GUY'S HOSPITAL REPORTS.

EDITED BY

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AND

HERBERT FRENCH, M.D.

VOL. LXVIII.,

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PREFACE.

OWING to the difficulties of one kind and another that have arisen on account of the Great War there has been unavoidable delay in the issue of the present volume of the GUY'S HOSPITAL REPORTS. Every Member of the Staff of the Hospital has been so fully engaged with extra work arising directly or indirectly out of the needs of the wounded and sick that little, if any, time has been available for the writing of papers or reports; hence, although many of the articles in this volume were already in print by August, 1914, their publication has been delayed for nearly a year, and we tender our apologies to their authors for this. We also apologise to those with whom we are in the habit of exchanging the GUY'S HOSPITAL REPORTS and to our regular subscribers. We hope that the present difficulties will be overcome and that the annual issue of the GUY'S HOSPITAL REPORTS will not be interrupted, and that the issue of the volume due in 1915 will not be delayed unduly.

THE EDITORS.

August, 1915.

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From an Oil Painting on the Staircase leading to the Governors' Court Room, Guy's Hospital.

THOMAS BRYANT,

HON. M.Ch. R.U.I., HON. M.D. DUBLIN, HON. F.R.C.S.I.

In Memoriam.

THOMAS BRYANT.

By

C. H. GOLDING-BIRD.

It is a great thing to have lived and worked through a period of transition; the old beaten track has no longer to be followed, but new paths have to be carved out, and scope is thereby given to the man of action both to display his abilities, advance knowledge, and benefit mankind. Such was the period during which Thomas Bryant carried on his life's work; for surgery as he learnt it as a student was but the mechanical handmaid to medicine, any explanation of its inner processes being purely empirical, whilst its intended results—from causes then beyond the ken of the surgeon—were almost as difficult to attain with certainty as they had been in the dark ages.

The long list of his publications which is printed in this volume after this survey of Bryant's life does not, therefore, indicate the mind of a man wandering with uncertainty over a vast field of knowledge, but that of one who, appreciating the changes that were rapidly arising in his environment, did his best to add to the sum of human knowledge by investigating every new event in the theory and practice of surgery, as each came to the front.

Great, indeed, though his predecessors at Guy's had been, their lines had not fallen in such pleasant places as his, and the surgery in which they dealt was becoming rapidly, in his days, elevated to one of the greatest sciences the century had seen.

Bryant was the son of Thomas Egerton Bryant, who was educated at Guy's, and who practiced in Kennington in the first half of the last century, where he held an honoured position, and found time, in spite of busy practice and his surgeoncy to the Lambeth Infirmary, to devote much attention to the then young science of pathology. Some of his specimens adorn the shelves of the Museum at Guy's. He was also at one time President of the Medical Society of London, and we may justly infer that it was from him that his son inherited many of the tastes that served him in good stead in his professional life.

Thomas Bryant was born on May 20th, 1828, was educated at King's College School, and was apprenticed, as the custom then was, to Mr. Duke, of Kennington. He entered Guy's in 1846, when the surgical staff consisted of men who made history, and whom Bryant was never weary of quoting, and whose teaching largely influenced his own line of thought and practice. They were Aston Key (to whom he was dresser), John Morgan, Bransby Cooper, Thomas Callaway, Edward Cock, John Hilton, John Birkett, and Alfred Poland. Five years before Sir Astley Cooper had died, so only indirectly did he come under this great surgeon's influence, but he was a profound student of Sir Astley's writings, whilst he must have heard much about that surgeon's methods of practice from his father, Egerton Bryant, whose intimate friend Sir Astley had always been.

Bryant qualified (M.R.C.S.) in his third year and became a Fellow of the College in 1853. In 1857 he was appointed Assistant surgeon to Guy's, and two years later taught operative surgery. He never went through the anatomical mill in the dissecting room, a disadvantage that he always subsequently felt; but as it was not until 1871 that he became full surgeon, he had the advantage of the practice and of the opportunities of teaching in the Out-Patient Department, which were, without doubt, as valuable to himself, as many a Guy's man can to this day bear witness they were to the students of that time.

He had been some ten years on the staff when the present writer became his pupil, and subsequently his dresser, by which

time Bryant was entitled to teach with the authority derived from experience, and so his methods and style at this stage are worth recording. To begin with, his personality at once attracted the student's attention, whilst his cheery, encouraging, and even optimistic temperament threw a glamour over his methods, that made the student feel that after all surgery could not be so dry a subject, nor so difficult to master, as he had at first, perhaps, thought. Sympathetic with the student in his difficulties, Bryant was always ready to listen to the inquiries of the most junior man, whilst his gift of clearly expressing himself, and of being able to convey what was in his own mind, left no doubt in his hearer's that he had obtained the fullest explanation of his difficulty. It was no offhand instruction that Bryant gave, but it was clear to all that he took trouble to place himself in his inquirer's position, and thus an appreciative answer could always be drawn from him. In this way he at once manifested himself as one to be trusted, and the numbers that crowded his out-patient room were sufficient evidence of the success of his methods. Rarely, too, was he without the presence of one or more professional friends, who not infrequently brought difficult cases themselves for the purpose of elucidation.

His kindness to the poor was another feature in his work, for if there is one thing more repellent to the junior student than another, it is a harsh, unsympathetic attitude towards the class of patients that crowd our hospitals. This characteristic, too, had a still further-reaching influence, for whilst it filled his room with the Bermondsey population—with whom his name was a by-word—it added also to the extent of his out-patient practice, and provided him with a superabundance of material for teaching purposes. He was also a man of method and precision. He would inculcate the uselessness of attending out-patients in a lax or *laisser aller* spirit, as too often happens, the student hoping to pick up something somehow! No! That did not suit Bryant at all, so he encouraged the taking of notes, one of his ways being to advise the interleaving of whatever text-book was read, and of noting thereon against the subject in

the book any striking remark the student might hear or any case he might see that bore upon it.

He always came down himself armed with note-books in which he rapidly jotted down almost every case; and at one time introduced a plan of tickets, with numbers corresponding to his notes, so that he might turn up any case that should reappear after a long interval, for the study of the continuity of cases was a great point with him. These note-books furnished him later on with the material for statistics, of which he was particularly fond, and from which the writer cannot help thinking he too often drew conclusions that bare statistics hardly justified.

At the time of which we are speaking class teaching was hardly introduced into the London medical schools. The student had to both select the clinical teacher he admired and lay himself out to garner all the knowledge that he could in his own way. So the personal equation of the teacher was rated at a higher value and drew more disciples than it possibly does at the present time when so much instruction is of the classified or "spoon-meat" variety!

In Bryant's time the assistant surgeons had fewer beds than later on, when surgery in the medical wards was developed; yet his bi-weekly visit to the wards was attended by a very large following, and especially of men preparing for their final, since they valued the clear, incisive and positive statements made rather than the more speculative, though none the less valuable, discussions that they might meet with in the clinical teachings of others.

It was whilst still Assistant Surgeon that Bryant wrote his world-renowned text-book on the Practice of Surgery, a work much needed by students and junior practitioners, for whom "Druitt" was hardly enough and the three-volume Holmes' System too much. The book, like the author's *vivâ voce* teaching, was clear, practical, and unhesitating in expression; and being the outcome of a large and ripened experience, brought every subject vividly before the mind, and was hence as much appreciated by the busy practitioner as it was by the student of

surgery. It first appeared in 1872, the year after Mr. Bryant's appointment as full Surgeon to the Hospital, and at once became the universal and popular text-book of the day. It appealed especially to Guy's men, for it was written from material gathered almost entirely within the walls of the Hospital, whilst, as the author says in the preface, of its five hundred illustrations, four hundred were from objects in the Guy's Museum, or direct from nature; and many old Guy's men who may look into it to this day will recognise over and over again admirably executed engravings of surgical affections from cases which they may even themselves have dressed in the out-patient department or the wards.

Released at length from what some have considered the drudgery of out-patients—though to Bryant a never-ending source of experience and interest—he worked even harder than before—if that were possible—in the interests of the students; and it was a hard matter to get near the bedside for the press of his clientèle, in Job and Lydia, which were his two general wards, and as he was punctual to the minute in his arrival at the hospital his dressers and others never had to "mark time" in the Colonnade for him. He was always anxious that those he taught should have grounds for their belief in his diagnosis and explanations, and, therefore, whenever it could be done without pain or inconvenience to the patient he insisted on his pupils examining the cases for themselves, and always seemed to have time to devote to their difficulties or questions. Bryant was not a man to hurry, but every minute of his time in the hospital was accounted for, and it was by method that he got through the mass of work which he often accomplished in an afternoon.

Until toward the end of his term of service in the hospital it was customary to attend two days a week to visit the wards, and two days, Tuesday and Friday, were the only operating days, save for cases of sudden emergency. In the days of his own studentship it was the custom for the senior surgeons to sit in the front of the theatre, when possible, to witness the operations of their juniors and to give them the benefit of their advice; and

Bryant's presence at an exceptional operation was not unfrequently sought by a junior, for it was known that he would not come in any unfriendly criticising spirit, but with every encouragement either of word or manner. However difficult the end to be accomplished, either in his own work or that of others he might be assisting at, he never lost heart, but was optimistic almost to a fault; and in private practice this stood him in good stead, for his patients could but feel that the best possible was being done for them, and that whilst there was life there was, indeed, hope.

The writer—who knew Bryant intimately—cannot but feel that in thus acting he often sacrificed himself in order to encourage others; and many were the times that when, perhaps returning from a private case where he had been assisting Mr. Bryant, he has seen him give way to an exhibition of great and heartfelt concern over a patient, of whose ultimate recovery, just before, one would not for a moment have doubted, from the hopeful view publicly taken, in order to spare the feelings of the friends. All this was thoroughly genuine, there was none of the false buoying up of expectations that had no justification in fact, but where the smallest justification existed the most was made of it.

It has already been said that the great change in the science and practice of surgery—the greatest, indeed, that it has ever seen—occurred during Mr. Bryant's career, and it was about the time of his becoming full surgeon that the transition began to make itself universally felt. Educated on the lines of Sir Astley Cooper's teaching, a great admirer and follower of Aston Key, the new state of things brought about by the investigations of Pasteur and Lister seemed to Bryant to promise too much. Hitherto the best endeavours of the surgeon were again and again frustrated by "inflammatory or surgical fever," with its local complications, which, in the absence of knowledge of its ætiology, all attempts to ward off or to treat efficiently had been futile, and although it slowly was being understood that cleanliness was requisite for a good result, even this was only practised in a way that would make the modern student of surgery smile.

A towel clean from the laundry was perfection! but the washing of hands just before operation was by no means constantly practised, and instruments were clean to use as they came from the cupboard of the instrument-keeper. Dressings were lint dipped in water, or poultices! and for a granulating surface that was "unhealthy," such applications as Lotio ac. Nitrici or Lotio Plumbi, as the case required, were employed. It was, however, appreciated that roughly-handled tissues were more likely to fare badly than those that were cleanly divided, and the entrance of air was considered as the great exciting agent for harm, whilst this theory seemed borne out by the sure results that followed subcutaneous surgery.

Although chloroform was employed as an anæsthetic (ether only crossed the Tweed later on), yet its crude administration produced risks that it was well to avoid by as rapid operation as possible, and to "whip off" a leg in sixty seconds, with some surgeons, as John Poland, was a distinct point.

Now all these facts must be borne in mind as explanatory of Bryant's methods of operating. He never cared for exploratory surgery, but felt it his duty to produce a definite result, when he took the knife in hand, for the benefit of his patient. He was a rapid, but not hurried, operator; every cut was "clean," without hesitancy, and made with the intention of reaching the objective as quickly, and with as little injury, as possible, and as soon as practicable afterwards he closed the wound. In this he was very particular and neat, and with the view of obtaining as much primary union as posssible, every part was minutely adapted. The next step taken was in accord with the teaching of his senior, John Birkett. Every particle of moisture was thoroughly expressed, and clean towels and napkins suitably folded were placed on either side of the incision and firmly bandaged on. The use in those days of strapping was also considered suitable for supporting flaps, and so no wonder that the mechanical care employed was often defeated by the biochemical influence of the "Emplastrum Plumbi." However, the writer can affirm from personal observation that, on the whole,

the cleanest and best results were to be found in the wards of John Birkett and Thomas Bryant.

Of individual operations and the means employed in after-treatment to defeat the too real bogie of "Inflammation," pages might be written, but it would serve no present purpose, and would be but nowadays of archæological interest.

Specialism in Bryant's time, save for the eye, the ear, and diseases of women, was hardly existent. The teaching inculcated in Guy's then and for long after was that specialism was quite unsuitable in general hospitals and was to be discouraged. In medicine, Samuel Wilks strongly insisted on this view, and on the surgical side Bryant as strongly supported it, a position, in the light of recent discovery and greater range of possibilities for the surgeon, quite untenable. It, however, accounts for Bryant's catholicity of practice. There is hardly a surgical matter on which he did not write, and, indeed, on which he was not an authority in his day.

Yet, in spite of himself, he came to be thought a specialist in abdominal work—almost confined to colotomy, in breast surgery, and in ovariotomy, in all of which both in private and in public he had a very large practice.

As an ovariotomist, however, Bryant for years was held in the highest repute, and that at a time when this operation was considered the gravest a surgeon could be called upon to perform, surrounded as it was by risks due to septicism, hæmorrhage, and other causes not then well understood; so that he who undertook it was, of necessity, a bold man. His results, however, due to his rapid and skilful operating and his strict attention to cleanliness, as then understood, and to the avoidance of all unnecessary manipulation, were such as fully to justify the high opinion formed of him as an ovariotomist.

And here it may not be uninteresting to record how he operated in these cases in the early seventies.

In the first place, if it were a case in which there was reason to think there were likely to be adhesions, palliative treatment by repeated tappings, rather than radical operation was con-

sidered the safer treatment, for the exposure and manipulation of the viscera necessary to free adhesions, experience had at that time shown, were almost certain to be followed by fatal peritonitis. The case having, therefore, been duly selected, it was isolated in an upper room in Clinical, and only those were allowed to be present who had not been for three days in the post-mortem room or had not had dealings with a septic patient. The special table had a large drawer, metal lined, into which the fluid of the cyst could be run through the Spencer-Wells cannula and rubber tube, but no disinfectant was ever employed in cleansing it. The various instruments were placed to hand on a clean towel and the various ligatures and sutures of silk were hung on brass hooks screwed into the sides of the adjacent window frame, as the cleanest place and easiest of access.

The surgeon and his assistants wore macintosh aprons, and a large new sheet of the same material, with an oval aperture in its middle, its edges smeared underneath with eplastrum plumbi, was thrown over the body of the patient, the plaster thus fixing the waterproof all round the area of the abdominal incision. The parietes were then incised, it being the duty of one dresser to keep a hand on either side of the belly to press forward the tumour and thus keep the wound filled by its bulk that no intestine might protrude. The cyst then having been tapped, it was drawn forward till its pedicle lay outside, the dresser's hands keeping the lips of the wound pressed together to the utmost extent. The pedicle was then ligatured and the tumour removed.

The treatment of the pedicle was a cause for great anxiety. Until Lawson Tait introduced the "Staffordshire" knot, the ordinary ligation in two parts was uncertain in its results, especially if the pedicle were a thick one; and hæmorrhage was to be subsequently feared, and certainly accounted for many a life lost; and there was the added danger of septicism from the pedicle being returned into the abdominal cavity. A marked improvement in results followed the extra-peritoneal method of treating the pedicle; this was done by fixing it in a suitable

steel clamp, before cutting away the cyst, and retaining it outside the closed wound; and its cut surface was often seared with the actual cautery.

When, as sometimes happened, the bulk of the tumour having been extruded, the last portions, owing to adhesions, would not come forward without bringing gut with them, the tumour was cut away as closely as possible with scissors, and the cup-like portion that remained was sewn to the parietes and allowed to granulate up.

Criticised in the light of present-day knowledge it certainly is a wonder that so many cases recovered as was actually the case, but the selection of "suitable cases" was an undoubted item in the results. The attempt to relieve by operation every ovarian case that presented itself was then quite unwarrantable.

It was also in the seventies that the use of the electric cautery was introduced, and Bryant was its great advocate in Guy's The bichromate battery was the source of electricity, and both the wire, ecraseur, and electric knife were employed. It was believed that by its use the seared surface would not be so liable to introduce septicism, whilst in certain cases is was a safeguard against hæmorrhage. Bryant used the ecraseur extensively for removal of the tongue, the loop of platinum wire being introduced through a puncture in the floor of the mouth, and placed as far back as possible over the tongue, being kept there by needles thrust through the organ. The ends of the wire then being tightened up in the ecraseur handle, the current was passed through to heat the wire, and so in a few minutes the tongue could be burnt out. If hurriedly done, hæmorrhage was likely to follow, but with care this could be avoided; still, the sloughing stump was a serious matter in after-treatment.

Mr. Bryant's attitude at first in regard to Listerism—as it came to be known—is comprehensible enough. This method of treatment seemed to him to promise too much, and hence in the first edition (1871) of his "Practice of Surgery" he merely contented himself with extensive quotations from Lister's writings, his own commentary being, "I have given the practice [*i.e.*, of

antiseptics] in the words of the distinguished originator, feeling that if it had no other influence to back it than Professor Lister's name and authority, it ought to have a fair trial and be honestly tested."

Thus far, perhaps, an open mind may be justified, but it is rather difficult to gauge the reason for the position that Bryant afterwards assumed, and maintained, of unqualified opposition to Lister's new line of practice.

It can only be supposed that, having achieved better results than those of others by the employment of the means of after-treatment already alluded to, he could not bring himself to believe that much more was to be gained by the use of carbolic acid and other chemical adjuncts. Possibly, also, some prejudice against Listerism may have been aroused in his mind by the undoubted unpleasantnesses—not to say dangers—that the early use of the then not very pure carbolic acid often gave rise to, but the fact indisputably remains that Bryant never could bring himself to adopt the antiseptic methods of Lister. As time went on it was, however, impossible for him to resist the pressure of circumstances, and so in an imperfect way he made use of carbolic lotion and gauze, but he never employed the "spray," to which undoubtedly much of the early successes of, and widened field of operation for, surgeons was due; though it was rightly abandoned when, the nature of bacteria being understood, it was found possible to combat their influence by means less disagreeable to the operator and less injurious to the wound.

For all this, Bryant, true to his spirit of inquiry, tried to find out for himself some way of improving his results, which were now falling behind those of others, and, gradually dropping carbolic acid, etc., he began to employ (and continued to do so to the last) iodine water and peroxide of hydrogen. Curiously enough both these drugs have in late times again been much vaunted and employed, but if the writer may judge by what he had the opportunity of seeing in Bryant's practice, he certainly would never trust to them himself, either as preventives against infection or in combating its results.

Another subject which developed itself in Bryant's time was that of the ligation of arteries, either in their continuity or after division. The recognised method in his early days was simple ligature with silk, the ends of the ligatures being left out of the wound, and the hope was that within ten days they would come away, with gentle traction, and leave the vessel obliterated, an end not always, by any means, obtained, for secondary hæmorrhage was both common and commonly fatal. In the septic state of the wound the modern surgeon can see the explanation of this, but it was not at first so regarded, and attempts of different kinds were made to do away with ligatures altogether. Into this practical matter Bryant entered heart and soul, and the outcome was the introduction of the torsion forceps that bore his name, and held the field for years before the Spencer-Wells pattern ousted them.

He was, indeed, the great exponent, at Guy's, of torsion as against ligature of arteries, and his name will always be associated with it. He was the first to employ it on the largest arteries, as the femoral and external iliac, and he was always most careful that the dressers should understand the *raison d'être* of the process that they might apply it intelligently. He also introduced a suggested method of occluding an artery in it continuity, by drawing it up, at the point selected, into a suitably fitting silver cannula, by means of a hook which, having been first protruded from the cannula, encircled the vessel, and by the traction thus exerted the inner coats of the vessel were completely ruptured. Owing, however, to the imperfect antisepsis of the time the crushed vessel walls could not be relied upon to resist septic changes, so the method, after full trial, was abandoned. Bryant was for a time, however, very sanguine that this method would simplify the treatment of aneurysm, by the proximate occlusion of the vessel, but finding it finally too uncertain he continued to employ digital pressure, and he never failed to find willing hands, day and night, for the purpose.

By his never employing Listerian methods there is no doubt Bryant's area of operations, such as others could with certain

results perform, gradually became more and more limited; and such operations as the opening of a normal joint—as for removal of a loose cartilage or wiring a patella—were rarely amongst those that he employed. The same limitation also imposed itself on those operations in which the normal peritoneum had to be reckoned with.

Speaking of abdominal work, it may be here put on record that Bryant was the first, in Guy's, to explore what was then called "typhlitis," *i.e.*, appendicitis. It was a case where a tumour had formed and pus was present, and though he had no precedent to guide him, he boldly took the matter in hand, but never suspected—as did no one then—that these cases were of appendicular origin. The presence of the "date stone" concretion, he believed, indicated the origin of the trouble to be a foreign body, and taught that the swallowing of date or cherry stones was not without risk.

From 1871 to 1888 Bryant lectured at Guy's on Systematic Surgery, and his clear exposition of his subject, his careful and methodical marshalling of his facts, his frequent illustration from his own experience, and his avoidance of speaking above the apprehension of his audience, rendered his lectures very popular as well as instructive and interesting, and his occupancy of the theatre always meant well filled benches and an appreciative audience. In common with his surgical colleagues he annually gave his course of clinical lectures to the senior students, and here the qualities already referred to that characterised him as a teacher and the immense fund of experience from which he could draw, sufficed to render this part of the curriculum most popular. He spoke as though he were at the bedside, and had the art of making his audience feel as though they saw the very case; in a word, the lectures were clinical in the true sense of the word, and not the classified descriptions that now so often stand in their stead.

It may be said of Bryant that he was a voluminous writer, but everything he wrote was of studied character and only put forward after due deliberation; he never hurriedly put pen to

paper. For long he was a constant attendant at the discussions of the Clinical and other Societies, often himself a contributor of a case or paper, and whether in this latter character or as a debater he was listened to by all with profound respect and interest. He never failed to make his points clear, and in debate was restrained and courteous in expressing himself. It goes without the saying that the highest public professional positions were filled in due course by Bryant, but an enumeration of some of them must suffice here. He for twenty-four years served on the Council of the Royal College of Surgeons, from the year 1880, and thrice was he elected its President (1896-1899), and in the many public functions in which, in that capacity, he was called upon to take part, he, by his fine presence, handsome features and courteous and genial manner, showed himself to be an unusually adequate representative of the surgical profession. For ten years he was examiner in surgery at the College, and its Hunterian Professor of Surgery in 1888-9; and he delivered the Hunterian oration in 1893 before a crowded theatre, being honoured on that occasion by the presence of his late Majesty King Edward VII., at that time Prince of Wales.

In due course he became President of the Medical Society of London (1872); of the Hunterian Society (1873); of the Clinical Society (1885); and of the Royal Medical and Chirurgical Society (1898-9); and for many years was the representative of the Royal College of Surgeons on the General Medical Council.

Abroad his name was as well known as in his own country, and in America he was held in the highest estimation, his text-book on Surgery being also published there; and in 1890 he was elected a member of the Société de Chirurgie de Paris.

Devoted as he was to his public work, with which he never let anything interfere, he yet was able to carry on one of the largest private practices in London, and his assistance was sought from far and near, many of his patients coming from foreign countries. How he got through his work was a matter of astonishment, but the methodical routine to which he bound everything that he did, combined with a strong constitution, enabled

him successfully, and with never a complaint of over-work, to live a life of incessant activity that would have been impossible in a weaker man. He numbered royalty amongst his patients, and held eventually the post of Surgeon-extraordinary to Queen Victoria and of Surgeon-in-ordinary to His late Majesty King Edward VII.

His high position was entirely of his own making. He began life, like other juniors often have to, with the burden of resident pupils, at the time that he lived in Finsbury Circus, but the necessity for this adjunct to income in his case did not last long, and soon the city home and the private hansom gave place to the west-end house and the then indispensable carriage and pair. His home life was a very happy one; he was a man of domestic habits, and not a club frequenter. He married early (1862), for, as he once remarked to the writer, he believed in the value of a wife in helping a man to win in the battle of life, and that it was well to marry betimes that each might help the other to their mutual interests. His wife was his constant companion, only predeceasing him by three years. Four sons and two daughters were the issue of the marriage.

The inexorable but wise Guy's rule that its staff should resign at the age of sixty caused Bryant's retirement, whilst still in the full tide of his professional work and successful practice. He never to the last day abated one of his official duties, and was fortunate in finding still an outlet for his untiring energy in work that he undertook at the Bolingbroke Hospital at Wandsworth, where he attended as regularly and with as great an interest as at his old hospital. Feeling himself still so young and active—for his age did not seem to have touched him at all—he felt his retirement from Guy's most keenly, but always said that it was right, for a longer tenure of office would be detrimental to the aspirations of the younger men.

The son of a Guy's man and the father of another, Bryant had the greatest affection for his Alma Mater, and he always acknowledged the deep debt of gratitude that he owed it for his professional success; whilst his colleagues knew full well

how much he himself had done in the Hospital and Medical School to help both to maintain and augment their already high position.

So occupied was he from morning to night that he found little opportunity for joining in social functions which to many are an important adjunct to professional success, though he was well fitted to shine in private life; but he did not fail to discharge, with dignity and satisfaction, the many demands society made upon him as President of the Royal College of Surgeons, when not only dinners have to be eaten, but speeches, which are open to the fullest criticism have to be made.

For very many years he had been a member of the oldest but one of the medical dining clubs in London, the United Hospitals Club, and even when past eighty years of age he was as regular an attendant at the meetings as he ever had been, and it was only quite towards the close of his life, when the weight of years told upon him, that he relinquished the pleasure of meeting his professional brethren of Guy's and St. Thomas's, whilst they, on their part never ceased to regret the absence of one whose cheery manner and happy face always helped to maintain the bright character of their gatherings.

Quietly, his life's work faithfully, usefully and conscientiously performed, at the advanced age of eighty-six, Thomas Bryant passed away on December 31st, 1914, and his interment in Kensal Green Cemetery was attended by many who both officially and privately desired to pay a last loving tribute to the memory of a great surgeon and a kind and valued friend.

With Mr. Bryant's death the last link that bound the modern school of surgery in Guy's with that of Astley Cooper, Key, and Birkett has been broken, and the fact that the methods and much of the teaching of these past masters in surgery are nowadays obsolete must not be allowed to lessen our appreciation of the great services they rendered in their generation. Bryant, as in the case of his own masters, could not fully apply to his art the scientific knowledge of the days in which he lived and worked, and where that knowledge is only unfolded in the latter part of

a man's professional career, when his modes of thought, investigation and practice are already crystallised into shape, it cannot be expected that he can be as sympathetic with the newer views and methods as the rising generation of surgeons, nor even able to appraise their line of thought and practice at their full value. Bryant worked hard to keep himself *au courant* with the times, but the ability to put into practice every scientific nicety that crops up is more than difficult for one whose training was of a different type, though as thorough and as sound as it was possible to obtain at the time of his professional education. Regarded in this light, the hesitancy that a man of Bryant's standing and ability showed to adopt and, perhaps, fully grasp the many new discoveries that crowded upon him in his latter years can easily be explained and respected, and the memory is then left of one who in his day strove unceasingly for the advancement of knowledge and the good of his fellow-creatures.

A BIOGRAPHICAL NOTE.

Eldest son of the late Mr. Thomas Egerton Bryant, M.R.C.S., an intimate friend of Sir Astley Cooper, was born in Kennington on May 20th, 1828.

Educated at King's College. 1846, entered Guy's Hospital; 1848, Dresser to Mr. Key; 1849, M.R.C.S. Took the Fellowship by Examination on May 12th, 1853; 1857, appointed Assistant Surgeon; 1859, Lecturer on Operative Surgery; 1864, Lettsomian Lecturer; 1871, Full Surgeon; 1875, Lecturer on Surgery; 1888, Consulting Surgeon.

