P.
C. C. H. Cuff
D. Richards

First Examination for the Degree of Bachelor of Medicine.
Chemistry and Physics.
R. S. Millar
Elementary Anatomy and Biology.
C. T. Helsham

Royal College of Physicians of London.

Examination for the Membership.

G. H. Hunt, M.A., B.M., B.Ch. | W. Johnson, M.D., B.S.
A. C. Jordan, M.D., B.C.

Final Examination for the Licence.

January.

T. S. Allen
H. G. Crawford
A. L. Gardner
A. C. Jephson
G. L. Preston
P. Smith
J. L. Stewart

April.

C. Aldis
T. I. Bonnett
A. H. Birks
T. P. Cole
C. F. Constant
D. A. Davies
W. E. S. Digby
C. A. Gatley
W. S. George
L. P. Harris
R. S. Kennedy
W. S. Lacey
L. Milton
F. C. Newman
R. G. Oram
R. D. Passey
M. Pern
H. F. Stephens
W. P. Vicary
H. Webb

June and July.

K. B. Clarke
S. S. Crosse
J. M. Joly
V. A. Luna
B. McDermott
W. Matthews
G. S. Miller
T. E. Roberts
M. Scott
V. F. Soothill
C. Warner
O. R. L. Wilson

October.

H. R. Bastard
F. N. Doubleday
W. K. Fry
H. Gardiner, M.B., B.S.
G. E. Genge-Andrews
H. J. Hoby
S. Keith
W. C. D. Maile
E. G. Reeve
W. R. Revnell
J. F. G. Richards
W. Robinson
H. C. Rook
C. M. Ryley
A. S. Seabrooke
W. J. D. Smyth
L. B. Stringer
W. L. Webb
A. M. Zamora

Royal College of Surgeons of England.

Final Examination for the Fellowship.

H. L. Attwater, M.B., B.C.
A. B. O'Brien, M.D., B.S.
L. Bromley, M.B., B.C.
C. C. Holman, M.B., B.C.
J. G. Saner, M.B., B.C.
T. D. M. Stout, M.B., B.S.
W. H. Trethowan, M.B., B.S.

Primary Examination for the Fellowship.

A. S. Liebson

Final Examination for the Membership.

January.

T. S. Allen
H. G. Crawford
A. L. Gardner
A. C. Jephson
G. L. Preston
P. Smith
J. L. Stewart

April.

C. Aldis
T. I. Bennett
A. H. Birks
T. P. Cole
C. F. Constant
D. A. Davies
W. E. S. Digby
C. A. Gatley
W. S. George
L. P. Harris
R. S. Kennedy
W. S. Lacey
L. Milton
F. C. Newman
R. G. Oram
R. D. Passey
M. Pern
H. F. Stephens
W. P. Vicary
H. Webb

June and July.

K. B. Clarke
S. S. Crosse
J. M. Joly
V. A. Luna
B. McDermott
W. Matthews
G. S. Miller
T. E. Roberts
M. Scott
V. F. Soothill
C. Warner
O. R. L. Wilson

October.

H. R. Bastard
F. N. Doubleday
W. K. Fry
H. Gardiner, M.B., B.S.
G. E. Genge-Andrews
H. J. Hoby
S. Keith
W. C. D. Maile
E. G. Reeve
W. R. Reynell
J. F. G. Richards
W. Robinson
H. C. Rook
C. M. Ryley
A. S. Seabrooke
W. J. D. Smyth
L. B. Stringer
W. L. Webb
A. M. Zamora

Final Examination for the Licence in Dental Surgery.

May.

R. R. Adams
S. W. Bevis
A. J. Chapman
J. S. Cocks
J. E. Davies
H. Daw
C. H. Hounsden
E. S. Huckett
F. A. Jaques
C. H. Medlock
W. R. Morris
C. D. Neale
A. J. Percy
L. R. Pickett
H. E. Shepherd
W. S. Stranack
R. D. Tanner
W. A. Thompson
F. I. Tipper
T. B. Tustian

November.

C. A. Achner
W. Adderley
H. L. Bailey
D. H. Barr
R. Boutwood
A. D. Buck
T. P. Cooper
H. O. Dumayne
J. E. R. Evans
L. P. Harris
R. M. King
F. W. Lawrence
J. W. Mayer
H. B. Neely
W. D. Partridge
I. W. Pasmore
C. J. Phillips
M. Schneider
S. J. F. Webb

Royal College of Physicians of London and Royal College of Surgeons of England.

Examination for the Diploma of Public Health.

M. M. Cowasjee, M.R.C.S., L.R.C.P. | W. R. Gibson, F.R.C.S., L.R.C.P.
F. M. Turner, M.D., B.C.

Society of Apothecaries of London.

Final Examination for the Licence.

Medicine, Surgery and Midwifery.

L. K. Edmeades

London School of Tropical Medicine.

Examination for the Certificate.

L. Doudney, M.R.C.S., L.R.C.P. | N. A Sharp, M.R.C.S., L.R.C.P.
A. D. J. Williams, M.R.C.S., L.R.C.P.

Royal College of Surgeons, Edinburgh.

Admitted to the Fellowship.

L. Myer, M.R.C.S., L.R.C.P., L.D.S. | F. Kahlenberg, M.R.C.S., L.R.C.P.

Royal Army Medical Corps.

G. A. Blake, M.B., B.S.

Indian Medical Service.

J. B. Hance, M.B., B.C. (4th place).
G. Y. Thomson, M.B., B.S. (6th place).

MEDALLISTS AND PRIZEMEN,

JULY, 1913.

Open Scholarships in Arts.

William Herbert Steavenson, Cheltenham College, £100.
Felix Raoul Leblanc, Royal College, Mauritius, £50.
Louis Gustave Philippe K/Vern, Royal College, Mauritius, Certificate.

Open Scholarships in Science.

Joseph Edouard Evariste de Robillard, Preliminary Science Class, Guy's Hospital, £120.

Harold John Bensted, Birkbeck College.
Joseph Christopher Campbell Howe, Preliminary Science Class, Guy's Hospital. } **Equal, £30 each.**

Scholarship for University Students.

Charles Putnam Symonds, B.A., New College, Oxford, £50.

Open Scholarships in Dental Mechanics.

October, 1912, **William James McBain Allan.**
Arthur Edmund Lowein, } **Equal, £10 each.**
May, 1913, **Leopold Harold Cross, £20.**

Scholarships in Dental Mechanics for Pupils of Guy's Hospital.

October, 1912. **George Hubert Howe,**
Charles Hardiman Laver, } **Equal, £10 each.**
May, 1913. **Joshua Lawrence Oates, £20.**

Junior Proficiency Prizes

James Gaymer Jones.
Peter George McEvedy. } **Equal, £17 10s. each.**

The Michael Harris Prize for Anatomy.

Jose Victory, £10.
Peter George McEvedy, Certificate.
James Gaymer Jones, Certificate.

The Sands-Cox Scholarship in Physiology.

Peter George McEvedy, £15.
James Gaymer Jones, Certificate.

Dental Prizes.

First Year's Prize in Dental Subjects.

James Millard Barnes,
Donald Clewer,
Maurice George Whitten, } Equal, £3 6s. 8d. each.

Second Year's Prize in Dental Subjects.

William Allen Bulleid. £10.

Second Year's Prize in General Subjects.

William Allen Bulleid, £10.
Edward Royston Saul, Certificate.
Cyril Henry Edwards, Certificate.

Prize for Operative Dental Surgery.

Cyril Henry Edwards, £10.

Newland-Pedley Gold Medal for Practical Dentistry

Arthur James Barber.

Golding-Bird Gold Medal & Scholarship in Bacteriology.

Arthur Joseph Eagleton Smith. £20.
Skene Keith, Certificate.