1872, President of the Medical Society of London; 1873, President of the Hunterian Society; 1880, Member of Council of the Royal College of Surgeons; re-elected 1888, 1896, resigning in 1904.

1882-1892, Member of Court of Examiners as well as representative of that Court on the Board of Examiners in Dental Surgery in 1887. 1885, President of the Clinical Society of London; 1887, he was honoured by the Royal University of Ireland with its honorary M.Ch. degree; 1888. Hunterian Professor of Surgery; 1889, Bradshaw Lecturer; 1890-1892. President of the Royal College of Surgeons; 1891-1904, Representative of the College on the General Medical Council; during part of this time acted as Hon. Treasurer.

In 1892 the University of Dublin gave him its Honorary M.D., while the Royal College of Surgeons in Ireland conferred on him its Honorary Fellowship.

On the occasion, in 1893, of the centenary of the death of John Hunter, Bryant had the honour of delivering the Hunterian Oration in the presence of their Royal Highnesses the Prince of Wales, afterwards H.M. King Edward VII., and the Duke of York, now His Majesty George V.

1896, appointed Surgeon-extraordinary to Queen Victoria, and later Surgeon-in-ordinary to H.M. King Edward VII.

1895, Vice-President of the Section of Surgery at the Annual Meeting of the British Medical Association in London; 1898, President of the Royal Medical and Chirurgical Society

Member and first Honorary Secretary of the first Committee which sat in London (1863) to report on Chloroform.

1862, married Adelaide Louisa, daughter of Mr. Benjamin Waldrond, whom he survived three years, and by whom he had four sons and three daughters.

Died December 31st, 1914.

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COMPILED BY J. H. E. WINSTON, Wills Librarian.

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CHARLATANS AND "MIRACLES."

By

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THE idea of writing this paper came to me in consequence of a conversation I had with the old friend whose interview with Count W—— forms an early part of it.

I wish it to be distinctly understood that I do not for one moment blame Count W—— or any of his kind; it is the people who support them who are to blame, and who deserve to be exploited and victimised to any extent. The charlatans are clever enough to seize their opportunity and make the most of it. One circumstance which I feel I must mention, even at the risk of being unpopular with the rank and file of the profession, which tends to help the charlatan or to send patients to regular practitioners abroad is the custom which exists in this country of sending patients to a specialist simply for consultation. In a very large majority of cases, especially where only general medicine and surgery are concerned, it works extremely well, but in specialisms the case is different.

Quite a number of patients require special manipulations which may be difficult even to a fairly dexterous and educated hand. How are these to be carried out by a man who has no knowledge of them, and even if he had, has not the necessary dexterity and practice required for their successful employment? There are some who are perfectly competent, but the majority are not, and how should they be? If all were, the need for the consultant and specialist would cease.

Without going into unnecessary detail I can explain what I mean by giving notes of a single case.

A lady, æt. 23, first consulted me July 15th, 1901. She was suffering from trachoma. I gave her a prescription for sulphate of copper drops, a piece of green-stone, and a letter to her doctor with instructions how it was to be used. She returned to me in October. She had been in two or three different places and had suffered most acutely at the hands of more than one medical man, and her descriptions of their treatment were rather funny. None of them could ever evert her lids without great difficulty and after several attempts, and when they had succeeded, they applied the green-stone in a very perfunctory way, giving a maximum of pain and a minimum of benefit. One man in particular, in spite of the suffering he caused, appealed to her sense of humour. He used to seat her in an arm-chair, place himself at the back, and in order to get exactly at the right height, stand on books, which generally slipped at the critical moment, and the whole performance had to begin over again.

The lady said, "What am I to do?" I said, "Come and stay in London and I will treat you myself." She could not manage to stay continuously, so we made a compromise, and she arranged to come for a fortnight at a time and at as short intervals as possible.

This she did. I treated her with applications of green-stone and mitigated nitrate of silver, and gave her at different times drops of copper sulphate, zinc sulphate, chinosol, formalin, and boracic lotion. On three or four occasions I scraped her lids or cut off exuberant granulations with scissors. In July, 1902, she was quite well, and has continued so ever since. What would have been the good of leaving this lady to the tender mercies of her book pedestal practitioner?

Another reason which it would not be fair to leave unnoticed exists amongst ourselves. I have alluded to it at the end of this paper: it is an excess of bluntness and want of sympathy. I am, perhaps, myself one of the worst offenders in this way,

but I take it that it is bad policy (unless one is asked point blank the question, Is there any hope?) to send a patient away entirely hopeless. If we do he will almost certainly go to some charlatan, get into the hands of one of the class mentioned at the end of this paper, or go abroad to be treated by some foreign celebrity.

And again, none of us are infallible, and we occasionally make most woeful blunders. If the blunders happen to be made by a man who for the time being is in the eye of the public, the patient (especially if he belongs to the upper class of society) at once makes up his mind that there is no appeal in England, and if he does not consult a charlatan here he goes abroad to consult a foreigner, who generally manages to keep him under treatment for an indefinite time.

A Visit to Count W——

An old friend and for many years patient of mine suffered from cataract in one eye, which he was quite content to leave alone. Like all others who have any chronic eye trouble and have been given an honest opinion, he was persuaded to consult one of the wonderful, or, as their admirers believe them to be, supernatural foreign charlatans, who from time to time honour us with a visit.

He found the Count occupying a suite of apartments at the C— — Hotel, consisting of consulting room, waiting room, secretary's room, optician's room, and another room. He was interviewed by a secretary, who was kind enough to give him an appointment for a consultation two days later. He kept the appointment, and was shown into the waiting room, which was crowded with well-dressed and presumably fairly intelligent people. Some were merely waiting, but others were having applications made to their eyes by nurses, whilst others, again, were holding pledgets of cotton wool soaked in drops to their eyes! What a study in psychology! Fancy a crowd of people belonging to the upper classes of society waiting like a lot of sheep at dipping time. It looks as if their mental capacity was not far above that of the sheep.

My friend was not dipped; but in due course was admitted to the presence of the Count, where his eyes were very thoroughly examined. He was told by the Count that he had cataract, but that on no account was he to be operated on; further, that he, the Count, could cure it by his special treatment. The result of the consultation was that the patient was ordered three pairs of glasses, the cheapest of which was to cost £1 1s. (the optician was on the spot). My friend looked at the glasses and admired them very much, but he wasn't taking any just then, nor has he decided to do so since. He was given a paper with something written on it (not a prescription), and sent to a particular chemist, where he obtained a bottle of drops, price 3s. 6d., including a dropper. The consultation fee was £1 1s. My friend paid two visits and got off with £2 2s., but he told me he saw two ladies draw cheques for over £100 each for treatment. The drops he got were brown in colour, their action negative, except that they stained cotton or linen fabrics. I called on the chemist, and he told me there were several kinds of drops which were sent to him; he knew nothing of their composition, they were "made in Germany." My friend used the drops conscientiously for some weeks without result.

Count W——, it appears, cannot now practise in England. He wrote to my friend—as, no doubt, he has to all his patients—telling him that he had bought a castle in Germany, where he proposed to carry on his practice. I wonder if his dupes will follow him there to get their periodical dipping. My friend will not be amongst the number, he has had enough of such practitioners.

But to the Count's credit be it said, that he either believes in his treatment or he is a thorough sportsman and certainly has the courage of his opinions, for I have been told by a colleague that he made the Count a bet of £100 that he did not cure a case of cataract by his treatment in nine months, and at the end of that time he received the Count's cheque for one hundred pounds.

My friend tells me that he has been known to have bad sight now about 18 or 19 years, during which time he has been implored to consult numbers of these men. Each has been described to him as being more wonderful than the last, but up to the present, in spite of pressure put on him by people he meets, and in spite of about 90 per cent. of them being women, he has only fallen twice.

The last wonder to whom he was recommended is a disciple of the great Dr. Fourniet, who at one time made himself a great nuisance to members of our profession, notably to Sir Anderson Crichett. He once wrote to me. I took no notice. He wrote again, demanding an explanation of my apparent neglect. I wrote back :—

Dear Sir,

Come and see me to-morrow at 1.30.

Yours faithfully,

CHARLES HIGGENS.

He did not come. Perhaps he was wise. I was disappointed. I only remember one of my patients who ever owned to having consulted Dr. Fourniet. He was a gentleman, æt. 64, who consulted me in March, 1902. His right eye could read Jaeger 14 a letter at a time at two inches, and vision was 6/60 C. +2cyl. 12sph., axis 105°.

There was extensive choroidal atrophy. The left eye had bare perception of light. The lens was partially opaque and there was much vitreous opacity. I put down as a query that there was retinal detachment. I saw him again in April of the same year; after that I lost sight of him professionally, though I met him from time to time and heard from mutual friends that he was under treatment by the latest wonder, the great Dr. Fourniet, who was curing him with glasses. I watched him with interest, but never mentioned his eyes to him.

In August, 1905, he returned to me and confessed his sins. He was the better off by about thirty pounds worth of glasses which the wonderful Dr. Fourniet had ordered him. They were

of different colours, and somewhat different foci, but had one property in common, he could see nothing with any of them: without, he could just find his way about. His right lens had become partially opaque. I advised him on the off chance of getting some improvement of sight to have the left lens extracted, which I did, and was agreeably surprised to find the eye improved so much that he could read Jaeger 4 with +4, and see to go about without glasses.

Another of the same class of practitioner of whose methods I have some knowledge, imparted to me by a victim, is an American lady doctor. The case was as follows :—

In July, 1890, a lady consulted me. She was suffering from chronic blepharitis and refractive troubles, and she has been to see me from time to time since, her last visit being in July, 1909. On two or three occasions she told me she wished she could get her husband to come to me; that he was under treatment by a lady doctor for cataract, and, further, she was ashamed to say he was the only man amongst her patients. I told her I doubted if the lady would know a cataract if she saw one, and I gave her one newly extracted to show the great doctor when next she accompanied her husband to see her, which she did on the same day. The lady, as I expected, had not the least idea that it was what she professed to cure—a cataract; was furious when told, and I am not certain that she did not become abusive, if not aggressive. However, the display of knowledge, or rather the want of it, on the part of the doctor had the desired effect, and the husband was brought to see me in April, 1892.

He was 45 years of age, and his eyes had been inflamed since quite early in life. When I saw him it was obviously a case of interstitial keratitis of very long standing, there was no active inflammation, but much corneal opacity, vitreous opacity, and myopia, with vision, right eye, Jaeger 16 at 8 inches, left eye, Jaeger 1 at 6 inches, 6/18 C. —7. His pupils were widely dilated, as I supposed, from a mydriatic.

The lady doctor was "curing him of cataract." The lenses were the only part of the media which, as far as could be made

out, retained their transparency. I learned that the treatment consisted in the application of drops, presumably atropine, then careful bandaging of the eyes, with strict injunctions not to remove the bandage until the next visit, in two days; and it was pointed out that there was great risk in removing the bandage, because the effect of the drops was to expose the optic nerve. At the next visit the bandage was removed, the eyes opened, and the patient assured that he was much improved and could see better, which no doubt was true, after having been kept in the dark for two days and then exposed to light with pupils very much wider than when the bandage was applied. But then came the test of the treatment: the patient and those who accompanied him were shown something (possibly dried gum) which had been produced from under the operator's nails or other convenient hiding place, and they were told "this much of the cataract had come away since the bandage was applied." Wonderful what an impression must have been made! But something much more wonderful happened in my consulting room. This gentleman said to me, "Will she do my eyes any harm?" I said, "No, as long as you do not allow her to use anything in the way of an instrument near your eyes; if you do you may get a cataract which may in the course of months be to some extent cured." Now for the wonder. He said, "I shall continue to go to see her for the sake of a quiet life; my mother and sister have the greatest confidence in her and will never let me have a minute's peace if I leave her." These were two of the doctor's female admirers and touts.

The question must present itself to every well balanced mind, how do these people get such an ascendency over other men and women as to be able to influence them as they do? Their dupes push, advertise them, and cram them down other people's throats in a way that is disgusting to ordinary beings, and they do it in perfectly good faith. If they stood in with the rogues they advertise it would appeal to one's business instinct, but they do not; they believe that they are doing good to suffering humanity by trying to persuade people to put themselves under the care

of their latest wonder. I could multiply instances almost indefinitely where people have been implored by someone who has a pet crank to put themselves, or their children, or anyone who is dependent on them, under the treatment of the special individual; but one instance will suffice:—

In 1897 I was attending a young lady who was dying of meningitis. One morning her father handed me a letter, with the remark, "I know that woman, she is not mad." The letter, which extended to two sheets of writing paper, was one long prayer, I might almost call it, that he would send for a Mrs. Someone, a Christian Scientist, who had just left a case and was providentially at liberty, to come and sit by his daughter and pray her back to life and health. When I had read the letter, I said, "Well, are you going to send for her? I have no objection." He replied, "What do you think?" I said, "I don't think." The lady's services were not asked for.

It seems to me that these practitioners not only humbug their dupes, but make them believe what they think fit. My friend noticed this in Count W——. On his second visit the Count assured him that he had greatly improved already by his treatment, and in various ways tried to make him believe it, but being perfectly certain that his eye was in exactly the same condition as it had been for years, he was not favourably impressed. On the contrary, he lost whatever little faith he might have had in the man, and was convinced that he was a fraud; he did not go near him again.

The secret of their success is simply the domination of a weak will by a stronger one, and it is in order to show this that I am describing what I call "miracles." The will need not be a very strong one, especially among the class of people who provide the charlatans' happy hunting ground: they are the "idle rich" who are always ready to run after any new sensation, especially if it has a smack of mystery about it; they are too utterly foolish in matters medical, are gregarious, and follow their leaders and each other. They do not like a straightforward honest opinion given them by a straighforward honest man, and

rather resent it. Very few ordinary, hard-headed, professional or business men or women are ever found in the consulting room of the charlatan; he would starve if he had to depend on such for his living.

Whilst I am on the subject of charlatanism I will call attention to some very doubtful practices on the part of members of our profession, and I do not think I can do so more satisfactorily than by relating the adventures of another very old friend, and for many years a patient. My friend's experiences can hardly be given in his own words, for his description was decidedly florid, and I could not well publish the whole of the adjectives he used. Here are the notes of his case :—

S. F., æt. 39, first consulted me January 31st, 1890. I had known him for some years previously, but had not seen him professionally. He had severe iritis in left eye. Had syphilis five years before. I treated him with atropine, blisters to the temple, and hyd. perchlor., which he took for many months. He entirely recovered from the iritis, and I did not see him again for about five years.

December 31st, 1895, he consulted me again. He complained that the sight of the left eye was defective. Right eye vision 6/9, left eye, 6/18, not improved by glasses.; left eye pupil moderately contracted, no reflex, optic disc pale; right eye pupil sluggish. Left eye colour sense very defective, sees red as brown. Ordered Pil. Hyd. Iod. Rub., gr. 1/16, three times a day.

January 26th, 1896, there was no change.

March 19th complained that pills purged him. Ordered mercurial inunctions.

April 10th, right eye vision 6/9, left eye 6/24. Mercurial inunction continued for twenty days.

On June 8th the treatment was discontinued.

June 30th, right eye 6/12, left eye 6/36.

I did not see him again till March 5th, 1897. The right eye remained the same, but the vision of the left had fallen below

6/60, and it could make out none of the Jaeger types. Another course of mercurial inunctions was prescribed.

April 5th, 1897, there was slight improvement.

July 28th, 1898, right eye 6/18, left eye perception of light only. The discs had now become very pale.

I have no note of the case after this till April 11th, 1901. The vision of the right eye had fallen to 6/60; the left still had perception of light. The discs were very white. Another course of Pil. Hyd. was ordered, which was altered to Pil. Hyd. Iod. Rub. on May 15th.

I did not see Mr. F. again for five years, but heard from his medical man, who had treated him in conjunction with me during the whole of the eleven years over which these notes extend, that he had said he was kept waiting so long on the last occasion he visited me, in spite of having made an appointment, that he should not go again. I asked to whom he had been; the medical man did not know. I said, "I will make a bet he has been going the rounds, seeing everyone and anyone."

On May 10th, 1906, he came to see me. He said, "Well, Higgens, I have come back to you. There are only two honest ophthalmic surgeons in London and one of them is John Couper, the rest are a lot of rogues." I said, "That is a rather sweeping assertion," and asked where he had been. He mentioned the names of two or three whom he had consulted, and I found that they had given him perfectly honest opinions. There was one whose name I mentioned particularly, and Mr. F. said, "He is a —— deal too honest; he said I was going blind and it was no use my coming again." That, though perfectly correct, was, perhaps, straining honesty a point or two.

He then proceeded to give his experiences of three others who, to say the least of it, erred somewhat in the opposite direction.

The first, after having examined his eyes, said, "I see what is the matter, and I can cure you by my special treatment." He then proceeded to explain that the patient must come to him

three times a week for the treatment, and that he was to be very particular to come at 10 on Monday, 11 on Wednesday, and 12 on Friday, and it was at the same time pointed out that not keeping time or missing a day would prejudice the success of the treatment.

Mr. F. said the treatment was electrical of some kind, and that each application took about ten minutes. He attended regularly three times a week and spent three guineas a week.

One day the doctor told him he was better and need not come for three weeks. Mr. F. was rather surprised at being suddenly told not to come for that length of time, when such particular instructions had been given him as to the importance of attending regularly. Mr. F. then told me that he had found out that the —— was going to be married and would be away three weeks. He said, "That was enough for me, I did not call again."

He then consulted No. 2, who examined his eyes and then recommended a course of treatment similar in some respects to that of No. 1, except that it was to be by injections (he believed of strychnine) instead of electricity. He said, "Thank you, I know that game," paid his fee and left.

He next consulted No. 3 who, after examination, said, "You have glaucoma and cataract. It is necessary that you should have four operations, which will cost one hundred guineas each. The first two must be performed at once, and I will make arrangements for you to go to my home without delay." He did not fall in with this proposal, but came to the conclusion that before he decided to be relieved of 400 guineas he would come and ask me about it, and, as already stated, came to see me on May 10th, 1906. On that date his right eye could read Jaeger 20 at 2 inches. The left had some perception of light. The lenses, with the exception of a few short opaque lines (probably congenital) at regular intervals around the periphery, were perfectly clear, the tension was normal, but the discs were white as egg shell. I pointed out to him that if he submitted to the first two operations, he would certainly require the second pair,

and that after all had been completed, with luck, he might see nearly as well with glasses as he could without before any operation was performed. I said a good deal more which I need not repeat, and asked him to go and see the man again and give him my opinion of him, but he said he did not care to do so.

I then said, "Will you write an account of your interview with Mr. —— and let me have it? If you will, I will write my report of your present condition, and get it verified by six ophthalmic surgeons whose names I will give you, and will you publish the lot in two or three of the leading daily papers?". He said he would, but returned again in a few days and said, "I am an old man, and do not care about getting into a law suit." I told him the man would never prosecute him, and that I should be only too glad if I could get him to prosecute me for my share in the business. However, I could not get him to do anything, and did not quite see how I could do it myself, so had to let it drop.

So-called "Miracles."

The following are notes of cases which, I take it, if treated and properly exploited by a charlatan and by his touts, would give him a reputation that would increase like a snowball until it arrived at enormous dimensions.

Miracle No. 1.—Mrs. R., a widow, æt. 43, consulted me on July 4th, 1885, sent by Mr. Sargant, of Northfleet. She presented the most extraordinary appearance: she was covered from head to foot in a garment of some black material and resembled an enormous black extinguisher. Guided by a gentleman who accompanied her she progressed towards me, and that was the only indication I had as to which was the front and which was the back of her. I guided her to a chair, and when she was comfortably settled she gave me the following history: Her eyes had become inflamed ten months before, but improved in two months; two months later she had a second attack which had now lasted six months. For some reason or other, which I

cannot account for, I made up my mind directly she entered my room that she was a fraud, and made the note, "hysteria." I set to work to remove her coverings, first the aforesaid black garment, then a very large black crepe veil which came nearly to her waist, next a black shade with green lining; her forehead was filthily dirty and stained green by the dye from the shade; then came a black bandage and beneath this a pair of the darkest smoke protectors. When I removed these she put her hands up to her eyes. I knocked them down rather roughly, and she said, "Oh, I can't bear the light." I said, "Yes you can, look at me at once," which she did. I said, "What do you think of me?" She simply gazed in blank astonishment and said nothing, and the gentleman who came with her looked more astonished than she did.

That was all; the cure was complete. Her eyes were perfectly normal, her vision was 6/6 and Jaeger 1.

Then she began to doubt, and said, "It is the light in your room." I said, "No, it is not; come out in the street." I took her by the arm and led her to my front door, and made her read the numbers 47 and 45 opposite. I then took her back to my consulting room, and told her that she was not to shut her eyes or dream of the light hurting them, that it never would again, that she was to go out at once, walk down Regent Street and look in every shop window, that she was to leave all the things I had removed with me, except the protectors, which I said she might wear till she got home and after that never put them on again. The gentleman looked at me as if unable to make up his mind whether I was an angel or a devil. He said, "What have you done?" I said, "Cured Mrs. R." He said, "But she has been to most of the leading oculists in London, why couldn't they cure her?" I said, "Because they believed there was something the matter and treated her for it; I have simply dominated her will, or rather want of will, and made her see that there is nothing the matter." He paid my fee and said he never parted with two guineas with greater pleasure.

On July 22nd, 1885, I received the following letter from Mrs. R., which speaks for itself :—

91, High Street,
Ilfracombe,
July 21*st*, 1885.

DEAR SIR,

I feel that I must write and try and thank you for your marvellous cure of my eyes; it was indeed a fortunate thing for me that I was recommended to come to you, or I dare-say I should have been another six months in a dark room. I was so surprised that I did not ask you anything that I intended to have asked, and shall never forget how beautiful the air felt on my face walking to the station. I have been here ten days and am better for the change and can now write two letters without making my eyes ache. Of course, the light seemed very bright for a few days, but I would not give way to the feeling. It is so delightful to be able to look about again and to read.

Thanking you for your kindness and trouble in sending the veil, etc., and with kind regards,

I remain,
Yours truly,
C. C. R.

P.S.—The cream here is so nice that I thought, perhaps, you might like some, and have sent it by this post, and hope the journey by train will not spoil it.

I heard of her from time to time, but never saw her again for 25 or 26 years. About 1909 she came to see me. She had been married some years, and brought her second husband to consult me. She looked extremely well, and told me she had had no trouble with her eyes since she came to me 25 years before. I have forgotten her husband's name and made no note of her call, so cannot fix the date exactly. He came to see me two or three times, and the last time I saw him he told me his wife had died quite recently, I think from cerebral hæmorrhage.

Miracle No. 2.—Miss A. P., æt. 13, was brought by her father to see me on July 20th, 1885. Her history was that she had come home from school quite blind in her left eye. Directly I saw her I felt certain that she was a fraud, and after writing the date and her name and age, made the note, "Hysteria, blind in left eye."

I sat her in a chair with her back to the window, put the trial frame on with a shade on the left side and a +2 D (or 20 as it was in those days) on the right. I changed the shade and glass about five or six times, and then left the shade in front of the right eye and the glass in front of the left. When she had read through the whole paragraph of Jaeger 1, I pointed out to her that she had done it with her left eye; she was quite sure she had not, so I said, "Cover your left eye up with your hand." She did so, and, of course, could see nothing. I said, "Now uncover it." As soon as she did so she could read. Again I pointed out that she was reading with her left eye; she said, "So I am, and it was blind." The cure was complete. I said, "Mind, you are not to go blind any more"; and she never did.

Her troubles were not imaginary, for she had 2 D of hypermetropia in each eye and said she could see no letters at 6m. with the left eye, the vision was only 6/18 with the right, but with +2 vision was 6/6 in each eye. I told her father, who had been gazing at me in wonder for some time, to take her home, let her run wild for a couple of months, then go back to school with glasses for reading and not allow her to be overworked.

Then came an astonishing result of the miracle. The father said, "What have you done?" I said, "Cured your girl by simply showing her that there was nothing the matter." He would not have that at any price; he said, "She was blind when she came home, so she was when she came into this room; you must have done something." I did not argue with him. His next act showed how fully he was convinced that I had some miraculous power, for he showed me a large, fatty tumour

on the back of his neck and asked me to cure it for him. I said, "It is a general surgeon's job, but I will remove it for you if you like." His reply was, "No, I don't mean that; I want you to cure it in the same way you have my daughter's eye." I said it was quite beyond me, but he obviously did not believe me, and went away rather hurt because I would not try.

I saw Mr. P. some years later. He then had albuminuric retinitis; he was convinced that I could cure it. I told him I would do my best, and wrote to his medical man telling him it was a case of Bright, of the treatment of which he probably knew more than I did.

Some years after the daughter came to see me again. She was married, and must have been a treat to her husband, for her whole history since I saw her before was neurotic. She said she had never gone blind again. I found she had hypermetropic astigmatism and set her up with glasses. She told me her father had died not long after I saw him.

Miracle No. 3.—J. M. S., æt. 14, was brought to me December 31st, 1883. Right eye vision less than 6/60 C +20 (2 D) 6/18; ordered +2 for all purposes. He was a queer sort of boy, and I was told was subject to fainting fits which were said to be brought on by reading. I found he was using concave glasses for that purpose, so there may have been something in it.

On February 28th, 1883, he had had no fits since using the glasses; his vision was: right, 6/60 C +2 6/6; left, 6/60 C +2 6/18.

I did not see the boy again till May 6th, 1885, when he was brought to me by his mother who was in great distress. I was told that two days before his sight had become suddenly dim, that he was blind in a few hours, and had remained so since. He had greatly alarmed his parents by coming into their bedroom in the night, feeling his way, to tell them that he had gone blind. He was pushed into my room, feeling his way with his hands; he over-acted the part so much that I was perfectly certain he was a fraud, and my experience of him eighteen months before did not predispose me in his favour. I

told him to sit down and then to look at me, which he did, and said he could not see me, but both his pupils reacted to light. I told him he could see perfectly well, and that there was nothing the matter with his sight, and in a few minutes he owned that he could see quite well. That was all. I told him not to go blind again. His mother was much struck by what occurred, and wanted to know what I had done. I said, "Nothing, but shown him that he is shamming and that I know it." She did not believe me, and for years thought that I had performed a miracle of some kind on him. I saw him again a year later; he had had no relapse, and his vision was 6/6 with each eye, but he still had to use +2 for reading. I am not sure whether this was a case of male hysteria, or one of wilful malingering; the only thing against the latter theory is that as far as I know he had nothing to get by it.

Miracle No. 4.—A young lady of about 19 or 20 was brought to me about two years ago. Unfortunately I cannot remember her name, so cannot refer to my notes, but in this case I can quite well trust my memory.

I was given notice of her advent by a telegram from her father telling me that his daughter had gone blind in both eyes during the night. I wired him to bring her at once, and she was brought about two hours after. As soon as I had read the telegram I made up my mind that the case was a fraud, and was quite prepared to treat it as such. The girl was guided into my room by her father pushing her from behind. She felt her way with her feet, extended both arms and hands in front of her and kept her eyes patially closed. I placed a chair for her in front of the window and guided her to it. Directly I touched her I felt certain that she could guide herself. I sat down in front of her and asked her a few questions as to how she lost her sight, and what was the condition of it before she went blind, and at the same time took some wooden matches out of a box and began breaking them. At each crack she winced, and after about half a dozen got quite nervous; at last she summoned up sufficient courage to ask me what I was doing.

I said, "Only making props to keep your eyelids open with. She asked if they would hurt. I told her they had very jagged and rough ends, and in the same breath said, "Open your eyes wide at once and look at me." She obeyed immediately. I said, "You have begun to see again." She said, "Oh yes, I can see quite well." I said, "Don't go blind again." That was all. I tested her vision; it was normal. She said she was so much obliged to me for curing her. Her father seemed too astonished to say anything. I pointed out to him that his daughter was cured, and said, "Bring her back if her sight goes wrong again." He said, "That I will," paid my fee and left. I have never heard any more about her.