Treasurer's Gold Medal for Clinical Medicine.

Charles Hamilton Gould.

Treasurer's Gold Medal for Clinical Surgery.

John Frederick Gwyther Richards.

THE PHYSICAL SOCIETY.

Honorary President.—Sir James Goodhart, Bart., M.D., LL.D.

Honorary Vice-Presidents.—Sir George Savage, M.D., Frederick Taylor, M.D., Charters J. Symonds, M.S.

Presidents.—T. I. Bennett, R. A. Fawcus, N. Garrard, J. A. Ryle, C. S. Lane Roberts, E. D. Scott, J. L. M. Symns, G. S. Miller, W. E. Tanner, E. S. Taylor, J. F. G. Richards, O. G. Morgan, A. Seabrooke, J. York Moore.

Hon. Secretaries.—G. W. Goodhart, M.A., M.D., G. T. Mullally, M.B., B.S.

CLINICAL APPOINTMENTS HELD DURING THE YEAR 1912.

HOUSE PHYSICIANS.

H. T. Depree	H. L. James	R. D. Passey
A. Sandison	W. T. Clarke (2m.)	M. R. Dobson
G. Marshall	N. Mutch	F. G. Lloyd
	T. B. Heaton	

HOUSE SURGEONS.

G. T. Mullally	G. R. Hind	J. A. Edmond
A. J. McNair	A. N. Cox	E. G. Schlesinger
A. H. Todd	J. L. M. Symns	R. Montgomery

ASSISTANT HOUSE SURGEONS.

A. H. Todd	W. S. George	C. A. R. Gatley
R. Montgomery	G. Marshall	F. G. Lloyd
H. F. Warner	F. Cook	J. L. M. Symns
B. R. Parmiter	H. L. James	V. Glendining
J. M. Jarvie	G. T. Foster-Smith	A. C. Jepson
	L. Milton	

OUT-PATIENT OFFICERS.

A. N. Cox	A. C. Jepson	V. Glendining
A. Sandison	A. J. McNair	N. Mutch
E. G. Schlesinger	F. G. Lloyd	G. Marshall
H. L. James	A. H. Todd	T. B. Heaton
W. E. S. Digby	R. Montgomery	J. L. M. Symns
	G. T. Foster-Smith	

OBSTETRIC RESIDENTS.

G. Maxted	T. S. Allen	J. A. Edmond
R. Stout	A. S. Roe	P. Smith

CLINICAL ASSISTANTS.

F. Cook
R. Montgomery
T. B. Heaton
G. T. Foster-Smith
T. I. Bennett
H. Webb
T. P. Cole
G. S. Miller
H. P. Warner
B. Blackwood
E. R. Hart
A. C. Jepson
R. D. Passey
C. A. R. Gatley
J. M. Joly
C. Warner
B. R. Parmiter
J. L. M. Symns
V. Glendining
W. E. S. Digby
L. Milton
W. S. George
R. S. Kennedy
S. Wickenden

CLINICAL ASSISTANTS IN THE MEDICAL WARDS.

E. L. Jones
A. Seabrooke
C. M. Ryley
O. R. L. Wilson
G. S. Miller
N. Garrard
J. W. Kemp
R. S. Kennedy
L. B. Stringer
W. S. George
H. J. Hoby
A. B. Danby
D. V. Pickering
H. C. Godding
W. E. S. Digby
J. M. Joly
V. F. Soothill
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S. S. Crosse
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H. J. Hoby
V. Soothill
C. Worster-Drought
E. W. Blake
W. Matthews
W. J. D. Smyth
T. A. Townsend
H. C. Rook
F. Tooth
A. M. Henry
G. E. Genge-Andrews
P. J. Watkin

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F. W. Lawson
G. L. Preston
L. D. Wright