A CASE OF FRACTURE OF THE BASE OF THE SKULL WHICH OCCURRED ON FEBRUARY 19. 1874.

WITH A POST-MORTEM EXAMINATION BY THE LATE DR. MOXON AND A SKETCH MADE AT THE TIME.

By

MR. R. CLEMENT LUCAS.

IT has been stated that cases properly reported retain their value at whatever age they may be recorded. In turning out an old drawer I found this case which occurred in 1874, with a drawing by myself of the condition of the skull. The case was admitted under Mr. John Birkett in Accident Ward on February 19th, 1874, the history being that the man had fallen 28 feet on to his head. The patient was admitted with coma, his left pupil being fixed and dilated, whilst his right was minutely contracted. He is said to have moved himself about, giving evidence of the absence of paralysis of limbs. He died a few hours afterwards, and the *post-mortem* was made by Dr. Moxon nineteen and a half hours later. His report runs as follows :—

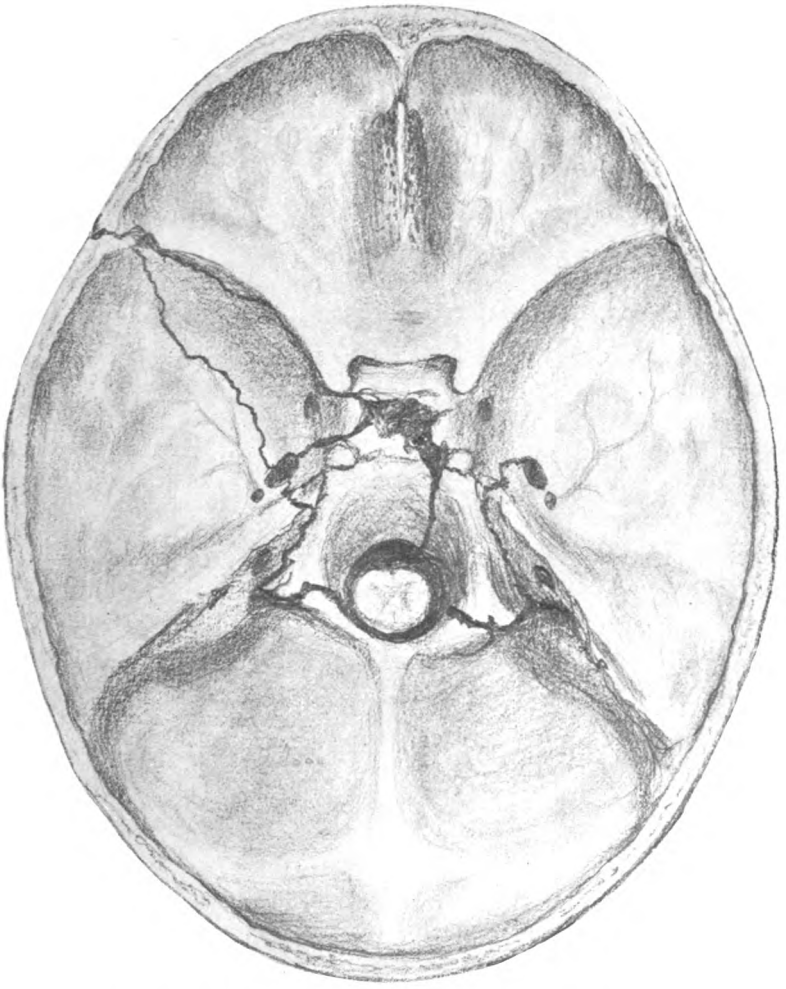
Alfred Gore, aged 47, atmospheric condition cold and dry: signs of decomposition, none; rigor mortis, slight; hypostasis, much; previous history, none, except that he had fallen 28 feet on to his head. The left eyelids both much swollen, and on admission no blood under inner part of the conjunctiva, a little at the outer angle, with much chemosis of clear fluid, ? "cere-

bro-spinal." Physiognomy, livid; nourishment, muscular; left eye swollen and blue from extravasated blood. Head, much fluid extravasated under the scalp near left side of the head, the vault of skull fractured from near to median line above, or rather it was fractured near the outer angle of the left orbit, the fracture running into that cavity and then upwards towards the vault a little in front of the middle meningeal branch of artery and then sub-dividing into two narrow fissures. The skull was broken at the base as sketched, both posterior clinoid processes were broken off and a transverse fissure existed through the sella turcica. A vertical split ran from this towards the foramen magnum, and the anterior part of the foramen magnum was fractured on both sides and forced out of place so as to rupture the lateral sinus on both sides, thence the fracture extended through the petrous portions, temporal at the apex and near to it, and on left side extended through middle fossa into the sphenoidal fissure. The orbit was full of blood; blood was also extravasated between periosteum of orbit and its bone. The brain was also lacerated for three lines depth close to the bifurcation of the Sylvian fissure on the left side. There was no bruising of the brain elsewhere. Blood occupied the arachnoid cavity, but not to any excessive amount. Blood was also covering thinly the brain convolutions beneath the arachnoid. Bloody fluid was found also in the lateral ventricle, and a smaller clot occupied the fourth ventricle, the iter, and third ventricle. There was extravasated blood into the pons varolii at its anterior part, most on to the left side. Lungs, bulky tubes contained frothy blood. Heart, 15 oz., healthy. Liver, 16 oz., healthy. Spleen, 7 oz., healthy and soft. Kidneys, 10 oz., healthy.

The admirable post-mortem examination made by the late Dr. Moxon, read by the light of my illustration, leaves little to comment on.

It is evident that the main injury was caused by the forcing upwards of the basilar process by the spinal column beneath. The fracture of the vault running upwards from the left orbit and dividing into two small crevices came from the same source

A Case of Fracture of the Base of the Skull which occurred on February 19th, 1874.



FRACTURE OF THE BASE OF THE SKULL.

A. G., æt. 47, fell 28 feet on to his head. Coma till death within 24 hours. Rupture of both lateral sinuses.

Drawn by R. CLEMENT LUCAS, *February 20th, 1874.*

and not from the direct blow on the skull. The fracture of both posterior clinoid processes were due probably to the forcing upwards of the posterior attachments of the tentorium cerebelli. There is a main crushing of the sella turcica from which a fracture runs vertically backwards across the basilar process to the foramen magnum. Two lateral fractures run outward from the foramen magnum to the position of the lateral sinus on either side. From this point the fractures run forward to the apex of the temporal bone and then cross outwards to the foramen ovale; that on the right ceases at that spot.

On the left side the fracture is continued along the outer margin of the sphenoid bone and joins with another from the sella turcica at the sphenoidal sinus.

The position of the foramina at the base of the skull are interesting as determining the course of the fracture, the latter seeking the points of least resistance.

A CASE OF TUMOUR OF THE LEFT CEREBELLO-PONTINE ANGLE.

By

W. JOHNSON, M.D., and W. M. MOLLISON, M.C.

The patient was a boy (F. W.), aged 16 years, who had never had any serious illness. Early in 1912 it was noticed that the left eye appeared somewhat staring and that it could not be completely closed. For two years previous to this, however, it had been remarked that the left side of the face was weaker than the right. About the time of the occurrence of the facial weakness, deafness on the left side also became apparent. In May, 1913, the patient suffered from headaches, which were worse in the morning and passed off during the day. He also vomited every day immediately after breakfast. Soon after this he was operated on, and these symptoms have not since recurred. In July, 1913, he showed a definite tendency to fall over towards the left side when walking.

On examination of the nervous system in January, 1914, the condition was as follows:—There is complete paralysis of the face on the left side, and the muscles show early reaction of degeneration. Both pupils react to light and accommodation. There is some weakness of the left external rectus, and nystagmus is well marked, the coarse, slow movement being to the left. No definite anæsthesia can be made out, but there is blunting of sensation on the left side of the face. Taste is lost on the anterior portion of the tongue. With the exception that the left masseter muscle is weaker than the right, it is not possible to

demonstrate any motor weakness. The knee-jerks are, however, exaggerated, there is a tendency to ankle-clonus, and the plantar reflex is extensor on the right side. That on the left is indeterminate. Slight inco-ordination of movement is present in the left arm. Walking is a little unsteady and the patient walks on a wider base than normal. Adiadochokinesis is well seen in the left arm. There has been no sphincter trouble.

Clinically, therefore, there is evidence of pressure on the fifth, sixth, seventh, and eighth cranial nerves on the left side, on the left lateral lobe of the cerebellum, and on the left pyramidal tract in the pons (*i.e.*, those pyramidal fibres, which later cross and control the right lower limb). Such an association of symptoms would be produced by a tumour in the left cerebello-pontine angle.

Mr. Ormond examined the eyes. Vision in the right 6/9, in the left 6/24. There is papillœdema on both sides, more marked on the left. The left globe is slightly dropped.

Examination of the ears : Both membranes are normal. With the "noise apparatus" there is total deafness in the left ear; the right is normal. The patient has no vertigo, nor has he ever had any.

The nystagmus is horizontal and of a slow, coarse character when the patient looks to the left, about 80 per minute and very irregular. It is quick and fine when the eyes are directed to the right and about 180 per minute. There is a spontaneous pointing error to the right with the left arm.

Functional examination of the labyrinths : Caloric test—After prolonged syringing of the right ear, with the head erect, with ice-cold water, no alteration was produced in the spontaneous nystagmus. The same result was obtained after syringing the left ear. The only effect of syringing the right (the sound side) was that the patient had a tendency to fall to the right.

Rotation : The right labyrinth reacts, the left does not. This fact was elicited, not by observing nystagmus, as this is impossible, but by noticing that the patient had distinct tendency to fall (to the right?) after turning in a counter-clockwise direc-

tion, and by noting the resulting errors in the pointing tests. After ten counter-clockwise turns (*i.e.*, to the left) there was with the *right* arm a pointing error to the left, while the spontaneous error with the *left* arm tended to disappear. After ten clockwise turns there was no appreciable pointing error with the right arm, and no change in the spontaneous error with the left; but after twenty turns there was a slight error to the right with the right arm and distinct increase in the spontaneous error with the left. The results of these tests are compatible with a left-sided cerebellar lesion.

Operation.—On May 1st the patient was anæsthetised, ether being given by insufflation into the trachea. The mastoid process was opened and the dura mater of the posterior cranial fossa exposed as extensively as possible internal to the lateral sinus. No pulsation was to be seen. This constituted the first stage of the operation; the wound was dressed and the boy returned to bed. One week later the patient was again anæsthetised. An incision was made into the dura mater previously exposed. Fluid at once escaped under considerable pressure, and the dura began to pulsate strongly; about an ounce of fluid was allowed to escape. It was seen that the fluid was escaping from a cavity lying apparently between the pia and arachnoid. At the bottom of the "cyst" the cerebellum could be seen and its leaves appeared flattened, but normal. It was concluded that the symptoms were all due to the pressure of this cyst, and nothing further was done. A rubber tube was placed in the cyst to drain it and the wound dressed.

The result of the operation was at first good; the patient felt well and said he could see better. Drainage of fluid from the cyst was very free, and after about six days, packing was resorted to without much success; later the wall of the cyst was painted with silver nitrate in the hope that the flow of fluid would cease, but this, too, proved useless. Some infection of the edges of the wound occurred, but this was combated with argyrol. Urotropine was introduced into the cavity and was also given by mouth. About fourteen days after the operation

a hernia developed; this was cut away, but formed again. The patient's condition varied, but got gradually worse, and he died somewhat suddenly on May 29th. Immediately following the second operation the temperature rose to 102° F., and, with the exception of three or four days, it remained about 103° till the day of death, when it rose to 104°.

Pathological Examination of the Brain.—The cerebrum and its meninges presented no abnormality. The base of the brain was seen to be occupied by a large tumour. The under surface of the pons and cerebellum were covered by dark blood clot, the result of a recent hæmorrhage into the pia-arachnoid. The main portion of the growth occupied the left cerebello-pontine angle; there was no growth present to the right of the pons. The left cerebellar hemisphere had become displaced backwards and was partly herniated through the occipital wound. About one-third of this hemisphere, chiefly that portion forming its outer border, together with part of the growth in its neighbourhood, had been removed during operation.

For the purpose of description it is possible to divide the tumour into two halves. The left half consisted of a cyst, oval in shape and measuring an inch and a half antero-posteriorly. It lay in a deep impression on the inferior surface of the left temporal lobe and had contained the clear fluid which was evacuated at the operation. Its wall consisted of firm tumour substance with a fibrous lining and was now filled with blood clot. This cystic cavity communicated, as will be seen in the photograph, with a smaller cystic cavity in the right half of the tumour. This half consisted mainly of soft pinkish-white growth, which encroached on to and invaded the left side of the pons and cerebellum. There was no definite line between the growth tissue and the normal brain substance. Here and there a few small hæmorrhages were present. Irregular outgrowths of the tumour were found in the substance of the pons, and these, being gelatinous in appearance, stood out rather conspicuously. The origin of the cranial nerves on the left side from the fifth to the twelfth was encroached upon and com-

I.

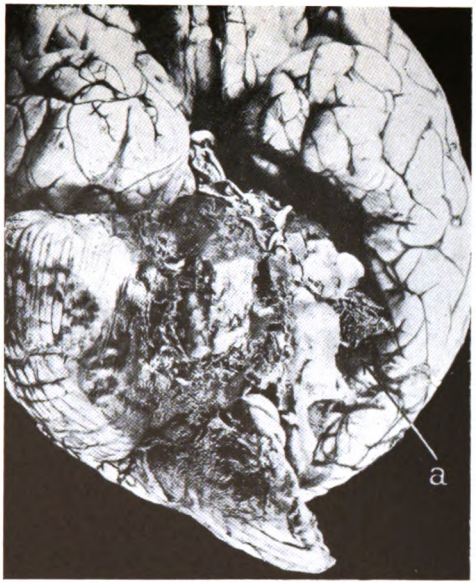


Photo I. shows the Pons and Medulla covered over by hæmorrhage and growth. The remains of the left cerebellar hemisphere is seen to be displaced markedly backward. (a). Position of cystic part of tumour.

II.

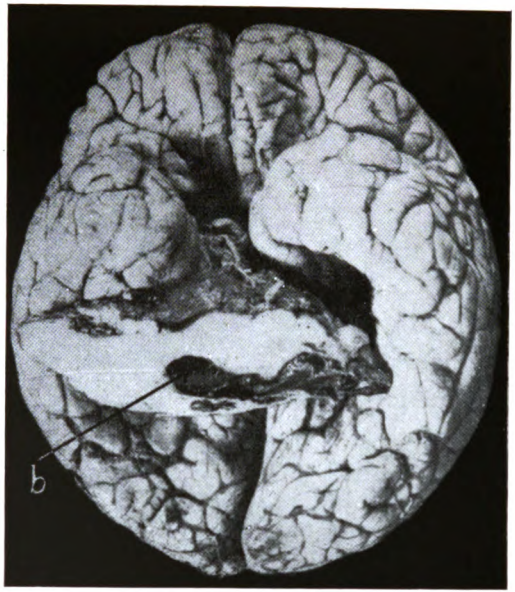
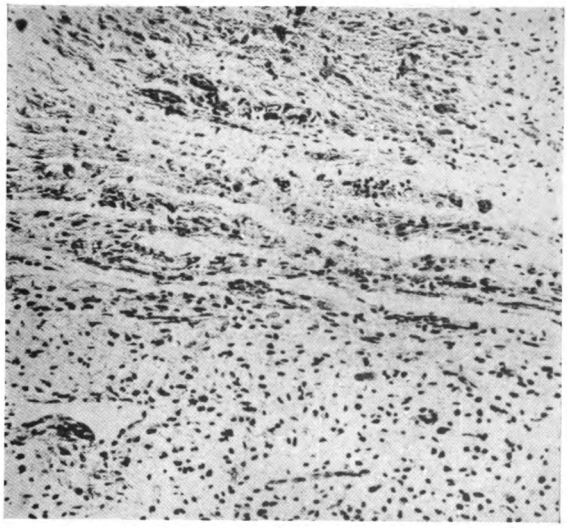


Photo II. shows the appearance on making a transverse section through the lower part of the Pons and upper part of the Cerebellum. (b). Blood clot occupying the cystic dilatation in the inner portion of the tumour.

III.



Microphotograph illustrating, in its lower half, the gliomatous nature of the tumour, and, in the upper, a more crowded appearance of cells, approaching almost a sarcomatous condition.

pressed by this solid part of the tumour. The seventh and eighth nerves were discovered as flattened bands running outwards on the under surface of the tumour.

There were other small cysts scattered through the growth. One, which was isolated from the rest and was half an inch or so in diameter, lay just to the left of the middle part of the vermis.

Histologically the tumour exhibits the structure of a glioma. In parts the section shows typical neuroglial formation. The ground substance consists of a delicate protoplasmic network formed by the branching processes of the tumour cells. The nuclei of these latter are large and well defined, and are surrounded by a thin layer of protoplasm. Occasionally small areas of degeneration occur, which appear to be early collections of fluid between the cells.

In other places, possibly the more rapidly growing portions of the tumour, the cells are more closely packed and the ground substance is proportionately less. Here the appearance approximates somewhat to a sarcomatous condition. This tendency, however, is insufficient to justify the application of the term glio-sarcoma to the specimen. The microphotograph is from a portion of the section which illustrates both types of structure.

FOUR CASES OF PITUITARY TUMOUR *

By

H. L. EASON, M.S., and W. JOHNSON, M.D.

In view of the comparative rarity of tumours of the pituitary body and of the interest recently aroused in them, the publication of the pathological investigation of four cases, which have died in Guy's Hospital, may prove of value. The first two descriptions are taken from cases which came to necropsy during the past year. The last two are from specimens in Guy's Hospital Museum which date back to 1883 and 1877 respectively. In the first case advantage has been taken of the fact that a complete examination of the eyes had been made by Mr. H. L. Eason to undertake an inquiry into the extent of the degenerative changes resulting in the visual system of nerve fibres and cells. In the second case, also, degeneration has been demonstrated in the optic tracts, but unfortunately in this instance it cannot be correlated with the state of the fields of vison.

Case 1.—E. W., a butcher, first noticed failure of sight on the outer side of the field of vision in both eyes in 1910, at the age of 25. The loss of sight appeared to come on suddenly, and, as far as the patient could tell, did not progress further. He had constant vertical headaches, "rushings" in the head, and occasional fits, but no vomiting or vertigo. There was no albuminuria, polyuria, or glycosuria. Admitted to Guy's Hospital in March, 1911.

Condition on Admission.—Eyes apparently normal, with the exception of some pallor of the optic discs. Right eye vision=6/36, left eye=6/9. Complete bilateral temporal hemianopia (vide perimeter chart). The reaction of the pupils to light on both sides was hemiopic. Knee-jerks

*For permission to re-publish the illustrations and portions of the text in this paper we are indebted to the Editor of the *Lancet*.

absent, thyroid isthmus present. Skin dry and hair dry and scanty. The patient is short and undeveloped, not looking anything like his age. Voice half broken and high pitched. No hair on face; testicles normal,

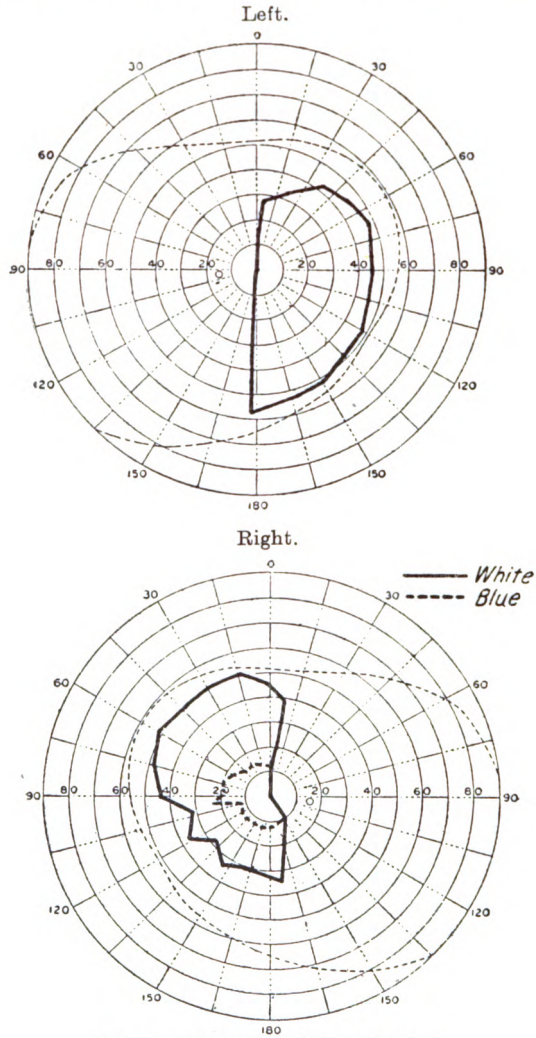


FIG. 1.—Perimeter Chart (Case 1).

but very little pubic hair. Sensation normal. Extremely drowsy and sleeps a great deal; when urged to get up he does so for a few minutes, but soon returns to bed. Face cyanosed and pigmented, resembling the

faces of Addison's disease to some extent. Blood normal, blood pressure 100. Wassermann reaction positive. A skiagraph shows some enlargement of the sella turcica. Weight 7 st. 10 lb.

The patient was continually under observation, and until April, 1913, his condition remained practically unchanged. It was evident that he was suffering from dyspituitarism, and in view of the positive Wassermann reaction he was treated with mercury and iodides, but with no effect; he was also given thyroid and pituitary extracts for long periods, with no effect on vision, though his headaches were relieved, and his general condition improved, with pituitary extract. By the end of 1912 his weight had increased to 8 st. 6 lb. The patient, with his drowsiness and arrested sexual development and absence of all symptoms of acromegaly, was evidently suffering from hypopituitarism, but, as the visual defect did not increase and no symptoms of intracranial pressure arose, it did not appear that any operation was indicated, either for decompression or for the removal of the tumour, and treatment was limited to the steady administration of extract of pituitary gland.

In February, 1913, the fields of vision were again tested, and the field for white was practically the same as in 1911. The colour fields for the right eye were charted, and showed some diminution in the blue field. The field for red and green were too small and indefinite to record, green being recognised as such only at the fixation point, and red called brown wherever visible.

The patient was exhibited at the combined Meeting of the Sections of Neurology and Ophthalmology of the Royal Society of Medicine in March, 1913 (H. L. Eason, Case II.), and up to that time his condition had remained unaltered. Shortly after this, however, he suddenly began to go down hill; he took to his bed, lost weight very rapidly, and passed into a state of asthenia and inanition. He had occasional syncopal attacks, but otherwise remained fairly normal; generally he had no appetite, but occasionally he expressed a desire for unusual food, such as pickled onions, of which he ate heartily. Up to the last his vision did not fail entirely, the nasal side of the field of vision remaining complete; there was no nasal discharge, epistaxis, or paralysis of any of the ocular muscles. The patient's sugar tolerance was never tested. A condition of semi-coma prevailed during the last three weeks. He died suddenly, apparently from heart failure, at 11 a.m. on June 18th, 1913. The autopsy was performed at 2 p.m. on the following day by Dr. French.

Autopsy.—General Appearance: The body looks like that of a boy of about 16 years; the cheeks are smooth, but there is just a trace of a moustache at the corners of the lips. The size and shape of the skull and face are not abnormal. The body is considerably wasted, and there is no unusual deposit of fat anywhere. The skin is generally pigmented, especially at the points of pressure and tension, e.*g.*, over the anterior superior spines and the great trochanter. Alimentary System: The œsophagus, stomach and intestines all normal. There is hardly any intra-abdominal fat, either in the mesenteries or the region of the kidney. The liver is small, yellowish in colour, and fatty. The gall-bladder contains

thick, dark-green bile. The pancreas is normal in naked-eye appearance. Circulatory System: Heart rather small, and muscle a little brown in colour; no dilatation or valvular disease. Pericardium normal. Great vessels normal, if rather small. Cerebral vessels normal. Respiratory System: Some old pleural adhesions about the apex and outer surface of the upper part of the left lung. Lungs themselves slightly œdematous, otherwise normal. Excretory System: Kidneys, ureters, and bladder normal. Ductless Glands: Thymus not seen. Carotid bodies not identified. Thyroid, spleen, suprarenals, all normal to the naked eye. Reproductive System: Penis and scrotum small. Pubic hair scanty. The testes also rather small, but apparently normal. Epididymis and vas normal. Nervous System: Calvaria smooth on both surfaces, and symmetrical, measuring a quarter of an inch at its thickest parts, and three-sixteenths in most of its circumference. Sutures normal. Dura mater normal, and no external hydrocephalus. On removing the skull cap a small amount of grumous yellow fluid was seen to be tracking upwards along the right Sylvian fissure from the base of the brain. The fluid was afterwards traced to a patch of yellowish degenerated brain substance on the under surface of the right frontal lobe. There was no flattening or other abnormality of the convolutions. The arachnoid and pia mater were normal. On lifting up the brain, a large tumour was seen in the neighbourhood of the interpeduncular space, and it was noted that the optic tracts on each side passed round the side of the growth to the optic foramen of the same side. The tracts and nerves were separated from each other by the whole width of the tumour, and there was no visible trace of a chiasm or of decussating nerve fibres. The brain was removed entire and hardened. The base of the skull in the region normally occupied by the sella turcica was extensively eroded, so that on the removal of the brain and tumour a cavity was left measuring 39 mm. antero-posteriorly and 41 mm. transversely. The floor of the sella turcica was expanded and flattened, measuring 21 mm. antero-posteriorly and 26 mm. from side to side, but there was little erosion, and no communication either with the sphenoidal sinus or the naso-pharynx. The anterior clinoid processes were eroded, especially on the right side, and there was no trace of the optic groove. The dorsum ephippii and the posterior clinoid processes had entirely disappeared, all that was left of the posterior part of the sella turcica being a low wide ridge of eroded bone, the growth having infiltrated the cancellous tissue of the body of the basi-sphenoid and occipital bones. The growth had extended laterally into both cavernous sinuses, the inner walls of the dura mater having almost completely disappeared. The carotid arteries, the nerves lying in the sinus were surrounded by the growth, but were not compressed or eroded by it. The outer walls were intact. Brain: The whole of the space between the pons, temporo-sphenoidal, and frontal lobes was occupied by a crimson, soft, smoothly lobulated growth springing from the hypophysis. The lower surface of the growth was very vascular, hæmorrhagic and diffluent, but anteriorly it consisted of several lobes, one of which had passed into the olfactory sulcus on the right side, separating the gyrus rectus from the inner orbital convolution. The

olfactory tract on this side was compressed between a lobule of the growth and the inner surface of the inner orbital convolution. The larger mass on the left side had entered the anterior part of the great longitudinal fissure, and had displaced outwards the inferior part of the orbital surface of the left frontal lobe. The whole of the inter-peduncular space was filled up by the tumour, which extended backwards upon the pons almost as far as the origin of the fifth nerve. The temporo-sphenoidal lobes on each side were distinctly flattened, but were not infiltrated by the growth. The optic tract on each side wound round the tumour, that on the right side being apparently normal, that on the left being much flattened. Not a trace of the chiasm remained visible to the naked eye. The degenerative changes found in the optic tracts suggest, however, that this destruction of the chiasma was of comparatively recent date. The third and fourth nerves traversed the tumour, though they were apparently not affected during life. The sixth nerve was not involved, though the growth had invaded the cavernous sinus.

A mesial section through brain and tumour shows the basal ganglia and corpus callosum to be displaced upwards. The superior and inferior walls of the lateral ventricles were in close apposition, their cavities being practically obliterated. It was possible to shell out the tumour quite easily from the bed which it had forced for itself in the base of the brain.