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G. S. Miller
M. Scott
H. Gould
H. Mather
J. F. G. Richards
C. Lambrinudi
A. H. Tamour
R. Creasy
T. E. Roberts
F. A. Hampton
R. C. Ozanne
R. H. Lucas
L. B. Stringer
H. Webb
A. C. Hancock
J. R. Barrow-Clough
W. Robinson
F. H. Dodd
F. V. Bevan-Brown
G. E. Genge-Andrews
C. Sherris
R. A. Fawcus
W. R. Reynell
C. H. L. Harper
T. I. Bennett
E. G. Martin
R. C. Matson
M. Z. Hanafy
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C. S. L. Roberts
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N. Garrard
A. M. Zamora
A. C. Clifford
S. Wickenden
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H. C. Rook
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A. B. Danby
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E. S. Taylor
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R. D. Passey
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A. K. Selim
R. S. Kennedy
F. D. Annesley
W. C. D. Maile
L. Hilton
A. H. Birks
W. R. Pryn
C. Warner
A. J. Drew
V. F. Soothill
W. E. Williams
W. J. D. Smyth
A. Seabrooke
F. W. Lawson
H. C. Godding

SURGEONS' DRESSERS.

J. A. Martin
M. Z. Hanafy
L. D. Wright
R. H. Lucas
J. W. Kemp
A. S. Erulkar
A. C. Hancock
H. Gould
H. W. Evans
H. Mather
H. S. Groves
C. Lambrinudi
E. H. Griffin
C. F. Pedley
J. York Moore
R. P. Ninnis
A. Wills
C. Dean
H. H. Elliot
Abd-el-Al
F. C. S. Broome
D. C. Scott
E. S. Taylor
A. C. Clifford
V. Atienza
P. H. Berry
A. M. Henry
E. M. Mahon
C. H. G. Penny
F. W. Lawson
J. R. Barrow-Clough
W. L. Gwyn-Davies
F. D. Annesley
J. F. Venables
C. S. L. Roberts
F. V. Bevan-Brown
R. O. H. Jones
C. G. McClymont
W. E. Tanner
A. N. Minns
D. R. Jones
A. T. W. Sheldon
L. Muir-Smith
T. A. Townsend
D. V. Pickering
C. H. L. Harper
W. R. Pryn
A. K. Selim
R. C. Matson
E. D Scott
A. J. Drew
A. J. E. Smith
A. V. Moberly
D. W. Jones
A. H. Taymour
P. Savage
F. H. Dodd
P. L. DuVerge
P. R. Chevreau
E. C. W. Starling
P. R. Boswell
S. Wilson
R. J. Hearn
H. P. Whitworth

OPHTHALMIC HOUSE-SURGEONS.

G. A. Blake
E. R. Hart

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J. York-Moore
C. F. Pedley
F. H. Dodd
C. G. McClymont
A. Wills
H. P. Whitworth
P. R. Boswell
A. N. Minns
H. H. Elliot
G. L. Attwater
N. H. W. Saw
E. R. Bailey
H. G. Dresing
O. G. Parry-Jones
L. A. J. Graham
W. A. Young
A. H. Taymour
W. K. McKay
J. F. Venables
P. Savage
P. R. Chevreau
S. Wilson
L. Muir-Smith
H. L. P. Peregrine
T. W. Sheldon
R. J. Hearn
G. B. Pritchard
F. E. R. Laborda
F. G. L. Scott
T. R. Trounce
W. M. Lansdale
S. S. B. Harrison
P. D. Scott
C. Lambrinudi
F. V. Bevan-Brown
R. O. H. Jones
P. L. DuVerge
W. E. Tanner
C. Dean
F. C. S. Broome
E. C. W. Starling
W. D. Galloway
D. R. Jones
H. L. Messenger
D. McManus
C. De W. Gibb
R. B. Campion
R. Sells
H. Parry-Price

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H. G. Crawford
H. J. Hoby
P. H. Berry
W. C. Whitworth
A. Tilbury
H. C. Godding
J. W. Kemp
F. Tooth
A. B. Danby
J. L. Perceval
W. H. Ogilvie

OBSTETRIC DRESSERS AND EXTERNS.

G. S. Miller
H. Webb
W. Robinson
A. B. Danby
V. Atienza
F. Collar
H. Sharp
W. R. Reynell
F. A. Hampton
R. C. Ozanne
N. Garrard
F. D. Annesley
W. L. Gwyn-Davies
H. W. Evans
C. H. L. Harper
A. C. Clifford
A. G. H. Moore
W. C. D. Maile
J. F. G. Richards
G. A. Pratt
F. B. Bull
R. Creasy
K. J. Keer
W. H. Ogilvie
A. M. Zamora
O. G. Morgan
R. A. Faweus
E. M. Mahon
A. V. Moberly
H. Gould
E. S. Taylor
J. A. Martin
V. F. Soothill
G. D. Eccles
C. H. G. Pochin
G. E. Genge-Andrews
S. Keith
A. S. Erulkar
G. W. King
E. G. Martin
J. A. Ryle
D. W. John
C. Sherris
R. H. Lucas
J. R. Barrow-Clough
E. D. Scott
P. H. Parry

EXTERN OBSTETRIC ATTENDANTS.

K. B. Clarke
O. R. L. Wilson
K. J. T. Keer
C. H. G. Pochin
G. D. Eccles
W. C. D. Maile
H. C. Rook
W. Robinson
J. A. Martin
D. W. Jones
N. Garrard
R. C. Ozanne
D. W. John
A. K. Selim
F. A. Hampton
H. J. Hoby
F. B. Bull
F. Collar
G. S. Miller
W. L. Webb
A. B. Danby
M. Z. Hanafy
A. V. Moberly
D. V. Pickering
J. A. Ryle
O. G. Morgan
R. A. Faweus
J. L. Perceval
A. M. Zamora
S. Wickenden
G. W. King
R. Creasy
J. F. G. Richards
G. A. Pratt
A. G. H. Moore
J. W. Kemp
A. C. Clifford
C. Sherris
W. H. Ogilvie
H. S. Groves
E. G. Martin
H. Mather
W. R. Reynell

POST-MORTEM CLERKS.

R. Creasy
S. Keith
H. C. Rook
F. Collar
G. E. Genge-Andrews
H. C. Godding
C. Sherris
A. S. Erulkar
E. S. Taylor
H. Mather
C. Dean
L. B. Stringer
E. M. Mahon
W. L. Webb
W. C. Whitworth
F. Tooth
J. T. Keer
O. G. Morgan
V. Atienza
P. H. Berry
A. C. Hancock
A. J. E. Smith
T. I. Bennett
H. Sharpe
D. C. Scott
J. L. Perceval
K. B. Clarke
J. A. Ryle
J. F. Venables
R. H. Lucas
T. W. Sheldon
C. H. G. Penny
R. C. Matson

CLERKS IN THE ELECTRICAL DEPARTMENT.

H. G. Crawford
T. B. Heaton
R. C. Ozanne
A. V. Moberly
E. M. Mahon
S. S. Crosse
J. W. Kemp
G. B. H. Jones
H. N. Eccles
A. M. Zamora
H. C. Godding

MEDICAL WARD CLERKS.