Microscopical Examination.—Sections were examined of portions of the thyroid, pancreas, suprarenals, liver, and testis. Thyroid: The alveoli appeared well formed and contained the normal amount of colloid. There was no evidence of cell proliferation and the interstitial tissues were normal in appearance. Pancreas and suprarenals: The structure of these organs was that of normal functionating glands. Liver: Extensive fatty change was present throughout the section and was confined to the peripheral portions of each lobule. The cells in the centre of the lobule were normal in appearance, and there was no cirrhotic change. Testis: The seminal tubules were normal in number and in character. There was a slight increase in the interstitial connective tissue, indicating a very early fibrotic change. Sections were examined with special reference to the condition of the interstitial testicular cells. These are markedly deficient in number. Several sections were examined from various parts of the testicle. Stained with Herxheimer's stain, the few interstitial cells present were found to be in a somewhat atrophied state. The pituitary tumour itself under the microscope was seen to be made up of large polyhedral cells, the protoplasm of which stained a faint pink and the nuclei a dark blue with the ordinary hæmatoxylin and eosin method. The chromatin granules show up distinctly in these latter. The cells themselves are arranged in long columns with occasionally a tendency to alveolar grouping, the whole appearance closely simulating the structure of the anterior glandular portion of the pituitary. No colloid material was discovered

and nothing corresponding to the posterior lobe was seen. The vascular spaces characteristic of this organ were well marked, and towards the centre of the tumour they became large hæmorrhagic cysts in which red blood cells were mixed with necrotic tumour cells. Towards the periphery, however, they became smaller and smaller until, when the capsule was reached, they ceased to exist. The capsule was thick and consisted of dense fibrous tissue. In numerous places the cells of the growth were extending into the capsule along the lymph channels. Its appearance, therefore, was that of a malignant epithelial tumour of the pituitary gland. The entire absence of secondary deposits is noteworthy.

CASE 2.—The patient, an unmarried woman aged 27, was admitted into Guy's Hospital under the care of Mr. L. A. Dunn. She had had continuous headache for the previous three weeks accompanied by vomiting. The left eye was completely blind and the vision in the right eye was markedly defective. Both eyes reacted to light and accommodation, but the reaction of the left was very deficient. The patient was seriously ill, and died shortly after admission, so that a detailed examination of the eye condition was impossible. She was extremely lethargic. Pulse 56 and low tension. Temperature subnormal. The left eye showed a definite degree of exophthalmos as compared with the right. Further details have been kindly supplied by Dr. H. J. Henderson and Mr. L. C. D. Irvine, of Amersham, who attended the patient in private. They inform us that in spite of her age menstruation had never occurred. The breasts were undeveloped and there was complete absence of pubic and axillary hair. Nothing is known of the sugar tolerance, but for some time past she had shown a great partiality for sweet things, and had been used to taking bread and butter thickly coated with brown sugar. A year ago Mr. Arnold Lawson had examined the eyes and found advanced optic atrophy in the left eye. This was confirmed on the examination of the patient here. There was no sugar or albumin in the urine.

Post-mortem Examination.—With the exception of the tumour nothing abnormal was found in any of the organs (Dr. Herbert French's report). The tumour: In the position of the pituitary body, filling up the interpeduncular space, there was a cystic tumour, the relations of which are shown in Fig. 5. It measured three-quarters of an inch antero-posteriorly and one and a quarter inches from side to side, and was almost exactly the size of a golf ball. On removal a deep hollowed-out depression was found at the base of the brain. The sella turcica, on which the tumour had rested, was flattened into a shallow concave surface. The vessels of the circle of Willis were present on the lateral walls, indicating that the original cyst had been small enough to be within the circle, and that as it enlarged the vessels became stretched on its outer walls. The optic chiasma lay stretched over the antero-inferior portion of the tumour, and had apparently given way through its centre. The right optic tract and nerve were not much damaged, but the portions of the left optic tract and nerve, near the chiasma, were pressed out into a wide thin band. The tumour was pressing them down on to the hard

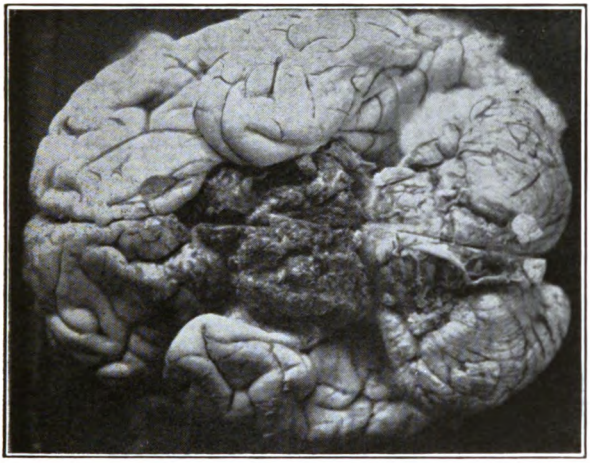


FIG. 2.

View of the tumour *in situ* (Case 1).

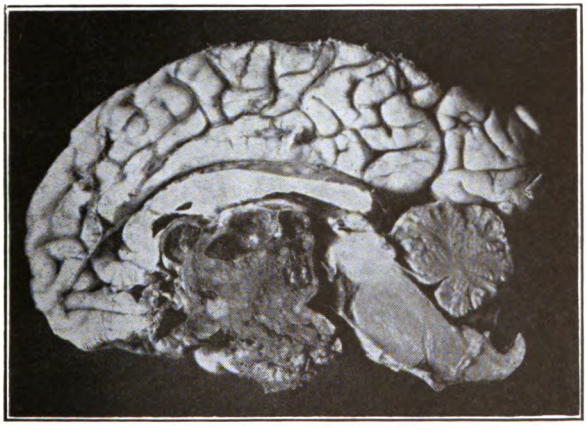


FIG. 3.

Mesial section through brain and tumour. The capsule of the tumour lies close up against the corpus callosum. The hæmorrhagic cysts are well seen (Case 1).

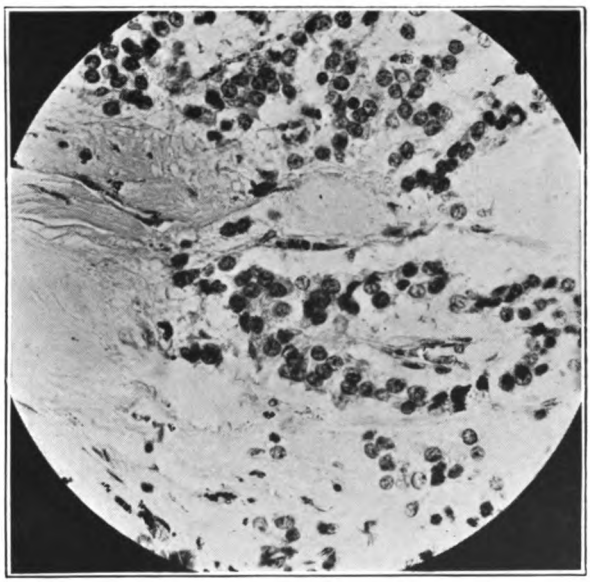
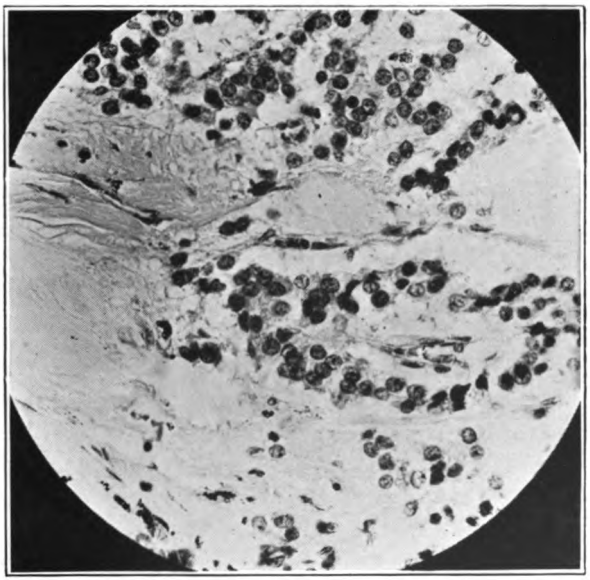


FIG. 4.

The upper portion of the field shows the edge of the growth. The lower portion shows up the capsule being invaded by groups of tumour cells (Case 1).



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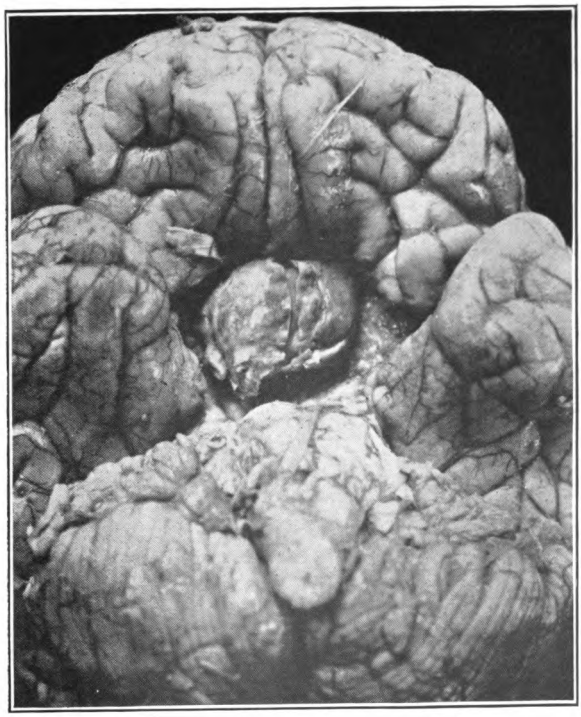


FIG. 5.

View of tumour in Case 2. The cyst has been distended with cotton wool to its normal size.

FIG. 6.

From a photograph (actual size) of the tumour in Case 2, showing the glandular masses in the capsule.

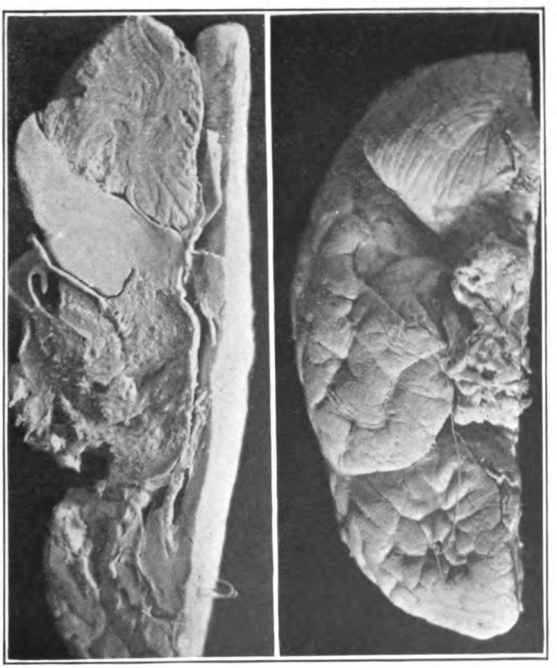


FIG. 7. FIG. 8.

Fig. 7.—Mesial view of No. 2876 showing the capsule of the tumour lying just beneath the corpus collosum as in Case 1.

Fig. 8.—View of the left half of the brain, No. 2876, showing the position and appearance of the tumour.

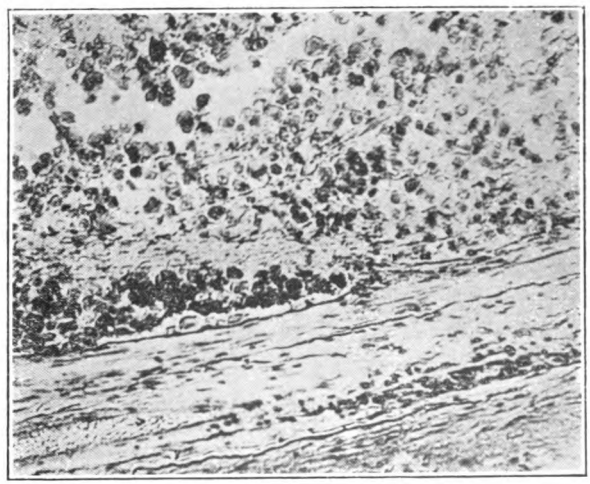


FIG. 9.

Reproduction of a photomicrograph of No. 2876 showing cells at the edge of the growth infiltrating the capsule.

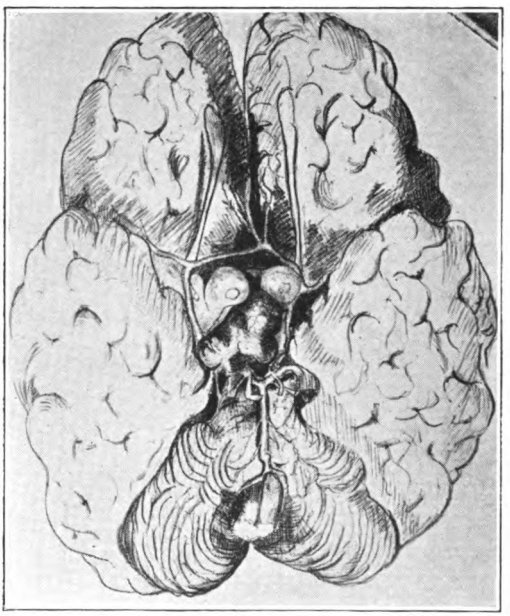


FIG. 10.

Reproduction of a drawing of the brain by Mr. Hamersley in the original post-mortem report (No. 2875). The tumour is largely hidden by the two swollen optic nerves.

bone at the base of the skull. On the other hand, the third and fourth cranial nerves, lying between the tumour and the soft structures forming the base of the brain, showed little or no flattening. On being cut through, the tumour was seen to consist of one large cyst composed of two loculi, which communicated freely with each other, and they contained a thin reddish fluid. The wall of the cyst was tough and fibrous and scattered over its upper surface there were four or five small masses of a reddish glandular substance closely similar in appearance to the anterior pituitary lobe. The largest was of the size of a pea, and the smallest about half this size. In addition there were still smaller masses of the same nature to be found scattered over the surface. The larger ones are shown in Fig. 6, which is about the natural size. It also shows the division into two loculi—the large one which formed the main portion of the cyst, and the smaller one which occupied the right anterior angle of the inter-peduncular space.

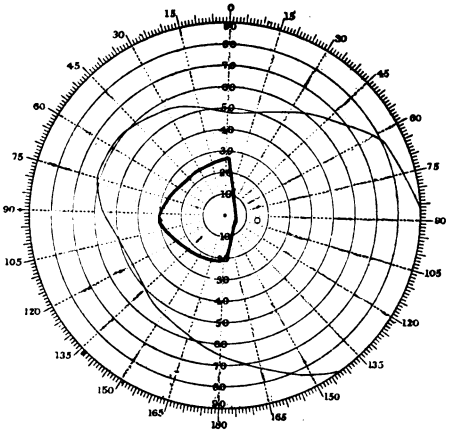
Macroscopically the brain was normal in every way, with the exception of a slight degree of dilatation of the lateral ventricles. Under the microscope the cyst wall was seen to consist mainly of laminated fibrous tissue. Everywhere between the laminæ were to be seen small collections of cells, which in places were aggregated to form the red glandular masses already referred to. The arrangement and character of these cells to all appearances were those of the anterior lobe of the pituitary. Large spaces packed with red blood corpuscles lay between the columns into which the cells were arranged, and in places large hæmorrhages were present. The cells themselves were polyhedral or oval in shape and had large dark nuclei. The majority were chromatophobes, but several approximated to the chromatophilic type. More externally they showed the same tendency to infiltrate the layers of the cyst wall, as was seen in the first tumour. It can only be described, therefore, as of the same nature, viz., malignant epithelial tumour of the pituitary gland, with complete absence of secondary deposits.

CASE 3.—A. W., aged 23, was admitted into Stephen Ward, Guy's Hospital, under the care of Dr. Pavy on August 29th, 1883. Three years before his admission he commenced to feel ill with pains in his jaw-bones, the back part of his neck, and the crown of his head. Eighteen months before his admission he found he was unable to see as well as formerly with his left eye, and six weeks before admission he lost the sight of it.

On admission it was noted that he was a short, strongly-built man, height 5 ft. 7 in., with dark hair, and no muscular wasting. His weight was not taken. His skin was cool and moist, but he complained of polyuria. The left eye was totally blind. In the right eye a note was made that he could only see one inch to the temporal side of the middle line, but one foot to the left, one foot above the fixation point, and about eight inches below. (No mention is made of the distance at which the test object was exhibited to the patient, but, presuming it was an examination at a distance of two or three feet from the patient, this would

correspond to a field approximately of the size delineated in the accompanying chart.) The patient had, therefore, total blindness of the left eye, and temporal hemianopia of the right. The pupils were about equal, and reacted to light and accommodation. Both optic discs were atrophied, otherwise the man's general condition was normal. A note was made that all the usual nerve reflexes were absent, with the exception of the cremasteric. There was no note of sexual inefficiency, or of any symptoms of acromegaly.

Right.



During his stay in the ward the patient seemed to complain of little but general weakness and continual headache. On September 21st it was remarked that his memory was very bad. He was always forgetting when he spoke to anyone, asking the same questions every time he saw a person go up the ward. He passed a large amount of urine, the average quantity being about 80 oz., the amount rising on some days as high as 170. There was no sugar or albumen in the urine. On October 20th the patient was very drowsy and dull, both hands were very weak, and he was constantly making grimaces. His sensory powers were tested on this date. and though hearing seemed to be comparatively good, taste and smell were distinctly defective. On October 24th the patient had a fit with no very definite or localised symptoms. He became comatose, and died later in the day. Just before death a note was made that the right pupil was dilated, the left being normal in size.

Post-mortem examination.—(Specimen No. 2876 in Guy's Hospital Museum).—The pituitary fossa was enlarged to form a diamond-shaped space, in which lay a soft lobulated tumour. This occupied the position of the pituitary body and extended forwards over the ethmoid ; laterally it overlapped the temporal-sphenoidal lobes, and posteriorly it covered over the upper part of the pons. On the left side the optic nerve and third, fourth, and fifth nerves were pressed into flattened bands. On

the right side, the optic nerve and third nerve were lost in the tumour, while the fourth and fifth nerves were stretched over it. The other cranial nerves were unaffected. The tumour itself was more or less circular in outline, and measured two and a half inches across. On section, thick cream-like material oozed away, leaving numerous cystic cavities with ragged walls.

Under the microscope the tumour was found to consist mainly of cellular elements, the cells being the chromatophilic type, though somewhat smaller than the cells in the two previous tumours. With the exception that there is much less stroma present, the general appearance, however, closely approximates to them. There is the same tendency for the cells to form columns and an alveolar arrangement, and also for those on the periphery to infiltrate the layers of the capsule. With the other two, therefore, it must be classed as a malignant epithelial tumour of the pituitary gland, showing no tendency to form secondary deposits.

CASE 4.—(Specimen No. 2875 in Guy's Hospital Museum).—The patient J. W., a male, aged 45, died in Stephen Ward in 1877. He had been under the care of Dr. Wilks. He was a thin, wasted man. Three weeks before admission he was seized with fits which were followed by left hemiplegia. On admission there was loss of memory, occipital headache, and nausea, but no vomiting. The vision was dim, but no definite optic neuritis or optic atrophy was present. The left external rectus muscle was paralysed. The patient was often delirious.

Pathological Examination.—The ventricles of the brain were slightly dilated and contained turbid fluid. At the base of the brain was a large gelatinous tumour which was in direct continuation with a somewhat enlarged pituitary body. The bone in the pituitary fossa is flattened out and partly absorbed. The tumour measured one and a half inches long by one inch broad and one inch thick. The optic commissure is stretched over it anteriorly, and the optic nerves, as seen in the photograph, are swollen up into two large swellings half an inch in diameter. Laterally and posteriorly the tumour is bounded by the crura cerebri. There was a patch of growth in the apex of the lower lobe of the left lung consisting of spindle cells arranged in loculi. Histologically the tumour showed a relatively large amount of fibrous stroma, in which lay groups of cells the majority of which were columnar in shape, a few being polyhedral. There was much mucinous material scattered through the section and several goblet cells were to be seen, which would suggest a possible origin from nasal mucous membrane. Its characters were those of a malignant columnar-celled growth. A secondary deposit was present in the apex of the lower lobe of the left lung, showing similar histological appearances.

The Examination of the Visual System of Nerve Fibres and Cells.—The left side of the brain in Case 1 was sectioned and utilised for this purpose. A reference to the figure illustrating

the fields of vision will show that the optic fibres destroyed by the tumour are confined to the nasal half of the retina, *i.e.*, the temporal half of the field of vision. And, further, that a portion only of these fibres is affected. From this it might be expected that the degeneration present in the optic tracts would be extremely limited and circumscribed.

The Optic Tracts.—The accompanying drawings, Fig. 11, of the left optic tract, however, made from Weigert-Pal preparations, illustrate the condition actually found. A narrow band of fibres (a) occupying the outer portion of the tract alone stain normally. Two other bands of fibres (b) and (c) also stain,

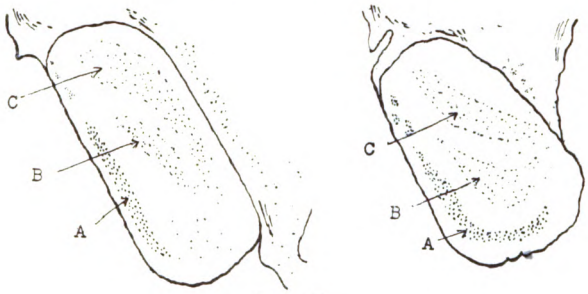


FIG. 11.

Posterior portion. Anterior portion.
Left Optic Tract, Case 1. Weigert-Pal Preparation.

only less distinctly. The remainder of the tract is completely degenerated. The degenerated area can be described as occupying mainly the central and lower part of the tract. This agrees with Henschen's conclusions as to the position of the nasal fibres in the optic tract. The fibres which stain normally (a), however, which are from the temporal side of the retina, are found lying in the external and lower portion of the tract and not in the superior and external position which he describes.

A comparison of the two drawings will show further that the relative position of the fibres remains approximately the same along the whole length of the tract. The first section is taken from the anterior part of the tract and the second from the

region of the external geniculate body. Sections taken through the subthalamic region still showed the degeneration in the optic tract fibres as they entered the external geniculate body and the pulvinar. In this region no particular zone was degenerated; the affected fibres seemed to be scattered amongst those which stained normally. Preparations stained by Marchi's method show the presence of early degeneration amongst those fibres which stain normally with the Weigert-Pal method.

The Cortical Visual Fibres.—Attention was next devoted to the state of those fibres which, after the relay of the optic tract fibres in the external geniculate body, continue the visual impulses to the occipital cortex. No change could be demonstrated. The sections through the optic radiations, stained by Weigert-Pal, were quite normal. Similarly, no change could be found in these fibres when traced to their termination in the cortex surrounding the calcarine fissure. Sections of this cortex were then stained by Nissl's method, and the large stellate cells in the fourth layer, *i.e.*, those concerned with the reception of the visual impulses, were examined. It cannot be said that there was any deficiency in the number of these cells or in their size. It was, however, found that a certain proportion of them exhibited chromatolytic changes, but this did not occur in any definite grouping.

The Fibres Concerned with the Reaction of the Pupils to Light.—The presence of the hemiopic pupil suggested that this case might throw some light on the question of the course which these fibres take. Serial sections of the posterior thalamic region and the mid brain were accordingly cut, and compared with normal preparations. The path described by Meynert, *i.e.*, fibres running dorsally to the anterior corpora quadrigemina, relaying in these bodies and continuing by means of new fibres which end in the oculo-motor nucleus, did not give any evidence of degeneration. Similarly, the investigation of the path described by Darkschiewitsch and Mendal, *i.e.*, fibres running to the ganglia Habenulæ, and continuing by new fibres which

reach the oculo-motor nucleus via the posterior commissure, also led to negative results. As, however, the majority of pupil fibres were unaffected in this case, and, further, that either theory states that the degenerated fibres end in cell relay stations in the neighbourhood of the primary optic ganglia, this result is not surprising.

In Case 2 the optic chiasma, although severely compressed, was not broken through as in Case 1. Primary optic atrophy had been observed clinically in the left eye, but not in the right. The condition of the optic tracts was as follows: The left optic tract was quite normal with the exception that a few degenerated fibres were found occupying its lower border. This area is indicated in the drawing. The right optic tract, on the other hand, was found to show marked degeneration, very

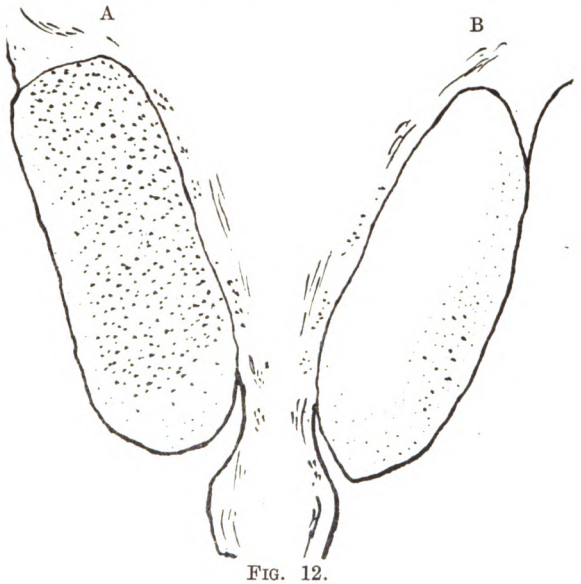


FIG. 12.

Left (A) and Right (B) Optic Tracts in Case 2. Weigert-Pal Preparations.

similar to that present in the other case. A band of fibres, situated in the outer and lower part of the tract, are seen to

be unchanged, although even they do not take the stain as well as do perfectly normal fibres. The remainder of the tract is completely degenerated.

Conclusions.—The only change, therefore, which it has been possible to demonstrate in the visual fibres as a result of a lesion of the optic chiasma, which had caused eye symptoms, in both cases, for upwards of two years, is a degeneration in the optic tracts. This degeneration was not found extending further back than the primary optic ganglia. The amount of change present in one case was more than was indicated by the visual loss. The statement that certain optic fibres reach the occipital cortex without relaying in the cell stations in the primary optic ganglia would appear to be refuted by the fact that no degeneration was found in the optic radiations and the fibres entering the calcarine cortex. Finally, both cases show that the uncrossed visual fibres tend to occupy the outer and lower portion of the optic tracts.

THE ADVANTAGES OF PHYSICAL GAMES OVER SET EXERCISES.*

By

M. S. PEMBREY.

Education has so weakened the force of instinct that in civilised communities there are many who do not know how to live. One expression of this defect is the demand for advice upon the physical training of the body. Are games sufficient, or are the set exercises of some system of physical culture necessary? The child guided by his primitive instincts has no doubt that games are superior to set exercises; play is his chief pleasure, and drill is a recognised punishment in many schools. Is the child right in his estimate or is it more reasonable to expect guidance in this question from the Chiefs of Government Departments who have issued courses of physical exercises? The evidence for and against the two views must be examined as a question of physiology. It is useless to expect guidance from enthusiasts who think that with a little knowledge of anatomy they can evolve systems of physical culture which will cure all manner of disease and regenerate the physical and moral tone of the race. Medical man have neglected this subject to the disadvantage of themselves and the public.

Many people are inclined to distrust the verdict of the child, for there is a widely spread idea that what a child likes is bad for him and what he dislikes is good discipline. There is, however, a steady increase of physiological evidence to show that

* Lecture given at the Royal Sanitary Institute, May 5th, 1914.

the likes and dislikes of a healthy child are a natural guide to health. He is a young animal, and under natural conditions he will grow and develop according to type.

Physical games have an origin more ancient than that of man himself. The young of many mammals show their inborn instinct to play and train their bodies by gambols. Everyone must have watched with pleasure the play of puppies or kittens, and most observers have noticed that the loss of the desire to play is a sign of ill-health or defective development. The training involved in such gambols produces the wonderful physical efficiency of the mature animal. If there be any truth in evolution the same biological laws should apply to man. No one will contest the statement that the healthy child possesses the same desire to play. At as early an age as six months the infant will engage in play and without any teaching from anyone gradually learns to control the contraction and relaxation of its muscles. Physical games, even the most complex, have been evolved from play. Healthy children provided with suitable opportunities develop their own games, and, as in the case of young animals, the ones who do not play are abnormal in body or mind, or both.