J. R. Barrow-Clough
A. V. Moberly
A. J. Drew
H. Mather
C. H. G. Penny
W. L. Gwyn-Davies
S. Wilson
A. Wills
A. N. Minns
C. Dean
H. L. P. Peregrine
C. F. Pedley
R. O. H. Jones
P. Savage
J. York Moore
P. R. Chevreau
G. B. Pritchard
H. L. Messenger
F. E. R. Laborda
H. G. Dresing
T. R. Trounce
W. M. Lansdale
L. A. J. Graham
J. E. Davies
J. S. Cocks
A. S. Liebson
W. H. Nicholls
J. H. Parry
J. F. H. Stallman
H. W. Evans
F. W. Lawson
A. J. E. Smith
R. C. Matson
H. S. Groves
L. Muir-Smith
F. C. S. Broome
H. P. Whitworth
T. W. Sheldon
W. D. Galloway
E. C. W. Starling
C. Lambrinudi
R. P. Ninnis
C. S. L. Roberts
D. C. Scott
J. F. Venables
W. A. Young
E. R. Bailey
D. McManus
G. L. Attwater
C R. Smith
O. F. Parry-Jones
H. Parry-Price
C. E. Petley
J. Stephenson
L. R. Pickett
D. O. Richards
B Burnside
H. Gould
Abd-el-Al
A. C. Hancock
E. D. Scott
F. D. Annesley
W. E. Tanner
D. R. Jones
H. H. Elliot
P. R. Boswell
R. J. Hearn
F. V. Bevan-Brown
A. H. Taymour
C. G. McClymont
W. K. McKay
P. L. DuVergé
F. H. Dodd
R. B. Campion
C. De W. Gibb
N. H. W. Saw
F. G. L. Scott
S. S. B. Harrison
P. D. Scott
R. Sells
A. H. Harkness
R. W. Cushing
L. Horsley
A L. Anthony
R. Quesada

SURGICAL WARD CLERKS.

G. B. Pritchard
H. L. Messenger
G. L. Attwater
F. E. R. Laborda
R. Quesada
J. S. Cocks
J. E. Davies
A. S. Liebson
A. H. Harkness
J. Stephenson
H. Q. F. Thompson
A. K. Day-Lewis
S. Sampson
R. W. P. Jackson
W. A. Young
E. R. Bailey
C. De W. Gibb
F. G. L. Scott
P. D. Scott
C. E. Petley
L. R. Pickett
R. W. Cushing
L. Horsley
J. H. Parry
J. F. Mackenzie
W. L. E. Reynolds
N. E. Kendall
C. P. Symonds
R. B. Campion
T. R. Trounce
L. A. J. Graham
W. H. Nicholls
S. S. B. Harrison
C. H. Medlock
B. Burnside
D. O. Richards
A. L. Anthony
R. W. P. Jackson
L. S. Fry
H. F. T. Hogben
K. Westman

DENTAL APPOINTMENTS HELD DURING THE YEAR 1912.

DENTAL HOUSE SURGEONS.

H. L. Meyer
W. S. Stranack
H. D. Shore
S. W. Bevis
D. Wain
H. Daw

ASSISTANT DENTAL HOUSE SURGEONS.

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W. Adderley
M. Schneider
A. D. Buck
T. E. Henderson
J. W. Mayer
H. B. Neely
L. P. Harris
W. S. Stranack
C. A. Achner
F. W. Lawrence
W. G. S. Neely

CLINICAL ASSISTANTS IN THE CONSERVATION ROOM.

W. G. S. Neely
A. J. Barber
M. F. Hopson
L. P. Harris
R. Boutwood
S. Hanreck
C. Glover
C. E. Thomas
D. B. Morrish

ASSISTANT DEMONSTRATORS IN DENTAL MECHANICS.

M. Schneider
M. F. Hopson
H. Millett
A. J. Barber

ASSISTANT DEMONSTRATORS IN DENTAL METALLURGY.

W. A. Bulleid
R. H. Rix
C. H. Edwards
C. A. Achner

ASSISTANT DEMONSTRATORS OF DENTAL MICROSCOPY.

A. D. Buck
T. P. Cooper

DRESSERS IN THE GAS ROOM.

F. B. Stradling
G. L. Pemberton
J. E. Davies
A. F. Salsbury
V. F. H. Golledge
W. J. O'Kane
D. H. Barr
H. L. Bailey
I. W. Pasmore
J. H. H. Griffin
A. D. Buck
C. Glover
M. F. Hopson
M. Schneider
L. R. Pickett
O. N. Mash
W. T. Flooks
T. P. Cooper
C. J. Phillips
C. H. Medlock
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I. R. Tustian
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H. B. Neely
F. W. Lawrence
G. K. Moore
R. Boutwood
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C. D. Neal
W. R. Morris
H. N. Purdom
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A. S. Clarke
S. Hanreck
J. S. Palmer
R. W. Ballard
P. S. McFarlane
R. W. Spong
G. F. Charles
W. G. S. Neely
C. A. Achner
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F. J. Neal
C. H. Edwards
J. M. Stebbings
L. T. Montgomery
L. P. Harris
S. Adams
W. T. Flooks
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S. Stevens
R. H. Rix
L. C. Cohen
A. B. G. Underwood
C. F. Haine
L. D. Windermer
W. L. Partridge
R. M. Veale
M. F. Hopson
W. O. Roberts
B. Isaacs
F. H. Morrell
A. J. Barber
E. C. Rycroft

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L. Alabone	A. A. Brown	S. W. Coffin
R. G. Bradley	R. Cowell	L. S. Pilbeam
K. C. Pitman	G. V. Saunders	R. A. Collector
C. J. Crocker	R. J. Pickett	H. L. Smith
A. B. G. Underwood	A. A. C. Crowe	W. O. Roberts
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T. P. Cooper	B. Isaacs	G. L. Cutts
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M. Pearson	W. C. Hammond-Williams	E. R. Saul
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L. T. Montgomery	K. C. Brough	C. H. Edwards
R. C. W. Staley	V. R. Dyke	F. J. Neal
E. C. Rycroft	M. G. Whitten	K. T. McAlpin
S. E. Johnson		

CASUALTY DRESSERS.

R. W. Ballard	C. Glover	S. Hanreck
M. F. Hopson	L. C. Cohen	B. Isaacs
W. G. S. Neely	L. T. Montgomery	R. H. Gaverick
E. E. Johnson	R. H. Rix	A. J. Barber
S. Adams	G. F. Charles	E. C. Rycroft
E. R. Saul	C. E. Thomas	R. Boutwood
R. L. Donn	C. F. Haime	L. D. Windermer
L. P. Harris	W. O. Roberts	S. Stevens
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G. V. Saunders	H. G. James	A. H. Cole
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C. H. Oliver	M. Pearson	R. J. Pickett
L. S. Pilbeam	A. B. G. Underwood	W. A. Bulleid
H. L. Smith	H. C. Duggan	S. E. Johnson
J. L. Garrard	P. King	

GUY'S HOSPITAL.

MEDICAL AND SURGICAL STAFF.
1913.

Consulting Physicians.—P. H. Pye-Smith, M.D., F.R.S.; Sir James Goodhart, Bart., M.D., LL.D.; F. Taylor, M.D.

Consulting Surgeons.—Thomas Bryant, M.Ch.; Sir H. G. Howse, M.S.; W. H. A. Jacobson, M.Ch.; R. Clement Lucas, B.S.; C. H. Golding-Bird, M.B.; Charters J. Symonds, M.S.

Consulting Physician for Mental Diseases.—Sir George Savage, M.D.

Consulting Ophthalmic Surgeons.—C. Higgens, Esq.; W. A. Brailey, M.D.

Consulting Aural Surgeon.—W. Laidlaw Purves, M.D.

Consulting Dental Surgeons.—F. Newland-Pedley, Esq.; W. A. Maggs, Esq.

Consulting Anæsthetist.—Tom Bird, Esq.; H. F. Lancaster, M.D.

Physicians and Assistant Physicians.

W. Hale White, M.D.
Sir E. Cooper Perry, M.D.
L. E. Shaw, M.D.
J. Fawcett, M.D.
A. P. Beddard, M.D.
H. S. French, M.D.
A. F. Hertz, M.D.
H. C. Cameron, M.D.
G. H. Hunt, M.B.

Surgeons and Assistant Surgeons.