It is a deplorable sign of the times that it should be necessary to teach young children how to play, and that the Board of Education should admit children only three years of age to its schools and provide them with cots and instruction in physical exercise. It would be better if compulsory education did not begin until seven or eight years of age, for the early aggregation of young children has been responsible for the heavy mortality from measles, whooping-cough, and other infectious diseases. Early education is a bar to play; sitting still at a desk is a severe tax upon an energetic child, causes it more fatigue than running about at will, and is a fruitful source of spinal curvature. In order to go to school and satisfy the demands of the powers that be the child must be clean and tidy in person and clothes. No healthy child likes to be clean and tidy except on Sundays and other special occasions; it is harassed and oppressed by frequent warnings not to spoil its clothes or get dirty at play. The

chief aim should be to encourage the healthy exercise of play in the early years of rapid growth.

Physical games are an education not only of the body, but also of the mind. Of this there is no doubt, even although scientific knowledge is so incomplete that many of the complex processes involved cannot be explained. It will be well, therefore, to consider in detail the effects* of games upon the body as a whole, for as such it works. Muscular movements are accompanied by combustion and the energy is obtained from the food. The appetite of a healthy child is proportionate to the need of food. More play means more work for all parts of the body and a greater demand for food. Exercise is the most powerful stimulus, and the healthy body reacts with increased growth and efficiency. After active play the child becomes tired and responds to this natural safeguard, fatigue, by sleeping soundly. The real nature of fatigue and sleep we do not know, but the underlying purpose is clear. Sleep is the great restorative, and the sounder it is the greater its value.

Learning to walk is a complex education. The child has to discover the relationship of its limbs and learn to respond to the sensations arising from its skin, muscles, and tendons; it must contract in due order some muscles and at the same time relax others; it must pump the blood more actively through its vessels and breathe more rapidly and deeply. No knowledge will enable us to teach it how to effect this wonderful combination of processes; it must learn by practice in the same way that young animals do. Along with the general sensations are trained also the eye and the sense of equilibrium, otherwise the child would be unable to avoid obstacles or recognise the relation of its body and limbs to external objects.

Nature shows the guiding principles of all sound methods of training—progression; the child does not learn to run before it learns to walk. Running increases greatly the effects produced

*For further details see "The Physiological Principles of Physical Training," Journ. Royal Army Medical Corps, vol. x., 1908, p. 339; and Journ. Royal Sanitary Institute, vol. xxxii., 1911, p. 345.

upon the body by walking, and further complexities in education are introduced when the child in its games jumps, hops, and skips.

The mental discipline of games with other children is of the greatest value. The child learns to work in harmony for its own benefit as well as that of its small circle of friends; sometimes in a subordinate position it must restrain itself, at other times as a leader it must assume authority and see that orders are obeyed. Leadership in games is almost invariably awarded to merit, and if the weaker go to the wall it is but the recognition of the survival of the fit. In competitions over games, or even in the fights which may result therefrom, the child learns that in nature there is no such thing as equality, and by combination finds a means of enforcing a rough justice. In short, the child learns "to play the game" in all its full significance. Self-reliance is increased and unhesitating response to the emergencies which may arise during a game.

One of the chief advantages of games is the open-air life which they involve. The exposure to sun, wind, and cold produces a stimulating effect upon the cutaneous senses which leads to appropriate responses in the nervous and chemical processes of the body. Resistance to disease is increased by the outdoor life, and in the long run it is cheaper to provide playing fields than gymnastic instructors.

Games are a protection against excessive mental work; in an active game the whole attention is absorbed in pleasure, and the healthy muscular and nervous fatigue results in a sleepiness which is incompatible with mental activity.

So far the advantage of games to children alone has been considered. To young people and adults of all ages health is of equal value. Happiness is bodily and mental health, and is far more valuable than anything else in the world, whatever visionaries may say. Games are so diverse and graduated that there are some suitable for all ages and constitutions. Played for their own sake games give a relief from mental worry, and, if regularly pursued, are a protection against a breakdown due

to excess of nervous work. The destinies of a country are the safer in the hands of cabinet ministers when they can in a time of crisis take pleasure in a game of golf.

It may be asked, Are there no disadvantages in games? There are. The drawbacks are due chiefly to competition. Play is in danger of degradation when it is not for pleasure, but for public notoriety and pecuniary gain. Violent games for the untrained are injurious; progression is necessary for efficiency. In women the pursuit of games and sport may conflict seriously with their duty to the race, their real duty to themselves; the test for efficiency in women is motherhood.

The evidence for and against gymnastic or set exercises must now be examined. Such systems are chiefly of foreign origin and mechanistic in view. Muscular exercise is under control and can be dispensed in doses. There are many men who say that they have no time for games or outdoor work; in set exercises they maintain they have an antidote to sedentary life. The man who every morning and evening religiously uses an "exerciser" fastened behind his bedroom door may obtain benefit, for any exercise is better than none, and the belief that he is doing something for his physical and moral good will be of value. At the same time the man gives evidence that he lacks the health of a really sane man. When he has his nervous breakdown he will consider himself a martyr to the pressure of modern life and will expect and probably receive in full measure the sympathy and admiration of many friends. He has taken exercise as a medicine and as an antidote to a vicious indulgence in mental activity; he has overlooked the fact that in the long ancestry of man muscular activity has been a condition of a full life, and that even in modern life the child starts with ancestral characteristics.

There is a medicinal flavour in set exercises; some are called nutritive, others corrective, and others depletive. Most of them cause undue fatigue, lack interest, and are monotonous. They are carried out in response to command, give little scope for leadership or individuality, and lack the pleasure and variety of

games. In many cases their rhythmic nature is a disadvantage, for there is no need for that alert response to emergencies which games demand. The set exercises are designed to bring about the uniform development of all the muscles; this ideal is wrong, for a "muscle bound" condition may result.

The breathing exercises are unsound in theory, and in practice increase the danger of infectious diseases, owing to the spray of saliva which is discharged from the mouth.

Set exercises can be defended as a discipline for the healthy, but in the treatment of the sick and deformed is their true position. Unfortunately owing to the neglect of the medical profession the therapeutic use of set exercises has been exploited in undesirable ways. If the authors of systems of physical culture would remember that "only the sick need the physician" they would encounter less opposition in their craze for set exercises; if they would study the doctrine of evolution and read Herbert Spenser's "Essays on Education" they would recognise that there is latent in the healthy infant the capacity for a perfect physical development. The child's games are the ancestral means of training, and any attempt to displace them by systems of physical culture based upon set exercises is a sign of ignorance.

A NOTE FROM THE MEDICAL CLINICAL LABORATORY ON UROBILINURIA IN HEART FAILURE.

By

E. J. COOKE and N. MUTCH, M.A., M.D.

The observations recorded in this paper show that many patients suffering from heart failure excrete urobilin in their urine. This excretion occurs in absence of jaundice or of pathological changes in the liver other than those secondary to over-distension of the radicles of the inferior vena cava, and when even the most delicate tests fail to detect bile pigments in the urine. It has been possible to correlate this excretion with the tenderness of the liver, from which it would appear probable that it is determined by compression of the bile capillaries by active distension of the hepatic venules. It is not very closely associated with the increase in size of the liver, and, therefore, is not due to permanent changes in that organ. In other words, it is a sign of present heart failure and not of a nutmeg liver. One further point has been made clear, that the pigmentation of the skin often found after repeated attacks of heart failure is not due to urobilin.

The patients selected for observation presented lesions of the mitral valve uncomplicated by aortic disease, and gave a history of rheumatism. They had never been jaundiced, and at the

time of examination did not show any trace of bile pigmentation. Exact measurements of physical signs were made when possible. The degree of liver tenderness, the pigmentation, and the urobilinuria, however, could not be measured accurately, and in these instances an American method advocated elsewhere by one of us* was employed. It is applicable to the estimation of most clinical signs and symptoms. The absence of a sign is denoted by—; its doubtful presence by (+); its definite presence by +; if very well marked by 2+; if extreme by 3+.

The following tests were applied to urine which had been diluted to a standard specific gravity 1015.

Urobilin.—To 50 c.c. of urine in a 100 c.c. glass cylinder were added 10 c.c. of glacial acetic acid and 10 c.c. of amyl alcohol. The cylinder was inverted three or four times and the solution of urobilin in amyl alcohol examined with a spectroscope. The thickness of fluid was 3 cm. A spectrum without a broad absorption band denoted absence of urobilin; a faint doubtful band indicated a trace of urobilin (+); a well-defined band in the green, urobilin +; obscuration of the entire blue end of the spectrum, urobilin 2+; if the extract was of a dark sherry colour and considerable dilution was necessary before a band could be seen against a bright sky the measure of the urobilin present was 3+.

Bile Pigments.—100 c.c. of urine were made alkaline with ammonia and saturated with ammonium chloride. After standing the precipitated urates were removed on to a filter paper and nitric acid applied. A green colouration is produced if traces of bile pigments are in the original urine, but it was never observed in any of our cases.

The following table is a summary of our observations :—

* N. M. Quart. Journ. of Med., 1914, vol. vii,, p. 441.

No. of Case.	Name, Sex, Age.	HISTORY.			HEART.				LIVER.			Pigmentation.	URINE.		AUTOPSY.
					Percussion.				Size.						
		1st Attack of Rheumatism	1st Attack of Heart Failure.	Jaundice.	L. of Sternum.	R. of Sternum.	Mitral Bruits.	Aortic Bruits.	Below Ribs. R Nip. Line.	Above Umbil. Mid. Line.	Tenderness		Urobilin.	Bile Pigments.	
		Years	ago.		cms.	cms.			cms.	cms.					
1	E. W. *F.* 50	35	**20**	Never	11	3	Systolic Presystolic	Absent	15	1 (below)	2+	2+	2+	—	
					Orthopnœa, slight œdema, and auriculo-fibrillation										
2	W. S. *M.* 21	12	1 week	—	14	4	Systolic Mid-diastolic	—	15	2	2+	+	2+	—	
					Orthopnœa, no anasarca, marked œdema of bases										
			1 mth	later	—	—	—	—	12	4	(+)	+	(+)	—	
3	E. T. *F.* 50	18	1	—	13	1	Systolic Presystolic	—	13	0	2+	2+	2+	—	
					Orthopnœa, slight œdema, auriculo-fibrillation										
4	S. L. *F.* 44	?	3	—	11	2	Systolic	—	13	1	2+	2+	2+	—	
					Orthopnœa, anasarca										
			1 mth	later	—	—	—	—	Just	palpable	(+)	2+	(+)	—	
5	A. B. *F.* 32	5	3	—	14	3	Systolic Mid-diastolic	—	3	13	+	(+)	2+	—	
					Orthopnœa, slight anasarca										

No. of Case.	Name, Sex, Age.	HISTORY. 1st Attack of Rheumatism	1st Attack of Heart Failure.	Jaundice.	HEART. Percussion. L. of Sternum.	R. of Sternum.	Mitral Bruits.	Aortic Bruits.	LIVER. Size. Below Ribs. R Nip. Line.	Above Umbil. Mid. Line.	Tenderness	Pigmentation.	URINE. Urobilin.	Bile Pigments.	AUTOPSY.
		Years	ago.		cms.	cms.			cms.	cms.					
6	A. M. *M.* 13	3	1	—	13	5	Systolic Mid-diastolic	—	13	4	+	+	+	—	
					Orthopnœa, no anasarca, lungs clear										
			3 wks	later	—	—	—	—	13	4	+	+	+	—	
7	M. E. *F.* 20	?	1	—	13	3	Systolic Mid-diastolic	—	10	6	+	+	+	—	Heart weighed 600 grms. Mitral and tricuspid stenosis. Adherent pericardium. Advanced nutmeg liver.
					Orthopnœa, anasarca, and ascites										
8	E. B. *M.* 14	7	3	—	12	3	Systolic Pre-systolic	—	8	6	+	(+)	+	—	Mitral and tricuspid stenosis. Heart weighed 572 grms. Nutmeg liver.
					Orthopnœa and anasarca										
9	A. C. *F.* 40	30	7	—	15	4	Systolic	—	8	8	+	+	+	—	
					Orthopnœa, anasarca, auriculo-fibrillation										
10	J. H. *M.* 31	16	3	—	14	3	Systolic	—	8	8	+	+	+	—	Tricuspid and mitral endocarditis. Well-marked nutmeg liver. Heart weighed 590 gms.
					Orthopnœa, no anasarca, auriculo-fibrillation										
11	E. P. *F.* 35	25	9 wks	—	13	3	Systolic	—	5	13	+	(+)	+	—	
					Orthopnœa, no anasarca, slight œdema of lungs										

No. of Case.	Name, Sex, Age.	HISTORY. 1st Attack of Rheumatism	1st Attack of Heart Failure.	Jaundice.	HEART. Percussion. L. of Sternum.	R. of Sternum.	Mitral Bruits.	Aortic Bruits.	LIVER. Size. Below Ribs. R. Nip. Line.	Above Umbil. Mid. Line.	Tenderness	Pigmentation.	URINE. Urobilin.	Bile Pigments.	AUTOPSY.
		Years	ago.		cms	cms.			cms.	cms.					
12	M. C. *F.* 42	?	2 mths	—	11	1	Systolic	—	3	13	+	2+	+	—	
		Dyspnœa on standing, no anasarca													
			3 wks	later	—	—	—	—	Just	palpable	—	2+	(+)	—	
13	J. N. *F.* 46	38	1	—	13	3	Systolic Presystolic	—	Just	palpable	+	(+)	+	—	Heart weighed 584 grms. and there was moderate stenosis of the mitral orifice.
		Dyspnœa on walking, no anasarca													
14	A. D. *F.* 12	?	3 mths	—	9	1	Systolic Presystolic	—	Just	palpable	+	(+)	+	—	
		Dyspnœa on walking, no anasarca													
15	A. W. *F.* 13	4	1	—	13	2	Systolic Presystolic	—	6	5	—	(+)	(+)	—	
		Orthopnœa, anasarca and ascites													
16	A. G. *F.* 55	?	1 mth	—	10	3	Systolic Middiastolic	—	Just	palpable	(+)	+	(+)	—	
		Orthopnœa, no anasarca, slight œdema of lungs													

No. of Case.	Name, Sex, Age.	HISTORY.			HEART.				LIVER.			Pigmentation.	URINE.		AUTOPSY.
					Percussion.				Size.						
		1st Attack of Rheumatism	1st Attack of Heart Failure.	Jaundice.	L. of Sternum.	R. of Sternum.	Mitral Bruits.	Aortic Bruits.	Below Ribs. R Nip. Line.	Above Umbil. Mid. Line.	Tenderness		Urobilin.	Bile Pigments.	
		Years	ago.		cms.	cms.			cms.	cms.					
17	S. H. *F.* 34	6	1 week	—	9	1	Systolic Mid-diastolic	—	Not	palpable	—	—	(+)	—	
		Orthopnœa, no anasarca, marked œdema of lungs													
18	F. M. *F.* 53	10	6	—	13	3	Systolic Pre-systolic	—	Just	palpable	—	—	—	—	
		Dyspnœa, no anasarca													
19	M. G. *F.* 17	8	6	—	13	3	Systolic	—	Not	palpable	—	(×)	—	—	
		Dyspnœa on walking, no anasarca													
			5 mths	later	14	3	Systolic	—	Just	palpable	+	(+)	+	—	
		Orthopnœa, marked anasarca													
20	M. C. *F.* 14	4	3 mths	—	10	1	Systolic	—	Not	palpable	—	—	—	—	
		No signs of heart failure													
21	A. S. *F.* 12	4	1	—	10	2	Systolic Mid-diastolic	—	Not	palpable	—	—	—	—	
		Dyspnœa on standing, slight anasarca													

Liver Tenderness.—It will be seen that the changes in liver tenderness and in urobilinuria are strictly parallel to each other when one passes from patient to patient, or examining a single patient traces his progress from heart failure to partial compensation (Cases 2, 4, and 12), or from a state of cardiac equilibrium to one of heart failure (Case 19).

Size of the Liver.—No close relationship can be seen between the size of the liver and the degree of urobilinuria, and the following grave discrepancies may be noted. Case 5. The liver was only 3 cm. below the costal margin, but urobilinuria was 2+, whereas in Case 15 the liver was 6 cm. below the costal margin, and there were mere traces of urobilin in the urine. Many similar instances are apparent from the table.

Pigmentation.—There is no accurate agreement between the degree of cutaneous discolouration and the amount of urobilin excreted, and the following cases may be instanced :—

Cases 4 and 12. Pigmentation was very well marked, but only traces of urobilin were found in the urine.

Case 5. Pigmentation was doubtful, but the urine contained large quantities of urobilin.

When observations were made on the same patient at different times the pigmentation was found to be almost constant in degree, whilst the urobilin excretion varied greatly (Cases 4, 12, and 19).

Skin was obtained from pigmented patients who had died from heart failure (Cases 7, 10, and 13), and the urobilin extracted by the following technique. The skin and subcutaneous fat were separated as completely as possible from each other and extracted separately with alcohol and ammonia for a week. Equal volumes of water were then added and the solutions made acid with acetic acid and filtered. The filtrates were extracted with chloroform to which, after separation, two volumes of ether were added. The resulting solutions were shaken with very dilute ammonia. The aqueous extracts were acidified with acetic acid

and urobilin removed with amyl alcohol. In this way pale brown liquids were obtained, showing faint absorption bands in the green portion of the spectrum. An alcoholic solution of zinc acetate was added and a green fluorescence detected by passing parallel rays of light from an arc lamp through the liquid, thus confirming the presence of small amounts of urobilin. But the quantity was disproportionately small when compared with the degree of pigmentation, and similar amounts were obtained from the skin of a normal man who was brought into the hospital dead from drowning and in whose viscera no pathological changes were found other than those caused by the fatal accident. About 100 sq. cm. of skin were used for each experiment. When reagents were applied directly to the pigmented skin, reactions followed which suggested that the pigment was a melanin. Thus, 10 per cent. ferric alum darkened it, as also did 10 per cent. caustic soda and caustic soda with a little sodium nitroprusside.

Conclusions.—Urobilinuria serves as a delicate indication of over-distension of the hepatic venules caused by partial failure of the heart's function. Pigmentation of the skin in patients who have suffered from several attacks of heart failure is due to a deposition of melanin.

We wish to thank the members of the Staff who have so kindly given us opportunities for making observations on patients under their care.

THE CLINICAL SIGNIFICANCE OF RESPIRATORY VARIATIONS.

By

M. S. PEMBREY AND J. H. RYFFEL.

THE mutual recognition by physicians and physiologists of the value of their special work is necessary for the further advancement of their sciences, for, with the increase of knowledge it is impossible for anyone to be cognisant of the whole range of any single subject. Respiration affords an excellent example of the results which may be obtained by such combined work. In the present paper an attempt will be made to show how the knowledge of disease may be advanced by a study of respiration from the physiological point of view.

The respiratory movements have a two-fold significance: they indicate the condition of the nerve-cells of the respiratory centre in the medulla oblongata and the composition of the blood. Local variations in the movements of the thorax are, as a rule, only indications of local changes in the pulmonary system. The rate, depth, and rhythm of breathing are recorded in the ordinary routine of clinical examinations, and the diagnosis of abnormalities in the thoracic organs has been elaborated by the different methods of inspection, palpation, percussion, auscultation, and more recently by examinations by means of the X-rays. The clinician has tended to over-rate the importance of morbid anatomy and underestimate the value of physiology; this may be due in part to the fact that he spends far more time in the post-mortem room than in the physiological laboratory; in part, it may be the fault of the physiologist who has neglected the appli-

cation of his experimental results to clinical work. The full significance of variations in the rate of respiration is still unknown and will so remain until it is considered worthy of more attention than a record made by the nurse. The depth of breathing receives quite inadequate consideration; the volume breathed per minute is very rarely determined. A graphic record of the respiratory rhythm is not difficult, and the neglect of the stethograph must be due to an underestimate of its value.

Respiration is the intake of oxygen and the output of carbon dioxide. The determining factor in the absorption of oxygen and the discharge of carbon dioxide is the living tissue, and the main purpose of the pulmonary ventilation is the supply of oxygen to the blood and the removal of carbon dioxide, so that the purified blood may serve as the medium in which the gaseous exchange of the tissues, the so-called *internal* respiration, can take place. It will be of value to trace briefly the gradual development of this knowledge and thereby to show how the oscillations in opinions[1] concerning the regulation of the breathing have in some cases explained and in others obscured the significance of respiratory variations.

Aristotle held the view that respiration drew air into the heart and arteries and thus cooled the blood. This opinion remained as the authoritative one for many years, even until the fifteenth century. A truer insight came directly the experimental method was employed by the founders of the Royal Society, and it is of special interest in these days when many of our countrymen think that scientific knowledge is generally imported from Germany to note that the chief work on respiration has been due to English observers. At a meeting of the Royal Society in 1667, Hooke[2] showed the following experiment: The ribs and diaphragm of a dog were cut away and the trachea was connected with a pair of bellows. The dog went into convulsions, but revived when air was blown into the lungs. Numerous small holes were made in the surface of the lungs, and by means of two bellows the lungs were kept distended with fresh air; the dog lay still and its heart beat regularly. A piece of lung was cut off and it

was noted that the blood circulated even when the lungs were collapsed. From these observations Hooke came to the conclusion that the cause of death after the opening of the thorax was not the stoppage of the circulation, but the want of a sufficient supply of fresh air. A somewhat similar experiment had been shown previously by Croon[3] before the same Society; he strangled a pullet until it gave no signs of life, and then restored it by blowing air into its lungs.

Boyle,[4] in 1666, proved by numerous experiments with the air-pump that fresh air was essential to life, both animal and vegetable, and he was of the opinion "that the depuration of the blood was one of the ordinary and principal uses of respiration." About the same time Mayow[5] discovered the true function of respiration. He showed that air was a mixture and that one of its constituents, which he called the nitro-aerial gas, was necessary for the support of a flame and all forms of life. The nitro-aerial gas, which we now call oxygen, combined with sulphur and other substances with the production of acids, and during calcination combined with metals, and thus increased the weight of the product. Mayow saw the analogy of respiration to combustion, and held that the function of respiration was to absorb the nitro-aerial gas and remove the vapours arising from the blood. He maintained that the placenta was to be regarded as a lung from which the umbilical vessels take up the nitro-aerial gas and carry it to the fœtus, and he recognised that the embryo of a chick absorbed the gas through the porous shell of the egg during the progress of incubation.

The Reverend Stephen Hales[6] made, about the year 1726, the classical vivisections which laid the foundations of the knowledge of the dynamics of the circulation. In addition to many other subjects he studied the respiration of animals and man and found by experiments upon himself that air was absorbed during respiration and "noxious vapours" were produced by re-breathing air in a bladder. These noxious substances could be removed by potash and the air rendered fit for breathing. He suggested, therefore, the use of a bladder of air and such an absorbent in

the foul air of coal mines and thus anticipated the modern rescue-apparatus. Hales believed that during respiration the air cooled the blood and removed aqueous vapour and noxious substances, but he rejected the view of Mayow that the blood combined with the nitro-aerial gas. About thirty years later, in 1757, Black[7] discovered that a quantity of "fixed air," for so he called carbon dioxide, was given off from the lungs during breathing, and he proved that the addition of that gas was the chief difference between expired and inspired air. Animals placed in the gas died of suffocation.

In 1772 Priestley[8] published his "Observations on Different Kinds of Air," in which he showed that growing plants restored the property of supporting life to air vitiated by the respiration of animals or by the burning of a candle. He also discovered that carbon dioxide was produced by putrefaction and by plants during the night time. Priestley isolated oxygen and nitrogen and showed that the change of colour in venous blood on exposure to the air was due to the action of oxygen, and this change took place even when the blood was separated from the air by a moist membrane and by the walls of the blood-vessels in the lungs. He concluded that respiration deprived the air of a portion of its oxygen and imparted to it a quantity of aqueous vapour and "phlogiston." Priestley's views were obscured by his acceptance of the famous, but erroneous, theory of phlogiston which was propounded by Stahl about the year 1697. Phlogiston was the material and principle of fire, not fire itself, and in the combustion of a phlogisticated substance phlogiston was driven off, and the substance lost weight and became dephlogisticated.

The credit of overthrowing the old theory of "phlogiston" is due to Lavoisier,[9] who extended and explained the discoveries of Mayow, Black, and Priestley. He observed, as Mayow had done a hundred years before, that the calcination of metals was due to a combination with oxygen, but in respiration oxygen was not only absorbed, but combined with carbon to form carbon dioxide. In conjunction with Laplace he proved experimentally that animal heat arose from a process of combustion, oxygen, as

they thought, combining with carbon in the blood. Lavoisier[10] held that the seat of combustion was in the lungs, but in earlier writings he had admitted that it might be in the other organs of the body. The demonstration that the essential seat of respiration is in the living tissues and not in the blood is due to the modern work of Pflüger[11] and his pupils.

By these stages the knowledge has been obtained that the purpose of respiration is the supply of oxygen needed by the tissues and the removal of the carbon dioxide produced by them. It now remains to discuss briefly the controversy concerning the means whereby the pulmonary ventilation is regulated to fulfil this purpose. Legallois[12] (1824) and Flourens[13] (1842) localised the respiratory centre in the medulla oblongata by experiments so clear that there is general agreement at the present time that the rhythm of the respiratory movements depends upon the integrity of a small area of the floor of the fourth ventricle near the point of the calamus scriptorius. The rhythmic activity of the bilaterally symmetrical centres might be due to automatic chemical changes in the cells, or it might be the response to nervous impulses and to changes in the composition of the blood. Upon this question a long controversy has been maintained, and so numerous are the experiments which have been made by different observers that a special article would be needed if justice were to be done to the subject. Only an outline can be given now. Pflüger[14] and his pupils recognised the gaseous composition of the blood as the important factor, and found that an increased pressure of carbon dioxide and a diminished pressure of oxygen in the blood could act as the stimulus to the respiratory centre. On the other hand, the theory of reflex regulation by means of impulses passing up the vagus nerve to the respiratory centre was based upon the important experiments performed by Hering and Breuer[15] in 1868 and those carried out by Head[16] twenty years later in Hering's laboratory. The stimulation of the endings of the vagus produced by the distension of the lungs during inspiration evoked expiration, and the expiratory condition in turn induced inspiration. This theory of the regulation

of the pulmonary ventilation appealed strongly to, and was keenly supported by, physiologists of the mechanistic school, but the old view that the gaseous composition of the blood was the determining factor retained its hold upon physicians. Recently a reaction in physiological opinion has been brought about by the important work of Haldane and his pupils. The simple and efficient method devised by Haldane[17] for the collection of the alveolar air of man furnished a means of examining the regulation of the pulmonary ventilation, and it was proved that the pressure of carbon dioxide was the determining factor. The respiratory centre responded with great delicacy to carbon dioxide, but was uninfluenced by wide variations in the pressure of oxygen. For a time the reaction appeared to go too far, and carbon dioxide became *the* chemical messenger or hormone of respiration.

The purpose of respiration is the supply of oxygen as well as the removal of carbon dioxide, and on general principles it is to be expected that the respiratory movements are excited by other stimuli as well as carbon dioxide. Lack of oxygen was recognised by Pflüger[18] to be associated with increased breathing, and he suggested nearly fifty years ago that reducing substances produced under such circumstances acted as the actual stimulus. Geppert and Zuntz[19] concluded that during the hyperpnœa of muscular work some product of muscular activity was absorbed by the blood and carried to the medulla oblongata where it stimulated the respiratory centre to increased activity. Löwy[20] came to a similar conclusion, and Lehmann[21] showed that the intravascular injection of a solution of tartaric acid stimulates and quickens the respiration of a rabbit, whereas a solution of sodium hydrate depresses the respiratory centre. Further confirmation that carbon dioxide is not the only acid which can stimulate the respiratory centre was found in the characteristic breathing of diabetic coma; the tension of carbon dioxide is then abnormally low and the deep breathing is to be attributed to oxybutyric acid or other products acting along with carbon dioxide. Such evidence cannot be dismissed on the ground that

the conditions are pathological. Gradations between an acidosis which is physiological and one which is pathological can be obtained. The production of lactic and other acids during muscular work is to be regarded not as an indication of a defective supply of oxygen, but as the discharge of a metabolite which will stimulate the respiratory and other centres for the benefit and protection of the organism.