Sir W. Arbuthnot Lane, Bart., M.S.
L. A. Dunn, M.S.
Sir Alfred Fripp, M.S., C.B., K.C.V.O.
F. J. Steward, M.S.
C. H. Fagge, M.S.
R. P. Rowlands, M.S.
P. Turner, M.S.
E. C. Hughes, M.C.
R. Davies-Colley, M.C.

Obstetric Surgeons.

G. Bellingham Smith, M.B., B.S.
H. Chapple, M.C.

Ophthalmic Surgeons.

H. L. Eason, M.S., M.D.
A. W. Ormond, Esq.

Surgeons in Charge of Throat and Ear Department.

W. M. Mollison, M.C.
T. B. Layton, M.S.

Surgeon in Charge of Actino-Therapeutic Department.

C. E. Iredell, M.D.

Surgeon in Charge of the Orthopædic Department.

W. H. Trethowan, B.S.

Surgeon in Charge of the Genito-Urinary Department.

A. R. Thompson, Ch.M.

Physician in Psychological Medicine.

Maurice Craig, M.D.

Physician in Charge of Skin Department.
SIR E. COOPER PERRY, M.D.

Physician in Charge of the Department for Nervous Diseases.
A. F. HERTZ, M.D.

Physician in Charge of the Department for Diseases of Children.
H. C. CAMERON, M.D.

Dental Surgeons.

R. WYNNE ROUW, ESQ.	M. F. HOPSON, ESQ.
H. L. PILLIN, ESQ.	J. B. PARFITT, ESQ.

Assistant Dental Surgeons.

J. L. PAYNE, ESQ.	F. J. PEARCE, ESQ.
E. B. DOWSETT, ESQ.	H. P. AUBREY, ESQ.

Resident Dental Officer.
F. BOCQUET BULL, ESQ.

Anæsthetists.

G. ROWELL, ESQ.	L. BROMLEY, M.C.
C. J. OGLE, ESQ.	H. GARDINER, M.B., B.S.
H. M. PAGE, ESQ.	G. T. MULLALLY, M.B., B.S
F. E. SHIPWAY, M.D.	A. H. TODD, M.B.
H. T. DEPREE, M.B.	A. J. MCNAIR, M.B., B.C.

Bacteriologist.
J. W. H. EYRE, M.D.

Chemical Pathologist.
J. H. RYFFEL, M.B.

Morbid Histologist.
G. W. NICHOLSON, M.D.

Radiographers.

E. W. H. SHENTON, ESQ.	W. LINDSAY LOCKE, M.B., B.Ch.

Medical Registrars and Tutors.

N. MUTCH, M.B., B.C.	H. W. BARBER, M.B., B.C.
E. P. POULTON, M.B., B.Ch.	T. B. HEATON, M.B., B.Ch.

Obstetric Assistant and Registrar.
A. J. MCNAIR, M.B., B.C.

Surgical Registrars and Tutors.

H. GARDINER, M.B., B.S.	A. H. TODD, M.B., B.S.

Ophthalmic Registrars and Clinical Assistants.

H. LEE, M.B., B.C.	G. MAXTED, M.B., B.S.

Resident Surgical Officer.
J. G. SANER, M.C.

Curator of the Museum.
R. DAVIES-COLLEY, M.C.

Warden of the College.
L. BROMLEY, M.C.

Lying-in Charity.
G. BELLINGHAM SMITH, M.B., B.S., AND H. CHAPPLE, M.C.

Dean of the Medical School.
H. C. CAMERON, M.D.

MEDICAL SCHOOL STAFF.

Medicine.

Clinical Medicine ... ... THE PHYSICIANS AND ASSISTANT PHYSICIANS.

Principles and Practice of Medicine (Lectures) W. HALE WHITE, M.D., SIR E. COOPER PERRY, M.D., L. E. SHAW, M.D., AND J. FAWCETT, M.D.

Practical Medicine ... ... N. MUTCH, M.B., B.C., E. P. POULTON, M.B., B.Ch., H. W. BARBER, M.B., B.C., AND T. B. HEATON, M.B., B.Ch.

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