The purpose of breathing is the supply of oxygen to, and the removal of carbon dioxide from, the blood. A regulated pulmonary ventilation would be inefficient unless it were accompanied by appropriate changes in the circulation of the blood. This accommodation appears to be effected both by nervous impulses and by chemical stimuli acting directly upon the heart and blood vessels or reflexly by means of the cardiac and vasomotor nerves. The heart and lungs must work in harmony, and in this connection the fact that both of these organs receive afferent and efferent nerves from the vagus is not without significance. Disturbances in breathing may be due primarily to disorder either of the heart, the blood, or the lungs, but sooner or later all are involved. Apart from complete recovery the defect may be compensated by changes in any one or any combination of the three.

By the process of action and reaction there appears now to have arrived a general agreement that the respiratory movements are regulated both by the chemical composition of the blood and by nervous impulses. This combination of rival views is supported not only by the recent physiological experiments of Scott,[22] but also by clinical observations. In many respects the position is not a new one, but a restatement of older views in more exact terms.

The Composition of the Alveolar Air and its Significance.— Before the work of Haldane and Priestley[23] it was possible to form only a rough estimate of the composition of the alveolar air based upon analyses of the expired air and determinations of the "dead space" of the respiratory passages. The lung catheter of Pflüger was a defective method and from its nature was

limited in application. There is no doubt that the introduction by Haldane of a simple and efficient method of collecting samples of the alveolar air of man will always remain as a landmark in the history of respiration. A sample of alveolar air is obtained by expiring deeply through a piece of rubber hose about four feet long. The air of the respiratory passages, the so-called "dead space," is driven by the deep expiration through the tube, and there is left in the tube air from the alveoli of the lungs. A portion of this air is collected in a vacuous sampler which is attached to the tube near the mouth-piece. The normal composition of alveolar air is in volumes per cent. :—carbon dioxide 5·6 mean, with a range from 6·3 to 4·7 in different subjects; oxygen 14·9 mean, with a range from 13·9 to 15·6. The pressure of the gas is calculated as follows :—

$$\frac{760 - 46 \times 5{\cdot}6}{100} = 40 \text{ mm. (approx.)}$$

760 = mm. Hg. atmospheric pressure.

46 = mm. Hg. tension of aqueous vapour for alveolar air saturated at 37°.

5·6 = percentage of gas found by analysis.

The oxygen of the blood is derived from the air in the alveoli; the carbon dioxide expired comes from the pulmonary blood and ultimately from the tissues. Alveolar air contains about 14 volumes per cent. of oxygen and 5·6 volumes per cent. of carbon dioxide; the tissues have little or no free oxygen and are constantly producing carbon dioxide. The medium of exchange is the blood and there would appear to be sufficient causes, both physical and chemical, to determine the passage of the oxygen inwards and the carbon dioxide outwards, thus :—

Oxygen, Alveolar air ⟶ Blood ⟶ Tissues.

Carbon Dioxide, Tissues ⟶ Blood ⟶ Alveolar air.

The composition of the alveolar air gives a measure of the pressures or tensions of the carbon dioxide and oxygen in the blood leaving the lungs. It would not be correct to say an exact measure, for there has been a strenuous controversy over

the question whether the gases pass by simple diffusion between the pulmonary capillaries and the alveolar air. Bohr[24] maintained that the alveolar epithelium possessed a secretory activity whereby oxygen could be driven into, and carbon dioxide out of, the blood. As regards the secretion of oxygen, Bohr was supported at first by Haldane and Lorrain Smith,[25] but recently Haldane and Douglas,[26] as the result of further experiments, have modified this view, and have arrived at the conclusion that the active secretion only occurs in response to the stimulus of want of oxygen. Further doubt has been thrown upon the secretory theory by the experiments of Krogh.[27] This physiologist, with a whole-hearted belief in Bohr's views, attempted to study the process of gaseous secretion, but failed to find any evidence of such activity, and was forced to conclude that the process as regards both carbon dioxide and oxygen was one of diffusion. Thus, a return has been made to the old views of Pflüger, which are supported by the experiments of Fredericq,[28] Zuntz, Loewy, and others. The balance of physiological opinion at the present time is in favour of the view that the composition of the alveolar air affords a practical measure of the pressures of the gases in the blood leaving the lungs. It has been shown already that the chief factor in the regulation of the breathing is the pressure of carbon dioxide in the blood supplying the respiratory centre in the medulla oblongata. It follows, therefore, that the composition of the alveolar air affords the most important indications of the significance of respiratory variations in health and disease. Care, however, is necessary in the application of this method and in the drawing of conclusions in certain conditions, physiological as well as pathological. A disturbing factor is present if the supply of blood to the respiratory centre is defective.

Carbon dioxide (CO_2) is present in the blood as carbonic acid (H_2CO_3); for this reason it is important to consider, in the first place, the reaction of the blood.

Reaction of the Blood.—The reaction of normal living blood and tissues is, according to physical measurements, very nearly neutral, the hydrogen and hydroxyl ions therein being nearly

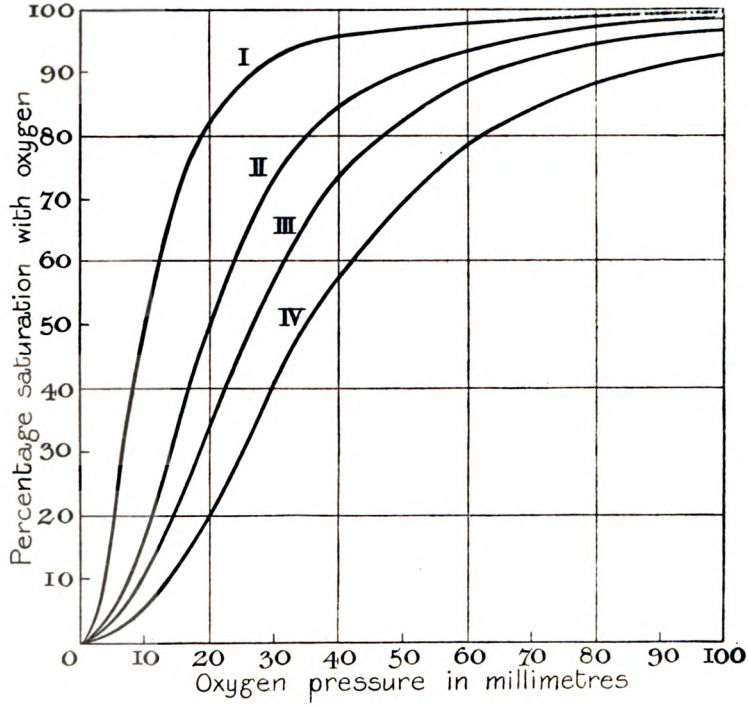
balanced. The fact that respiration is stimulated by the injection of acids into the blood stream and inhibited by the injection of alkalies shows that the respiratory centre is stimulated by the presence of hydrogen ions in the blood. Now an increase of carbon dioxide in the arterial blood stimulates the respiratory centre, and as carbon dioxide in solution is an acid, though a feeble one, it is reasonable to suppose that its action on the respiratory centre is dependent on its being an acid. By means of the hydrogen electrode, Hasselbalch and Lundsgaard[29] have recently shown that even small increases of the pressure of carbon dioxide to which blood is exposed increase the concentration of hydrogen ions therein, and have produced direct experimental evidence to show that the activity of the respiratory centre is normally regulated by the concentration of the hydrogen ions in the arterial blood.* Seeing that the pressure of carbon dioxide in the arterial blood as evidenced by the alveolar carbon dioxide is, under ordinary circumstances, fairly constant, it follows that the balance of other acids and bases in the blood is also kept constant by the action of the organs that excrete them, *i.e.*, mainly by the kidneys.

Measurements of the concentration of hydrogen ions must be the final criterion of acidity, but there is another method which is easier to apply and leads to the same conclusions, namely, the determination of the dissociation curve of oxyhæmoglobin in the blood as developed by Barcroft and his collaborators.[30] For this purpose a sample of blood is taken, whipped to remove fibrin, and exposed in a tonometer at 38° C. to a mixture of oxygen, nitrogen, and carbon dioxide, containing oxygen at a known pressure, about 30 mm. Hg., and carbon dioxide at the alveolar pressure of the subject. The percentage saturation of the hæmoglobin of the blood is then determined by means of the ferricyanide apparatus. In the earlier observations the percen-

*In this connection it is to be noted that in the presence of large excess of protein all acids behave as weak acids, the strong acids being largely neutralised by combination with the protein, so that carbonic acid is not much less efficient in producing a small rise of hydrogen ion concentration than is even hydrochloric acid.

tage saturation was determined at several different pressures of oxygen, but later it was found that accurate determination of one point was all that was necessary, other points being determined by means of a formula.*

In the figure, Curves i., ii., iii., and iv. represent the dissociation of the oxyhæmoglobin in the blood of a normal individual when it is exposed to pressures of oxygen up to 100 mm. and carbon dioxide at pressures 0, 20, 40, 90 mm. respectively, curve iii. being the normal curve at the alveolar carbon dioxide pressure.



* This formula is :— $\frac{y}{100} = \frac{k x^n}{1 + k x^n}$

Where y is the percentage saturation at the pressure of oxygen x mm., n is a constant which does not vary for human blood and $=2{\cdot}5$; k is determined by the observation which is made, so that other points can be calculated.

It will be seen that as the pressure of carbon dioxide increases, the blood takes up oxygen with greater difficulty and gives it up with greater ease. Lactic and other acids have a similar effect to that of carbon dioxide, while alkalies have the opposite effect. Starting with no carbon dioxide, the addition of lactic acid to make 0·12 per cent. in the blood has an equal effect to that of 40 mm. CO_2, and 0·17 per cent. lactic is equivalent to 90 mm. CO_2.

The dissociation curve in the presence of the alveolar carbon dioxide pressure is, under ordinary conditions, constant for a given individual, and, as will be seen, is less subject to variation than his alveolar carbon dioxide, but there is a considerable variation between different individuals, the normal limits of the percentage saturation at 30 mm. of oxygen being 50 and 64 per cent., so that unless the normal curve of an individual is known we cannot say for certain that his blood under any circumstances is abnormally acid or abnormally alkaline, unless the dissociation curve lies outside these limits.

We are now in a position to consider the causation of the various physiological and pathological changes in pulmonary ventilation.*

Respiratory Variations in Health.

Muscular Exercise.—During moderate exercise the ventilation of the lungs and the output of carbon dioxide are increased. The alveolar carbon dioxide is raised by a few millimetres, and the resulting slight acidosis of the blood is sufficient to account for the increased pulmonary ventilation. When the exercise ceases the pulmonary ventilation and alveolar carbon dioxide fall rapidly to normal.[31]

*Determinations of acidity with reference to respiration should be made on arterial blood. As this is not at present obtainable from man, blood is taken either from a vein of the forearm or from the finger, the assumption being made that when the arm is kept still the blood so obtained does not differ appreciably from arterial blood except as to the gases it contains.

The results of hard exercise are more complicated. Hyperpnœa is most marked soon after the start and the alveolar carbon dioxide at this period is considerably increased. Later, during the period of "second wind," the hyperpnœa is still great, but the alveolar carbon dioxide, though variable, is, in general, but slightly greater than normal. When the exercise ceases, the hyperpnœa takes some time to disappear and the alveolar carbon dioxide falls below normal, returning to normal in about three-quarters of an hour.

Thus, in subject B. the alveolar carbon dioxide, which at rest was 43 mm., was, after a run of 440 yards, increased to 58 mm., and after a run of 1,050 yards further it had fallen to 53 mm.; that of R., which at rest was 38 mm., was, after a run of 360 yards, increased to 53 mm., but after 440 yards more had fallen to 42 mm.; that of D. was at rest 38 mm., but after a run of 300 yards in 52 seconds the following results were obtained: 10 seconds afterwards, 46 mm.; 10 minutes afterwards, 36 mm.; 15 minutes afterwards, 33 mm.; 43 minutes afterwards, 38 mm. [32, 33]. The discrepancy between the alveolar carbon dioxide and the pulmonary ventilation is due to lactic acid formed in the muscles which makes its way into the blood and helps to render it more acid, so that there is a marked acidosis. Thus the alveolar carbon dioxide of a healthy man had, immediately after climbing 1,000 feet in twenty minutes, fallen from 40 mm. to 35 mm., but the saturation of his blood at 30 mm. of oxygen had fallen from his normal 62 per cent. to 47 per cent, and the lactic acid in his blood had increased from the normal 0·014 per cent. to 0·046 per cent. Another subject, as a result of the same climb, showed an increase of the lactic acid in his blood from 0·016 per cent. to 0·080 per cent.[34] The excess of lactic acid is partly excreted by the kidneys and sweat glands, partly destroyed in the body, but its disappearance is slower than the removal of carbon dioxide by the lungs, hence the alveolar carbon dioxide falls below normal after the exercise. The hyperpnœa of hard exercise is thus the result of an acidosis partly due to carbonic acid, but mainly to lactic acid.

Temperature.—When the rectal temperature rises, the output of carbon dioxide is increased, but the alveolar carbon dioxide falls.[35] Even without producing any noticeable change in the rectal temperature, a high external temperature causes a slight fall in the alveolar carbon dioxide, while a low external temperature causes a rise.[36] When the alveolar carbon dioxide falls as a result of high external temperature, the blood becomes more alkaline,[37] so that the change is not in the composition of the blood, but in the excitability of the respiratory centre. Where the internal temperature is raised this change may account for the increased excitability of the centre, but changes in the circulation as a result of vaso-motor phenomena in the skin and the effects of cutaneous stimulation have also to be considered.

Forced Respiration.—Voluntary forced breathing, by increasing momentarily the output of carbon dioxide without increasing its formation to the same extent, denudes the body of carbonic acid and lowers the alveolar carbon dioxide. Presumably there is no acid to take the place of the lost carbonic acid, so that the blood becomes more alkaline. When the voluntary control is removed respiration is diminished or stops altogether until the carbonic acid has returned to normal.[38] Where, as in this case, carbon dioxide is lost without other acid to take its place, the constitutional disturbance is much greater than where hyperpnœa is the result of acidosis. Apparently increased alkalinity of the blood, even if very slight, is an undesirable phenomenon, and forced breathing exercises are not to be recommended.

Lack of Oxygen.—Haldane and Poulton have shown that breathing air poor in oxygen for a short time causes increased respiration and a rapid fall in the alveolar carbon dioxide.[39] Similar experiments do not cause an appreciable rise in the lactic acid of the blood,[40] but unfortunately no determinations of the acidity of the blood have been made under these conditions. Barcroft, however, has shown an increased acidity of the blood in animals as a result of lack of oxygen, and twenty years ago Araki demonstrated the formation of lactic acid in

animals as a result of long exposure to an atmosphere sufficiently poor in oxygen to cause unconsciousness. Although lack of oxygen sufficiently marked and sufficiently long continued thus causes an acidosis, the possibility of lack of oxygen acting as a direct stimulus to the respiratory centre irrespective of the reaction of the blood cannot be said to have been excluded.

Residence at High Altitudes.—The changes in the blood and respiration caused by residence at an altitude sufficiently high to cause symptoms of mountain sickness have recently been the subject of two expeditions, one organised by Haldane,[41] the other by Barcroft.[42] The alveolar carbon dioxide falls rapidly at first and then more slowly for the first week or two until compensation is established. This is the result of a definite hyperpnœa and has the beneficial result of increasing the pressure of oxygen in the lungs. At the same time the hæmoglobin of the blood is increased both in concentration and in total quantity. According to Haldane there is another change produced in that the pulmonary epithelium begins to secrete oxygen into the blood, but this matter must still be considered *sub judice.*

If there were no change in the balance of bases and acids other than carbonic acid in the blood, the diminution in alveolar carbon dioxide would result in a more alkaline blood, but, as a matter of fact, the blood is slightly more acid than normal, the change corresponding with the increase of respiration. There is no appreciable increase in the lactic acid and acetone bodies of the urine. The lactic acid of the blood, though somewhat increased at first, falls to normal when compensation has been established and the alveolar carbon dioxide has reached its minimum, so that excessive formation of lactic acid or the acetone bodies is not the cause of the acidosis.* Now, when compensation has been established and signs of lack of oxygen have disappeared, the acidosis is greatest, so that it is scarcely possible

*This statement applies only to the body at rest. There is evidence that exercise causes a more marked increase of the lactic acid of the blood than would the same amount and degree of exercise at sea-level.

that there is formation of any acid product of incomplete oxidation to account for the change. The only other alternative is that the kidneys excrete less acid or more base until a new balance of bases and acids other than carbonic acid is arrived at, which is more in the acid direction than normal. This may be described as a retention acidosis, but it is beneficial in that the oxygen in the lungs is thereby increased and must be attributed to reaction and not failure on the part of the kidneys.

Respiratory Variations in Disease.

The following selection of examples of respiratory variations in disease has been made from cases which, with one or two exceptions, have been investigated in Guy's Hospital, and no attempt has been made to consider the literature bearing upon the subject.

Diabetes.—In this disease results so definite have been obtained that it is possible to estimate the acidosis* from the composition of the alveolar air and thus to obtain the best evidence upon which to base prognosis and treatment. The severity of the disease cannot be measured by the amount of sugar discharged in the urine, and determinations of the acetone bodies show merely the total amounts of these substances which have been excreted and do not indicate the degree of acid intoxication prevailing within the tissues. It is true that in cases of low alveolar carbon dioxide in diabetic patients the serum of the blood requires less acid than in the normal to bring it to a given degree of acidity, but this method is open to objection. By the use of the hydrogen electrode, Michaelis and Davidoff[43] have proved that the venous blood of a patient in coma is distinctly acid, and, therefore, it is justifiable in such cases to associate a low alveolar carbon dioxide with acidosis. Estimations of the urinary ammonia indicate the amounts of acetone bodies which

*According to Ehrmann (Berl. klin. Woch., Jan., 1913) a condition resembling diabetic coma can be produced by sodium butyrate; this, he suggests, is due to a specific action upon the nervous system and not to acid intoxication.

have been neutralised in this particular way, but give no representation of the quantity which remains in the tissues. More need not be said here upon this subject which has been considered in a special article by E. L. Kennaway.[44]

A patient suffering from well-marked diabetes may have alveolar air of a composition within normal limits, but if the acidosis increases, the percentage of carbon dioxide in the alveolar air falls, as shown by the following example:—

No. of Case.	Date.	Alveolar Air.		REMARKS.
		Carbon Dioxide. Vols. per cent.	Oxygen. Vols. per cent.	
23	8th July, 1904	5·6	13·4	Diabetic diet; no alkali.
	19th July, 1904	4·5	15·4	

If coma should threaten, the percentage of carbon dioxide in the alveolar air shows a further decrease, but rises as the condition improves.

No. of Case.	Date.	Alveolar Air.		REMARKS.
		Carbon Dioxide. Vols. per cent.	Oxygen. Vols. per cent.	
22	13th June, 1904	2·7	16·8	Diabetic diet. Coma threatening.
	27th June, 1904	3·4	15·4	Diabetic diet, 65 grms. starch and 42 grms. sodium bicarbonate.
	7th July, 1904	5·7	13·7	5th day of diabetic diet and 56 grms. sodium bicarbonate.

During coma the carbon dioxide reaches a very low point, and the gravest prognosis is justified if it falls below two volumes per cent.

No. of Case.	Date.	Alveolar Air.		REMARKS.
		Carbon Dioxide. Vols. per cent.	Oxygen. Vols. per cent.	
21	12th May, 1904	2·39	17·09	Milk diet.
24	3rd Feb., 1905	1·16	19·4	Milk and beef tea. Infused with sodium bicarbonate.
	4th Feb., 1905	1·26	19·83	——
27	11th June, 1906	1·58	19·08	Milk diet.

The administration of sodium bicarbonate raises the carbon dioxide in the alveolar air, and with massive doses a rise to about seven volumes per cent. has been observed.

These are some of the chief results obtained at Guy's Hospital by Beddard, Pembrey, and Spriggs,[45] who concluded that the effective stimulus to the respiratory centre was the increasing concentration of unneutralised acid substances in the blood. Confirmation has been forthcoming in the work of Straub[46] and of Fridericia,[47] and in more recent observations by Kennaway, Pembrey, and Poulton.[48]

In diabetes the respiratory variations are significant not of disease of the lungs or heart, but of an abnormal condition—acidosis—of the blood. The so-called "air-hunger" of diabetic coma is not due to lack of oxygen or excess of carbon dioxide, but to the stimulation of the respiratory centre by other acids as well as carbonic acid. The respiratory centre is so sensitive to acids that a determination of the composition of the alveolar air will indicate the severity of the disease more rapidly and accurately than any other method.

Uræmia.—In a number of cases with albuminuria the blood is more acid than normal, the alveolar carbon dioxide is diminished, and respiration is considerably increased. The cases may be divided into two groups :—

(i.) Those in which the urea of the blood is greatly increased and there are marked pathological changes in the kidneys ;

these may be called cases of true uræmia. Poulton and Ryffel[49] have investigated four of these cases. The urea of the blood ranged from 0·21 to 0·36 per cent., or about ten times the normal. The alveolar carbon dioxide varied from 14 mm. to 25 mm., the percentage saturation of the hæmoglobin in the blood at 30 mm. pressure of oxygen from 37·5 to 45 per cent. The lactic acid of the blood was not above normal.

(ii.) In the other group investigated by Lewis, Barcroft, and others[50] the urea of the blood was not characteristically above normal, nor were the changes in the kidneys of an extreme order. The patients were all 50 years of age or over. They all had thickened arteries, and their hearts showed various pathological changes, but they were only slightly cyanosed. The alveolar carbon dioxide was diminshed and the blood was more acid than normal. In two patients this acidity was of a very high order, and the blood taken on the day of their death contained excess of both lactic acid and urea. In the other patients, however, who were not moribund, neither the lactic acid nor the urea was above normal. The urine contained no excess of lactic acid or of acetone bodies, nor was there any evidence of excretion in the urine of abnormal quantities of any acid. For these reasons the conclusion was drawn that the acidosis, which is the cause of the hyperpnœa, is due to retention, not abnormal formation, of acid. This condition is, in fact, analogous to the results of residence at high altitudes, with this difference that the retention of acid is, in all probability, to be attributed to failure of excretory function on the part of the kidneys.

Capillary Bronchitis.—In this disease there is an obstruction to the free entry of air through the small bronchial tubes. The supply of oxygen would probably be affected more than the discharge of carbon dioxide, for, owing to its solubility, carbon dioxide diffuses more rapidly through the moist tissues and the exudations. In the case investigated[51] there was marked cyanosis and rapid and difficult breathing; the rate of respiration was 49, the pulse 126, the blood pressure 110 mm. Hg., and the temperature in the axilla 101°. Satisfactory samples of the

alveolar air could not be obtained, but the following are the analyses of the two samples taken:—2·28 and 4·07 volumes per cent. of carbon dioxide, and 18·22 and 15·70 volumes per cent. of oxygen. The ventilation of the lungs was $10\frac{1}{2}$ litres per minute when the patient was breathing air; within two minutes this was reduced to 5 litres, when the air was replaced by oxygen. The frequency of respiration fell from 49 to 33, the cyanosis disappeared and the breathing became easy and shallow. There is little doubt, therefore, that in this case the volume and rate of the pulmonary ventilation were determined by the lack of oxygen.

Cardiac Disease.—Difficult breathing and orthopnœa may be outstanding features of cases of heart disease. The factors which determine the rate and depth of breathing appear to be complex. If the cardiac weakness be compensated, when the patient is at rest, the pressure of carbon dioxide in the alveolar air is not much below the normal, but it falls if the patient becomes breathless on slight exertion. In cases uncompensated even during rest, such as failure of the right side of the heart secondary to mitral stenosis, the alveolar air may contain only two or three volumes of carbon dioxide per cent., and the administration of pure oxygen may reduce considerably the volume breathed and remove the cyanosis and distress. The volume breathed appears to be determined by the need of oxygen, but the rate of respiration may remain unaltered by breathing oxygen and would appear to be related to the action of the respiratory system as an auxillary pump for the blood passing from one side of the heart to the other. Further observations upon orthopnœa are necessary and are in progress.

In congenital disease of the heart the cyanosis is different from the ordinary form, and the blue colour of the extremities appears to be due chiefly to the slow passage of the blood through the capillaries and veins. There is evidence of a chronic deficiency of oxygen, and compensation for this defect is shown in the great increase in the number of the red corpuscles per cubic mm. of blood, in the increased percentage of hæmo-

globin and the large pulmonary ventilation. The composition of the alveolar air shows a low percentage of carbon dioxide, especially in those patients who exhibit the most signs of defective compensation. Two examples[52] may be given :—

No. of Case.	Date.	Alveolar Air.		Red Corpuscles. Per cubic mm.	Percentage of Hæmoglobin.
		Carbon Dioxide. Vols. per cent.	Oxygen. Vols. per cent.		
1	6th Aug., 1904	4·6	14·9	13,200,000	145
4	4th May, 1907	2·68	17·31	12,930,000	130

Pernicious Anæmia.—It is interesting that in this disease there is no dyspnœa when the patient is lying down, notwithstanding the great reduction in the percentage of hæmoglobin in the blood. The composition of the alveolar air may show little or no reduction in the percentage of carbon dioxide. The breathlessness on exertion appears to be due to the failure of the heart to pump the blood sufficiently rapidly through the lungs to compensate for the defective oxygen carrying power.

Similar results in this and other diseases have been obtained by Miss Fitzgerald.[53]

Cheyne-Stokes Respiration.—The examination[54] of cases of Cheyne-Stokes respiration from the physiological standpoint has explained the chief features of this well-known type of breathing. The periodicity of the respiration appears to be due to a diminished excitability of the nervous system associated with a defective supply of arterial blood; the carbon dioxide accumulates and the oxygen diminishes until the nerve cells of the respiratory centre are stimulated; the waxing respirations begin and culminate in hyperpnœa, whereby a large quantity of carbon dioxide is washed out and sufficient oxygen is taken in. Apnœa then follows owing to the absence of a sufficient pressure of carbon dioxide to stimulate the respiratory centre. This explanation is supported by the following observations :—

(i.) The inhalation of air containing more than 2 per cent. of carbon dioxide abolishes apnœa; the pressure of carbon dioxide is maintained at its stimulating value.

(ii.) The administration of pure oxygen abolishes apnœa; the pressure of carbon dioxide is maintained, for the respiratory centre is no longer excited by lack of oxygen to send out the forcible impulses which had previously resulted in excessive ventilation of the lungs, whereby carbon dioxide had been washed out of the alveoli and blood.

(iii.) Low percentages of oxygen in the air breathed abolish apnœa; the respiratory centre under this condition at no stage receives a sufficient supply of oxygen, and continuous breathing is the result.

Douglas and Haldane[55] have extended and confirmed this work. They have shown that it is easy to induce Cheyne-Stokes breathing in healthy men, but they did not find any evidence of alteration in the excitability of the nervous system.

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SOME PROBLEMS OF EXPERIMENTAL NEPHRITIS.

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THE connection between mercury and lead and disease of the kidneys in man has long been known, but the test of animal experiment was first applied by Pavy,[67] who in a paper published in the Guy's Reports for 1860 described the changes produced in the kidneys of rabbits by the administration of white precipitate (ammonio-chloride of mercury). His animals showed necrosis of the cells of the convoluted tubules, with deposit of phosphate of lime. The glomeruli were unaffected, and there was no albuminuria. Pavy's work, which was undertaken from the medico-legal point of view, seems to have attracted but little attention, and Saikowski,[84] who in 1866 described the renal changes caused by the experimental administration of calomel and corrosive sublimate to animals, claimed that his work was the first that had been done in that field. Saikowski's paper was followed by a number of communications from other workers, which have been reviewed by Klemperer,[43] all dealing with the histology of mercury nephritis, and more especially with the origin of the calcareous masses, the presence of which had been noted by Pavy. Lead was first employed experimentally by Ollivier[60] in 1863, who found that the carbonate given by the mouth to the usual laboratory animals produced granular and fatty changes in the convoluted tubules, with cast formation.

The next substance that was added to the list of renal poisons, verified by experiment, was chromic acid, which was found by Gergens[82] in 1876 to produce acute nephritis in dogs. Thereafter the number of substances found to be capable of producing nephritis in animals rapidly increased, but all the earlier work was confined to a description of the histology of the lesions so produced, and it was not till some time had elapsed that use was made of experimental nephritis in animals to attempt to solve some of the problems of the disease as seen in man. A great impulse was given to this line of research by Richter's[82.83] discovery in 1904 that it is possible to produce œdema in animals suffering from uranium nephritis, and nearly all the work published since that time has dealt with the problems presented by the pathology rather than the morbid anatomy of the kidney.

The selective action of certain poisons on the kidney, which produce acute necrosis and degenerative changes in certain structures, while leaving others apparently more or less intact, has attracted the attention of physiologists also, who have made use of it with the idea of deciding some as yet unsettled points in the mode of action of the normal kidney. How far, however, such selective action is real and not merely apparent is, as will appear later, still a debated point, and so far experimental nephritis has thrown light rather on the pathology than the physiology of the kidney.

The list of substances that are known to have the power of producing nephritis in animals is a long one. From the point of view of chemical constitution as apart from action they may be classified as follows:—

A. *Inorganic Substances.*—According to Sollmann[95] all metals so far studied cause nephritis. The list he gives includes aluminium, antimony, arsenic, beryllium, bismuth, cadmium, cerium, chromium, cobalt, copper, lead, manganese, mercury, nickel, phosphorus, platinum, silver, tungstan, uranium, zinc, and their salts. To Sollmann's list must be added oxalic acid[25] and the tartrates, which as Underhill[100] has recently shown, are capable of producing severe disintegrative changes in the tubules

of the kidney, a point of some practical importance in view of the frequent use of these salts as saline purgatives in cases of nephritis in man. The substances in the list vary very considerably in their efficiency, for I have found[14] that while potassium chromate in doses of 0·025—0·030 grammes per kilo of body weight produces in rats a nephritis that is fatal in about a week, chrome alum ($Cr_2(SO_4)_3K_2SO_4$), even in doses of 0·2 grammes per kilo repeated at intervals of a week for several weeks, failed to produce any renal change or even a transient albuminuria. Of the above-mentioned drugs only arsenic, chromium, lead, mercury, and uranium have been widely used.

B. *Organic Substances of Known Chemical Composition.*—Of these the most important for various reasons are cantharidin (Browicz[10]), aloin (Kohn[44]), vinylamin (Lindemann[47]), and para-hydroxy-phenyl-ethylamine (Harvey[37]). Alcohol, ether, and chloroform produce chronic changes in the kidney, which have been studied by Emerson,[28] as does also amyl nitrite. To these must be added on the authority of Sollmann all coal-tar products such as cresol, creosote, and the salicylates, all essential oils, santonin, and male fern. Martin and Pettit[53] have recently published the curious discovery that the feeding of rabbits and rats on milk powder only, without other food, produces albuminuria, hæmaturia, and sometimes glycosuria, with casts, while the kidneys show profound degenerative changes in the cells of the convoluted tubules, with some increase of the perivascular connective tissue.

C. *Organic Substances of Unknown Composition, including Living Cells and their Products.*—This class includes bacterial cultures, such as bacillus anthracis, bacillus pyocyaneus, and bacillus prodigiosus (Pernici and Scagliosi[78]), bacillus coli, especially when employed with uranium, when it produces changes of the chronic interstitial type (O'Hare[58]), bacterial products, such as diphtheria toxin, and, of less importance, tuberculin and mallein (Pearce and Eisenbrey[75]), snake venom (Novak[56]), eel serum (Petit[79]), hæmolytic immune serum, and specific nephrotoxic serum, produced in the usual way by the injection of

emulsion of kidney cells of one animal into an animal of another species, whose serum after repeated injections becomes toxic for the kidneys of an animal of the first species. The attempt to produce auto-nephrotoxins by ligature of the vessels of one kidney, and iso-nephrotoxins by the injection of washed kidney cells into an animal of the same species was a failure (Pearce[68]). Nephritis also occurs in rabbits, cats, and dogs, which have suffered from repeated non-fatal attacks of anaphylactic shock produced by the injection of horse serum or egg-white (Longcope[48]). The changes in this case are chiefly tubular and interstitial, and similar interstitial changes are also found in the liver and myocardium. Finally, we have, though it does not fall into any of the above three groups, Fischl's[29] discovery that the repeated production of lordosis by artificial means in animals tends to produce nephritis, a nephritis which may ultimately result in a form of granular kidney of the contracted type.

The two chief portions of the kidney, which are both histologically and functionally distinct, viz., the vascular or glomerular, and the tubular, are not equally affected by all renal poisons, and one of them may be severely damaged, while the other appears to escape all injury, or in other words most nephritis-producing substances have an action which is to a greater or lesser extent selective. They may, therefore, be divided into two groups, the vascular and the tubular. The division may be made on histological grounds, or the separation may be made by the "physiological" method of testing the functional efficiency of the two divisions of the kidney devised by Schlayer and Hedinger[88] Unfortunately from the point of view of classification these two methods of grouping do not give concordant results, and the nephritis produced by certain substances may be anatomically tubular, while when tested physiologically it proves to be mainly vascular in its reactions, or *vice versa*, so much so that Schlayer and Hedinger assert that it is impossible to separate the functionally vascular from the tubular type by histological methods. Of the drugs that have been used most frequently to produce experimental nephritis, and the action of

which has been tested physiologically, the chromates stand alone in that anatomically they produce a lesion of a purely tubular type, and glomerular lesions are always absent, while when tested physiologically the response points to the absence of vascular involvement. They would, therefore, seem to be purely tubular poisons. Two other of the commonly used renal irritants produce a nephritis which is anatomically almost purely tubular in type : these are uranium and mercury. In both cases there is widespread and severe necrosis of the cells of the convoluted tubules, while glomerular lesions, though they undoubtedly occur, as has been pointed out by Christian and others[18] in the case of uranium, and by Lyon[49] in the case of mercury, are slight, and from the histological point of view insignificant. The microscopic picture produced by both drugs is, therefore, very similar, but physiological tests show[88, 89] that in uranium nephritis the reactions are typical of the vascular type of lesion, while the mercury kidney gives reactions that are tubular in type. Diphtheria toxin, which owing to its availability and the frequent presence of evidences, such as albuminuria, of kidney involvement in clinical cases of the disease, has been frequently used in experimental work, occupies a somewhat anomalous position. According to Lyon's careful study, it produces some primary lesions of the glomerular capillaries, to which is added a certain amount of tubular involvement, while, as Schlayer and Hedinger[88] have shown in the case of the rabbit, and Pearce and Eisenbrey[75] in the case of the dog, the reactions in the early stage are tubular, while, as the lesion progresses in severity, they become of the vascular type. The histological picture in arsenic nephritis is chiefly one of affection of the glomeruli, though tubular changes are by no means absent, and the physiological reactions are of the vascular type.[88] It is the nearest approach that is so far known to a nephritis that is both anatomically and physiologically vascular. Of all the substances that produce a physiologically vascular nephritis, cantharidin has probably been most widely employed; the anatomy of the lesion produced was the subject of early studies by Eliaschoff[27] and by Welch[105] who,

however, differ in their interpretation of their findings; the anatomical injury to the vascular mechanism is severe, but, as has been emphasised by Aschoff[1] and by Oertel,[57] and as, indeed, was originally pointed out by Browicz[10] who first discovered its power of producing nephritis in animals, the tubular changes are marked and insistent; and as Oertel has recently demonstrated, the parenchymatous lesions are not confined either in the case of cantharidin or of mercury and uranium, to the kidneys, but are seen prominently also in the liver, a fact of importance when animals suffering from these forms of nephritis are used for experiments in metabolism, the results of which it is hoped to apply to cases of nephritis in man. A nephritis produced experimentally that is purely vascular both anatomically and physiologically is, therefore, unknown, and Aschoff, in the paper already quoted, as the result of his own work and that of Suzuki, goes so far as to say that all renal poisons act primarily on the parenchyma, *i.e.*, on different parts of the convoluted tubules, and that differences in functional disturbances are due to individual differences and complexity in the topographical action of poisons, and their varying effects on blood pressure, and not to injury of specific kidney constituents. Whether it is necessary to go quite so far as this or not, and the reputation of the authors is so high that all they say demands the most careful consideration, it is plain that the histological picture of any experimental kidney lesion gives no sure evidence of the extent of functional damage, and the physiological activity of a structure may be greatly impaired while anatomical evidence of injury is almost entirely wanting. Conversely there may be anatomically severe damage while the functions of the part are still unimpaired.

From the time that Bright published his observations on "Renal Disease accompanied by the Secretion of Albuminous Urine" in 1827, the relationship between the various forms of nephritic kidney seen in the post-mortem room became the subject of acute and prolonged controversy. The earlier view that the shrunken granular kidney is a later and terminal stage of

an acute inflammation was supported by Frerichs and others, but was attacked by Wilks and the English school of pathologists. Gradually the latter view prevailed, and probably all pathologists would now admit that while there is a form of contracted kidney which results after a long time from a previous attack of acute nephritis, such cases are few in number, and the common form of granular kidney, so frequently seen in elderly people, is slow and insidious in its onset, and is the result of a degenerative process whose primary point of attack may be either the blood vessels or the interstitial tissue of the kidney.

With the echoes of this controversy still in the air it was not to be wondered at that, when it was discovered that it was possible to produce in many different ways acute inflammation of the kidney, many workers attempted to produce a granular contracted kidney comparable to that seen in man, by inducing frequent attacks of acute nephritis. Lyon, whose paper has already been referred to, was one of these. Working with diphtheria toxin, corrosive sublimate, and cantharidin, his results in this direction were quite negative. To quote his words: "In no case, and by no variation of the experimental method, have I been able to produce and follow the evolution of changes at all analogous to those which we find in subacute and chronic diffuse nephritis in man. That such lesions may be secondary to an acute inflammation of the kidney cannot be gainsaid. But my results point to the fact that, in animals at least, the kidney is restored to its normal integrity after the subsidence of the acute inflammatory phenomena, and if it be permissible to draw any conclusions therefrom one might, with reasonable assurance, affirm that in man also acute inflammatory changes in the kidney may entirely disappear without the further development of subacute or chronic lesions if the noxious agent be no longer in operation. This is a recognised fact, and indirectly it emphasises the importance of the continuous action of some toxic substance in the slow insidious origin of the great majority of the cases of subacute and chronic nephritis." Lyon's experience does not stand alone. Other workers have experienced the same

failure, a fact which serves to emphasise the point that the injection of any toxic substance, however frequently and carefully repeated, is but a coarse and incomplete reproduction of what takes place when an animal is continually absorbing toxins from a focus within its own body.

Others, however, have been more successful, but all the work is not above suspicion unless controlled by previous observation of the animals from the point of view of the presence of albumin or casts in the urine, as spontaneous nephritis is by no means uncommon in laboratory animals, and Ophuls and McCoy[63] have reported many cases of spontaneous chronic nephritis in wild rats, and point out that the histological picture of the condition very closely resembles that seen in experimental chronic uranium nephritis. Among the earliest workers to claim a successful result was Aufrecht,[2] who by repeated injections of cantharidin into rabbits, produced contracted kidneys similar to those seen in man. A year before Aufrecht's paper appeared there was published an important paper by Charcot and Gombault.[13] These observers gave carbonate of lead to guinea pigs by the mouth over prolonged periods. The animals developed hæmaturia, but albuminuria was not prominent, and probably not more than could be accounted for by the amount of blood in the urine. After shorter periods of administration the kidneys were large and granular in appearance, and on section showed calcareous masses in the lumen of the tubules, as is usual in experimental lead poisoning, with degenerative changes in the tubule cells and flattening of the tubular epithelium. There was also thickening of Bowman's capsule. Where the epithelial changes were most marked there was a proliferation of connective tissue. After longer periods there was considerable proliferation of connective tissue and atrophy of the tubular epithelium, with the same glomerular changes as in the former cases. The kidneys bore a close resemblance to those seen in human cases of contracted kidney. Charcot and Gombault's results are all the more interesting in that one animal showed polyuria, one œdema and cardiac hypertrophy, and several pericarditis, all of which con-

ditions are frequent in the disease as seen in man. All recent work goes to show the greater importance of the inhalation of lead in the form of dust than its ingestion by the mouth in the case of lead poisoning in man, and possibly even more striking results than the above might be obtained in this way, but so far as I know none have been recorded which were undertaken with the hope of producing chronic renal disease.* Lead was also used by Ophuls,[61] who gave it to animals by the mouth over long periods. He found as result necrosis and degeneration of the tubular epithelium, with definite interstitial changes, starting round the blood vessels in the neighbourhood of the glomeruli. The animals showed no albuminuria, with the exception of one dog, and that only after great exertion, which is known to produce albuminuria in healthy persons.

Similar, but more marked, lesions were produced by the same worker with repeated doses of bichromate of potassium, but the percentage of successful results was less constant, and the lesions were more easily produced in rabbits than in dogs. Bichromate was also used by Pearce and his co-workers, with successful results. They also used uranium in rabbits, and in the case of both drugs some of the animals showed hypertrophy of the left side of the heart. About the same time Dickson[23, 24] published his results with uranium nitrate. Working with guinea pigs he produced definite and well-marked interstitial changes, and the kidneys were sometimes found to be small and granular. Albuminuria was sometimes, but not constantly, present. To bring about these changes small repeated doses of the drug were required, but in one case he produced them as the result of a severe attack of nephritis induced by a single dose of uranium. In his second paper he gives more details of the histological changes produced. These were proliferation of the interstitial tissue, more especially in the inner zone of the cortex, with pyknosis and fragmentation of the glomerular nuclei. The capsular

*Goadby's experiments, recorded in Legge and Goadby's recent book on Lead Poisoning and Lead Absorption, London, Arnold, 1912, only deal incidentally with the renal changes.

epithelium was also thickened and proliferated. There was some tubular degeneration and peri-arterial fibrosis. Hypertrophy of the left side of the heart occasionally occurred, and it is especially noteworthy that some of the guinea pigs used showed spontaneous and marked œdema. Siegel's[93] results with uranium are very much the same, only his animals, dogs, never showed œdema. Other substances which have been found to produce interstitial changes in the kidneys of animals are oxalic acid,[25] and bacterial cultures,[78] though here the interstitial change is not very prominent, with the exception of those cases recorded by Morse,[54] who used cultures of staphylococcus pyogenes. In his animals the kidneys had the general appearance of granular kidneys in man, but this result only occurred in three out of a large number of animals, as Morse himself acknowledges, and may have been a spontaneous change. A combination of bacillus coli and uranium has recently been used by O'Hare,[58] with the resulting production of marked chronic interstitial change.

Hypertrophy of the heart was also noted in mice by Ehrlich[26] and Levaditi[45] as the result of repeated injections of vinylamin (CH_2CHNH_2). This substance had been previously used by Lindemann,[47] who found as the result glomerular hyperæmia and necrosis of the renal tubules. He, however, failed to produce chronic interstitial change. According to the first-named authors Lindemann's results were due to impurities in the preparation of the drug he used, and the lesion produced by vinylamin, if pure, is a necrosis of the renal papilla, while the cortical zone is merely hyperæmic or escapes all damage. They had no difficulty in producing chronic changes, and the kidneys of their rabbits showed marked increase of the interstitial tissue with dilated tubules, and in addition to the heart hypertrophy already mentioned, in some of their animals was found albuminuric retinitis, a result of experimental nephritis which has not been reported by any other workers.

Harvey[37] has approached the question of experimental nephritis of the chronic type from another point of view. Acting on the supposition that certain cases of the disease in man are

possibly due to auto-intoxication of intestinal origin, he used para-hydroxy-phenyl-ethylamine, a substance which, under certain conditions, is formed in the alimentary canal of man. It has a marked effect on blood-pressure, causing a considerable rise, and, given over long periods, produces degenerative changes in the arterial wall. Rabbits which received it over prolonged periods, up to a year or more, intravenously or by the mouth, showed in 20 out of 33 cases extensive structural changes in the kidneys secondary to vascular sclerosis. The chief of these alterations were a thickening of Bowman's capsule, sclerosis of the glomerular arteries, a flattened ground-glass appearance of the tubular epithelium, and definite new formation of connective tissue round the damaged structures. Albuminuria was sometimes present, and in ten of the total number of cases the heart was considerably enlarged.

It will be seen that hypertrophy of the heart is by no means rare in chronic interstitial nephritis produced experimentally, and polyuria has been occasionally noted. The question whether the blood pressure is raised in such cases does not seem to have been answered, but in view of the fact that it is frequently raised in the form of the disease seen in man, and that heart hypertrophy occurs, it is more than probable that a heightened blood pressure is present. The reason for these general changes has been the subject of some experimental work. The rise of blood pressure is probably not due to the passage into the circulation of poisonous substances formed by the damaged kidney, as Pearce[73] has shown that extracts of kidneys which are the seat of various forms of nephritis produce not a rise, but a fall of blood pressure, and Ghiron[33] states that in the earlier stages of experimental nephritis, when the process is still acute, there is no rise in pressure. The rise and the hypertrophy of the heart that accompanies it are more probably due to the decrease in the functional capacity of the kidney produced by the strangling of the tubules and glomeruli by the newly-formed connective tissue; for it has been shown by Passler and Heineke[66] that the removal of more than half the substance of the cortex of the kidney in

dogs, with removal of the resulting compensatory hypertrophy of the remaining part of the organ at subsequent operations, produces in those animals which survive long enough and do not become cachectic, a rise in blood pressure, followed by hypertrophy of the left ventricle, with some polyuria, but no œdema or uræmia. Their results confirm those previously published by Paoli,[65] who found hypertrophy of the heart, and by Rose Bradford,[8, 9] who found polyuria after such partial resection of the kidney. It is suggestive also that Ghiron noted that in rats with sublimate nephritis, a rise of blood pressure artificially induced caused the damaged kidneys to act more efficiently, as shown by the increased excretion of water and urinary constituents. The rise may, therefore, be partly of the nature of a protective mechanism for the organism as a whole.

Œdema, both from its importance in the symptomatology of nephritis and because it has been one of the battle grounds on which have fought those who support the mechanist and those who support the vitalist theories of physiological action, has been the subject of many researches. A stray case of œdema in experimental nephritis has been reported here and there, *e.g.*, by Charcot and Gombault in their guinea pigs poisoned with lead, but the systematic study of the condition dates from Richter's[82] paper in 1904. Working with rabbits suffering from acute uranium nephritis, Richter found that it was possible to produce œdema with some constancy by feeding the animals on watery food such as turnips, while it did not follow a dry diet such as oats. The condition could also be induced by giving water or a one per cent. solution of sodium chloride by means of a stomach tube. Phosphates and magnesium sulphate had a similar effect, but to a less degree, while a solution of urea failed to produce the condition. The œdema was general, with excess of fluid in the thorax and peritoneum, but the anatomical peculiarities of the skin and subcutaneous tissues, more especially of the face, prevent the œdema ever being so marked in those situations as it is in man. The excess of fluid is confined to the cavities of the body and to the subcutaneous tissues. The kidneys also con-

tain more fluid than usual, and possibly the wall of the alimentary canal, but the heart and liver show no increase in their water content.[14] This is true of the nephritis due to uranium, but Opie[64] has recently demonstrated the presence of œdema of the liver and gall-bladder in cantharidin nephritis, an observation which may be correlated with the presence of degenerative changes in the liver in that condition recently emphasised by Oertel and others. Richter's observations have been fully confirmed by many other workers, but the possibility of producing generalised nephritic œdema has so far remained confined to cases of uranium nephritis.

The condition of the capillary wall is of primary importance in the mechanism of the production of œdema. Its importance has been recently re-asserted in the case of cardiac œdema by Starling and by Bolton, while Pearce[71] considers that a departure from its normal state is as necessary as is hydræmic plethora or the presence of nephritis itself for the appearance of nephritic œdema. The subject has been put to the test of experiment by Boycott.[6, 7] Starting with the observation that it is possible to produce true hydræmic plethora in rabbits suffering from acute uranium nephritis by the injection into a vein of large quantities, up to the estimated blood volume of the animal, of Ringer's solution, while such proceeding is not followed by plethora in the normal animal, he investigated the effect on the volume of the blood of normal and nephritic animals of the injection of Ringer's fluid of varying strengths. As the result he was led to conclude that acute uranium nephritis decreases the permeability of the capillary wall in both directions, a conclusion that was supported by further experiments where gelatine solutions were substituted for Ringer's fluid. In the case of both the normal and nephritic animals the kidneys were put out of action by ligature of the ureters. The bearing of these observations on the question of œdema is not quite clear, for while œdema may be produced in rabbits suffering from uranium nephritis with some ease, it cannot be produced by the same method in animals whose nephritis is due to

the chromates, and yet I have found,[15] working on the same lines as Boycott, that in such rabbits there is evidence of an alteration in the permeability of the vessel wall such that it hinders the passage from within outwards; hindrance to the passage of fluid in the reverse direction, from the tissues to the vessels, was not definitely proved.

Further experiments showed that this alteration was, in all probability, due to some substance which passed into the blood from the damaged kidneys, and not to the direct action on the vessel wall of the drug used to produce the nephritis. Somewhat akin to this observation is that of Blanck,[4] who found that when rabbits suffering from chromate nephritis were injected with the serum of animals with uranium nephritis they became œdematous. The phenomenon was, however, inconstant, and is discounted by the results of Heineke and Meyerstein,[38] who found that while œdema found its appearance in 64 per cent. of rabbits treated by Blanck's method, it was also produced in 62 per cent. of rabbits suffering from chromate nephritis which were treated with intravenous injections of normal rabbit serum. It is, however, true that the serum of dogs with spontaneous or artificial nephritis, though not that of rabbits, produces albuminuria and casts when injected into healthy dogs[69] and may produce acute hæmorrhagic nephritis,[46] which is not due to the presence of the renal poison, *e.g.*, uranium, in the blood, but to some unknown substance.

The importance of retention of chlorides in the body as a factor in the causation of œdema in human cases of nephritis has been brought into prominence by the French school of physicians, and is supported by the success they have obtained in the treatment of œdema by limiting the intake of chlorides in the diet. Widal and Javal in their book, La Cure de Dechloruration, give striking instances of this, and their results have been repeated in other countries, more especially in America. The factors underlying this chloride retention have been the subject of considerable debate. Three explanations are possible: firstly, that the primary point is the retention of water, and the chlorides

are retained to prevent the salt-content of the tissue fluids becoming too low; secondly, that it is the retention of chlorides that is the controlling factor in the situation, and that water is consequently retained in the body in order that the tissue fluids may not become too concentrated; or, thirdly, that the retention of both chlorides and water depends on some other as yet unknown factor, and that neither is the primary cause of the œdema.

The experimental evidence bearing on the point is very conflicting and uncertain. To support the theory of primary water retention it would be necessary to find a great increase in the water content of the tissues, with only a slight or no increase in the chloride content, while the second theory for its justification requires a high chloride content of the tissues, with some increase in the water content, but not sufficient to bring the concentration of the tissue fluids down to their normal level. It is only the discovery of extreme instances like these that would render one or other theory certain, and these conditions have not yet been fulfilled. According to Richter the administration of NaCl and water to animals with uranium nephritis produces a greater degree of œdema than the administration of water alone, while chlorides alone have no power to increase the degree of œdema. Further, if NaCl is given with half the usual amount of water the degree of œdema is no greater than is seen in animals receiving water only. He, therefore, concludes that the retention of water is more important than the retention of chlorides in the production of anasarca. Schirokauer[86] found that in experimental nephritic œdema the chloride content of the tissues was greater than normal, but this was also true to an equal degree of the fluid found in the thorax and peritoneum. Heineke and Meyerstein[38] state as the result of their researches that the retention of chlorides may precede that of water, while, on the other hand, according to Georgopoulos[31] in uranium as well as in cantharidin nephritis no constant relation exists between the excretion of water and salt; more water than salt is retained, this leading to a decrease in the chloride content of

the blood. This indicates that water retention is dependent on a primary disturbance of the water eliminating power of the kidney, and is not secondary to the retention; moreover, an increase of the chloride in the tissues with a reduction of chloride concentration in the blood could not be demonstrated in animals with or without œdema. It is clear from these very conflicting conclusions that the question of chloride retention has so far received very little light from experimental nephritis.

The problem of chloride retention is closely bound up with the question as to what parts of the kidney are concerned in the secretion of the chlorides and the other constituents of the urine. This and many others of the problems of the functions of the normal and inflamed kidney have been attacked by Schlayer and his fellow workers in an important series of researches, which have already been alluded to, but which must now be described more fully. They have for their basis the close relation that exists between the renal blood supply and the general circulation, and the fact that diuresis depends both on the vascular phenomena, both general and local, and the functional activity of the renal epithelium. Their method of experiment was as follows:—The animal (and rabbits were used throughout) was anæsthetised with urethane. Simultaneous graphic records were then obtained of the kidney volume, the arterial blood pressure, and the amount of urine secreted. They first studied the effect on these records in the normal animal of the application of sensory stimuli, such as tobacco smoke blown into the nostrils, and of the injection of adrenalin, and of diuretics such as 5 per cent. salt solution and caffein. Having discovered the response of the normal animal to these measures, they compared them with the response obtained in rabbits with various forms of experimental nephritis. In the tubular type of nephritis due to chromate or corrosive sublimate they found a primary polyuria, while the blood pressure was well maintained, and the vascular reactions, as evidenced by vaso-dilatation with 5 per cent. salt solution and caffein, and vaso-constriction with adrenalin were normal or slightly increased, becoming only slightly sub-

normal towards the end, even when anuria had set in. On the other hand, in the vascular form of nephritis, due to cantharidin or arsenic, there was no primary polyuria, and the vascular response of the kidney disappeared early with concomitant anuria. In diphtheria toxin nephritis the response at first gave no evidence of vascular involvement, while in the later stages the vascular mechanism was evidently affected and failed to respond to stimuli in the normal manner. Their results with uranium nephritis are of especial interest in view of the occurrence of œdema in that condition. In the early as well as in the later stages there is an absence of vascular response to stimulation, but intermediately there is a stage when the injection of 5 per cent. salt solution still causes dilatation and pulsation of the kidney vessels, but fails to produce diuresis, as it does in the normal animal. This modified vascular response was only noted in uranium animals, with the exception of one case of diphtheria toxin nephritis, and is interpreted by Schlayer as pointing to an alteration in the permeability of the wall of the glomerular capillaries. Later, even this response fails and œdema sets in. The attempt to correlate the alteration in the renal vascular function with the histological picture seen in the kidney has been made by Takayasu,[97] working with the kidneys of Schlayer's animals. He arrives at the conclusion that the impairment of renal vascular function in acute lesions cannot be judged from the anatomical picture. In the vascular type the glomerular changes are parallel to the extent of the physiological impairment of function, while in the tubular type in the early stages there is no evidence of vascular injury. The polyuria of this stage is probably due to increased irritability of the glomerular vessels. Later, when there is physiological vascular disturbance the glomerular changes are very slight, and the vascular disturbance is probably functional, but is not due, with its accompanying anuria, to blocking of the tubules by the swollen tubular cells and the accumulated debris, as it does not occur when the ureters are tied in normal animals. This portion of Schlayer's work with rabbits has been confirmed in most points in the dog by Pearce and his collaborators.[73]

The further results obtained by Schlayer and his school are of more questionable interpretation. Without an anæsthetic, rabbits suffering from tubular nephritis and also from uranium nephritis, showed a diminished power of excreting chloride and iodide, but not of excreting milk sugar. In the cantharidin and arsenic animals death followed so soon that the results are unsatisfactory, but, on the whole, seemed to show that the excretion of milk sugar was diminished, while the percentage of chloride and iodide in the urine was as great as in the case of normal animals. The conclusion drawn was that chloride and iodide are passed by the tubules, while milk sugar is excreted by the glomeruli. It has, however, been shown by Suzuki[96] that in uranium animals the dose of uranium sufficient to destroy all the epithelium of the convoluted tubules also produces anuria, so that as Schlayer's animals still passed urine some of the tubular cells must have escaped injury, and it is possible that the milk sugar was excreted by the still unharmed tubular cells. Further, while it is generally accepted that chlorides are excreted by the tubules, Underhill, Wells, and Goldschmidt,[102] as the result of working with the tubular type of nephritis produced by the injection of tartrates, came definitely to the conclusion that chlorides and water are excreted by the glomeruli, while urea is passed by the tubules.

In further papers[91. 92] Schlayer and Takayasu have applied their experimental results to cases of nephritis in man, with the hope of locating the place of the chief lesion in the kidneys by studying the excretion of chlorides, iodide, and milk sugar, and of affording a means of differentiating a tubular from a vascular type of the disease. Unfortunately, of the cases they investigated in this way only one came to the post-mortem table, consequently it cannot be said that their cases have shed any light on the correlation between the facts observed during life and the appearances in the kidney revealed by the microscope after death, nor have their results in this direction always been confirmed by other observers who have worked with their methods.

The whole question of the seat of excretion of the different constituents of the normal urine has received very little illumination from work on experimental nephritis. For instance, McNider[51, 52] comes to a directly opposite conclusion to that put forward by Underhill and his co-workers, and states that in uranium and other forms of nephritis, including the purely tubular form due to potassium chromate, anuria or diminution of the amount of urine is parallel and due to the amount of epithelial involvement. While this confusion exists it is evident that attempts to separate a tubular from a vascular type of nephritis in man based on the rate of excretion of the different constituents of the urine are of but small value.

More satisfactory are the attempts that have been made to determine the functional efficiency of the kidney as a whole and not of its vascular or tubular mechanisms as separate entities. The two most important methods of achieving this end are the determination of the amount of urea in the blood, and the injection of phenol-sulpho-nephthalein, and the observation of its rate of excretion, as advocated by Rowntree and Geraghty. The evidence of their value is mostly clinical, but they have been the subject of a certain amount of experimental work. The rate of excretion of phenol-sulpho-nephthalein and the amount of non-proteid nitrogen and urea in the blood vary from normal in the course of an attack of acute experimental nephritis in animals just as they do in the spontaneous disease in man, and return to the normal after the attack is over. As might be expected, the alteration in the former case is more rapid than in the latter.[30] Phenol-sulpho-nephthalein tests also point to the conclusion that the functional capacity of the kidney cannot be estimated by histological examination after death.[19]

Experimental nephritis has thrown but little light on the great problem of uræmia. The only work that has a direct bearing on the subject is that of Theohari and Giurea.[98] These observers found that in dogs with uranium nephritis it was possible to produce certain of the symptoms of uræmia by feeding the animals on a diet of raw or cooked meat. These symptoms were

chiefly vomiting, dyspnœa, and coma, and were not observed if the animals were limited to a diet of vegetables or deprived of food altogether. Their observations so far as I know stand alone, though others have frequently noted in animals with experimental nephritis such symptoms as vomiting which may possibly be of uræmic origin.

Nephritis artificially produced tends to get well spontaneously, which is, perhaps, the reason why it has been made but little use of for the purpose of investigating points of treatment. A few workers have, however, turned their attention to this aspect of the question. Hall and Herxheimer[36] have investigated the results in animals suffering from chronic interstitial nephritis produced by the injection of ammonium chromate of the operation of decapsulation advocated by Edebohls for that condition in man. They found that there was no advantage to be gained by the proceeding and that the re-vascularisation of the kidney, which it is claimed by the advocates of the operation takes place, did not occur. The new capsule that was formed contained no excess of vessels, and was merely a mass of connective tissue rather thicker and containing fewer vessels than did the capsule that had been removed. The influence of diuretics from the therapeutic point of view, the use of which in acute nephritis has often been deprecated on theoretical grounds, has been investigated by Christian and O'Hare.[20] They found that in severe fatal uranium nephritis, diuretin shortened the lives of the animals, while in less severe cases there was some ground for supposing that the action of the drug was beneficial.

So far, then, the study of nephritis by the experimental method has definitely solved none of the problems of the disease. This, however, is by no means to say that the results achieved are of no value. Many suggestive discoveries have been made, and much light has been thrown on the dark places of renal pathology, and there is every reason to hope that in the future animal experiment will furnish the answer to questions suggested by the disease as seen in man, an answer which could be reached by no other method.

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THE X-RAY APPEARANCES OF SOME BONY INJURIES CAUSED BY MODERN PROJECTILES.

By

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The injuries of bone seen in connection with the war vary markedly in degree of severity; on the one hand we see simple fracture while, on the other, practically total disintegration of bone may result. To group those injuries which have been caused by modern projectiles, and the appearances of which are so kaleidoscopic, we might adopt the following headings:—

1. Simple Fracture.*
2. Perforating Injuries.
3. Comminuted Fractures.
4. Septic Injuries.

Foreign bodies may be present in any of these types, though not necessarily constituting a complication, and these from an X-ray standpoint are practically all bullets or fragments of projectile. Sometimes, of course, other foreign bodies are carried into the tissues by the projectile, the more common of such being coins and buttons. Injuries caused by fragments of surrounding objects are said to be very common in the navy, and the wounds so produced are more lacerated. Of other X-ray

*The term Simple Fracture I here use in contradistinction to Comminuted, and not in its usual surgical sense.

opaque "foreign bodies," I should like to mention iodoform, which is sometimes introduced into septic wounds, and I have even known a mass of it cut down upon before its nature was revealed.

1. Simple Fractures, that is, those in which there is no comminution, are usually produced by spent bullets of any variety or by fragments of shell. It seems that such projectiles have not sufficient force to cut their way through bone, and are consequently deflected, producing a fracture commonly transverse in direction, as shown in Fig. 1, which illustrates such a fracture of the fourth metatarsal in a man who had sustained a superficial wound on the dorsum of the foot as a result of being struck by a spent rifle bullet.

2. Perforating injuries are usually seen in the more cancellous parts of the bones, and the projectile may pass straight through, leaving no indication of its passage except its track, and not infrequently a few small splintered fragments of bone at its point of exit therefrom. Small fragments may have become detached from the projectile during its passage through the bone and remain in the neighbourhood, while sometimes the bullet will lodge in the substance of the bone or may be found in the soft tissues on the other side. Fig. 2 shows where a rifle bullet has passed through the tibia from within outwards The postero-anterior view shows a few fragments of bone at the point where it emerged, while in the lateral view one seems to be looking straight through the hole from which fine fissures may be seen radiating up and down the shaft although there is no interruption in the continuity of the tibia.

The reason why some bones are shattered and others perforated seems to depend largely on the density of the bone struck and also on the angle at which it is struck. Especially is this the case when we are dealing with the modern Mauser bullet, which has most of its weight at the back, and consequently when it hits a bone at an angle it is liable to turn or attempt to turn base forwards much more readily than the old bullet, which was more cylindrical and not so conical in shape.

I am of the opinion that this explains some of the badly shattered bones one sees as the result of injury by rifle bullet.

Those cases in which the bullet has just grazed a cancellous bone are very similar to the perforating injuries. Figs. 3 and 4 show two examples of these "gutter" fractures; in Fig. 4 the wound was septic and a drainage tube is shown in position; the internal malleolus is also detached.

3. The Comminuted Fractures constitute the great bulk of the injuries seen, and the X-ray appearances in these are very varied. When a rifle bullet strikes a dense bone, if it is travelling at anything like its muzzle velocity, it almost invariably causes comminution, and this whether the bone has been squarely hit or only just grazed. Fig. 5 shows the result of a bullet striking the posterior border of the ulna, the position in which it grazed the bone being seen in the lateral view. The comminuted fragments are of some size. Fig. 6 presents a very different appearance; here both bones were struck fair and square, the comminution being considerably finer, while there are also many small fragments of the bullet in the neighbourhood. The bullet—a Mauser—first struck the radius and then the ulna, the fragments which were left behind being all near the wound of entrance, as shown in the antero-posterior view. As a rule, when there are small fragments of a bullet left in the wound, they are found near the wound of entrance, as in this case.

Fig. 7 shows a fracture of the ascending ramus of the lower jaw in a man who, while being removed from the trenches to the hospital, vomited the bullet up with a lot of blood. The injury here must have been caused by a bullet which had lost a great part of its momentum, but it is curious that it should have had just sufficient energy to penetrate the mucous membrane on the one side of the mouth and not enough to seriously injure that on the other.

4. Septic injuries from an X-ray point of view present changes the chief of which is the loss of lime salts, and the rate at which these are absorbed seems to depend on the virulence of the septic

process. Fig. 8 is an illustrative case; here there has occurred a very severe injury of the head and upper end of the shaft of the humerus. The wound was discharging freely, being very septic, and this accounts for the translucent appearance of the bones. This injury was caused by shrapnel, a fragment of which is seen behind the fractured clavicle; other minute pieces are to be seen elsewhere.

Fig. 9 is an interesting form of comminution where all the fragments are very small and practically spherical. This man, after receiving his wound, remained in France for some weeks, the spherical appearance of the fragments being apparently due to the absorption of their edges in a wound which was not aseptic.

Fragments of bone may sometimes be carried completely out of the wound, in which case the wound of exit is liable to be large and lacerated. Fig. 10 shows where some fragments of the fibula have been shot away and dispersal of others has occurred. The man is of the opinion that he was hit by a dum-dum bullet, but of this there is no reliable evidence.

Some of the results of these injuries when not interfered with surgically are shown in Figs. 11 and 12; in the former, union has occurred with the inclusion of a small fragment of metal, callus is not yet fully ossified, and there is a sinus leading down to the bone which continues to discharge. In the latter, firm bony union has occurred in the fibula, while there is a fragment of shrapnel in the soft parts which, though not æsthetic from an X-ray point of view, probably gives rise to no symptoms.

In Fig. 13 two views of a case of fracture of the humerus are shown in which union has occurred in unsatisfactory position, and there is also to be seen a fragment of dead bone which prevents the wound from completely healing. The translucent area surrounding the dead fragment is well shown in the postero-anterior view.

The X-ray Appearances of some Bony Injuries caused by Modern Projectiles.

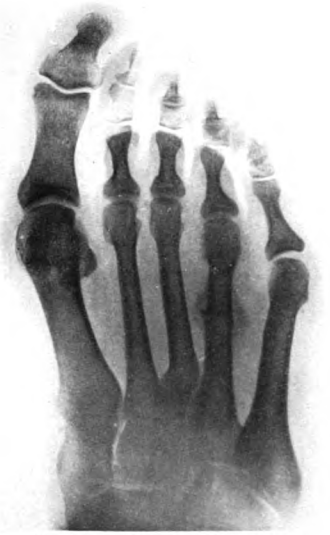


FIG. 1.—Plantar-dorsal View.

Lateral View.

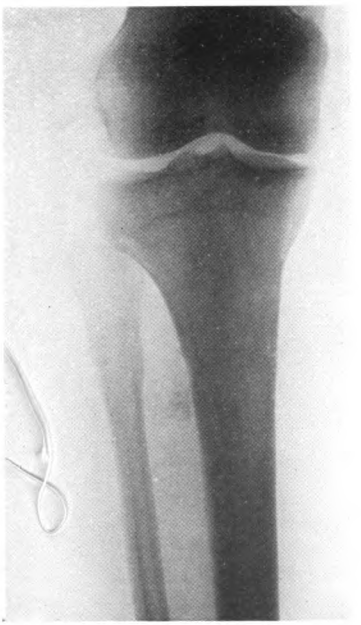


FIG. 2.—Postero-anterior View.

Lateral View.

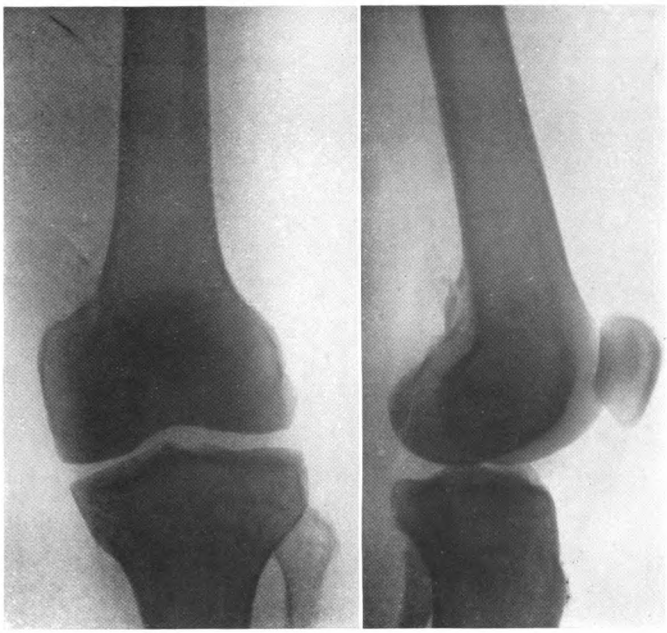


FIG. 3.—Postero-anterior View. Lateral View.

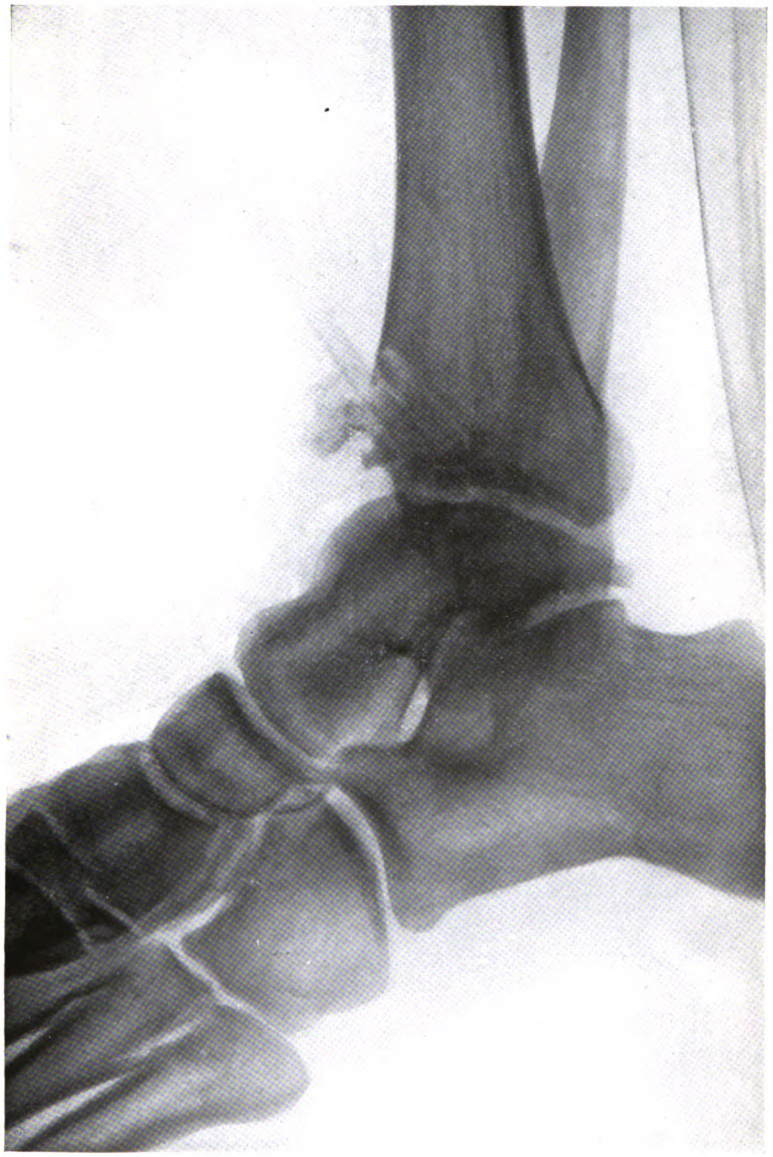


FIG. 4.—Lateral View.

The X-ray Appearances of some Bony Injuries caused by Modern Projectiles.

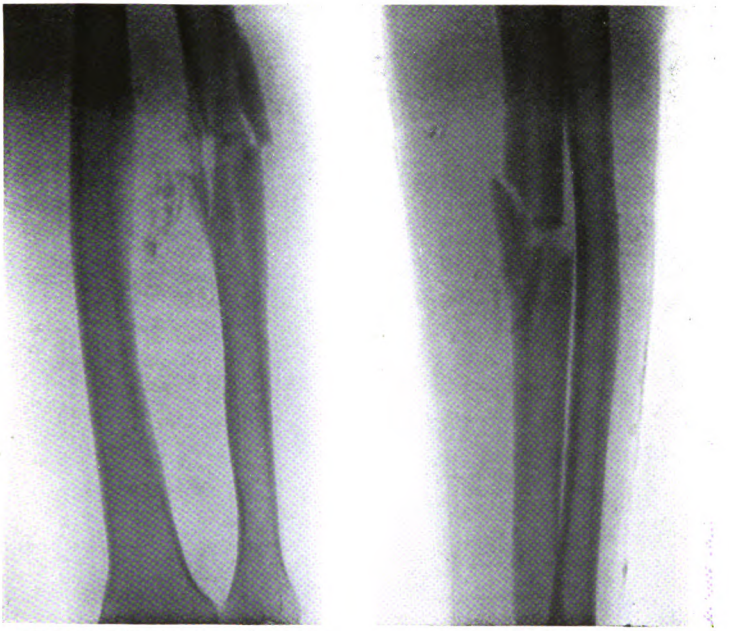


FIG. 5.—Antero-posterior View. Lateral View.

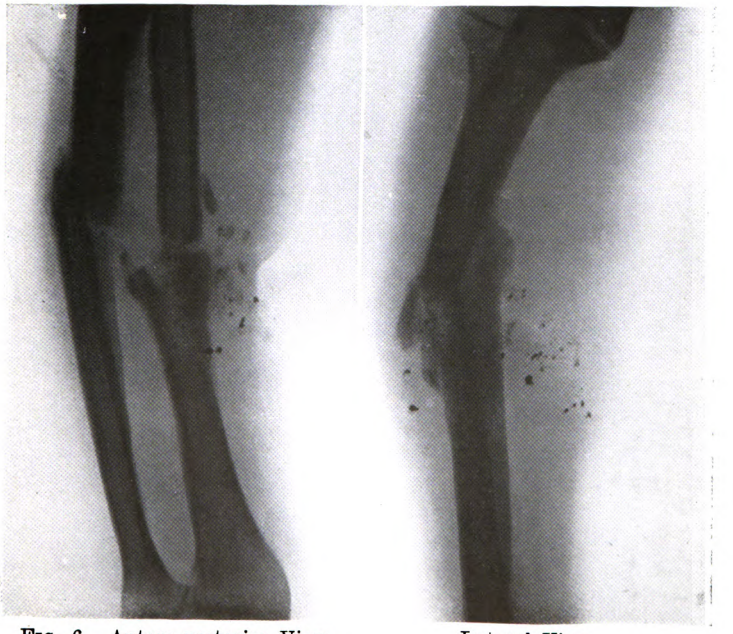


FIG. 6.—Antero-posterior View. Lateral View.

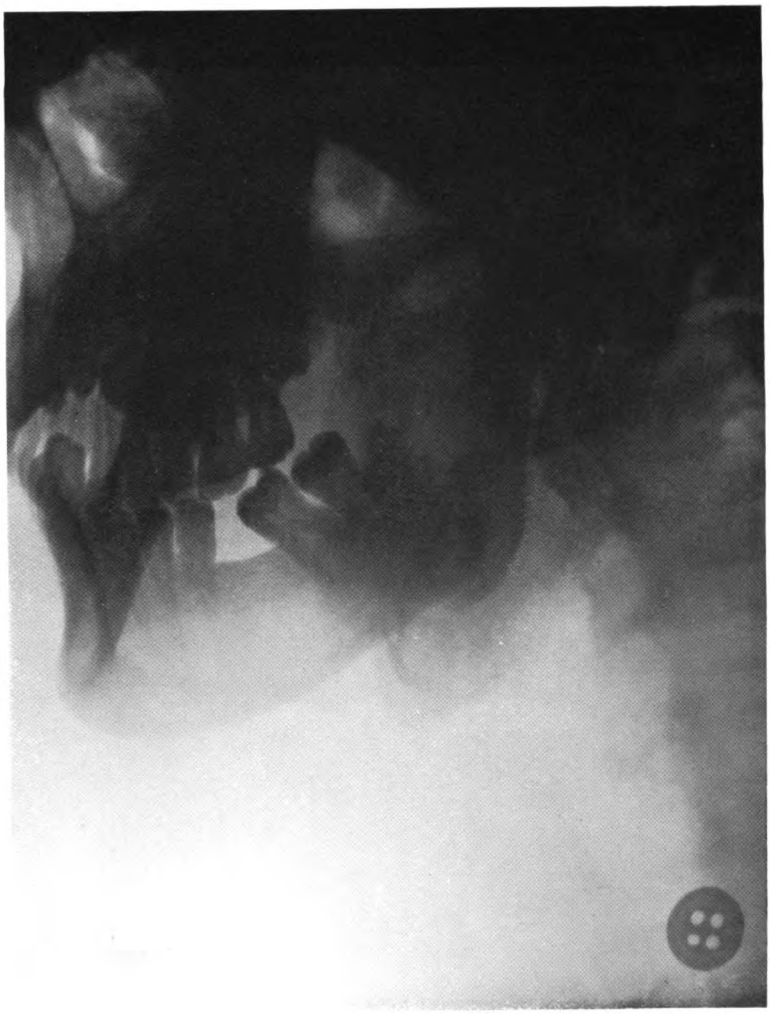


FIG. 7.

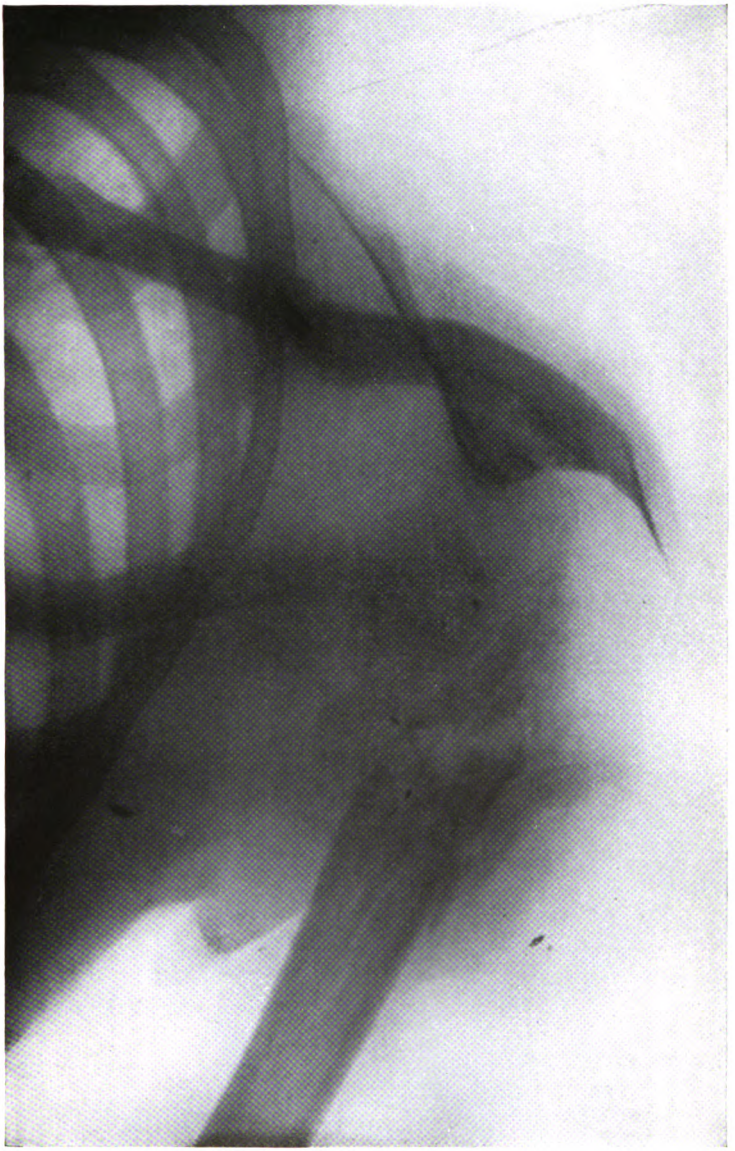


FIG. 8.

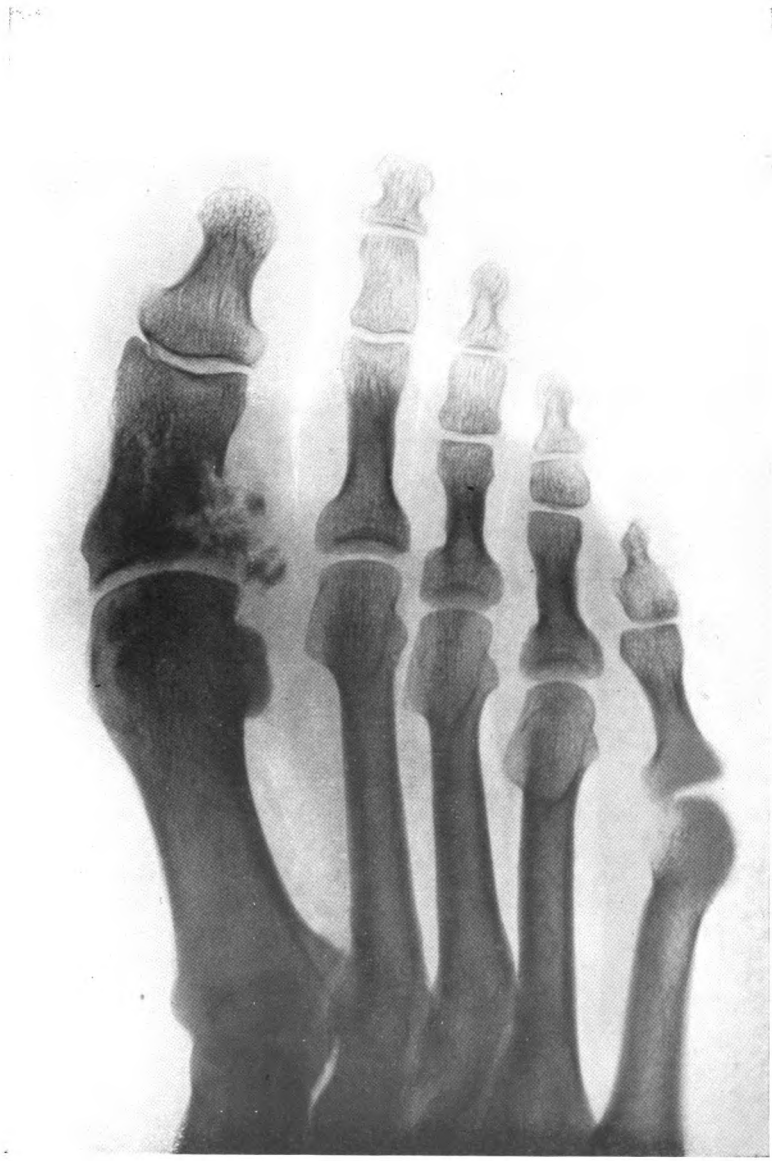
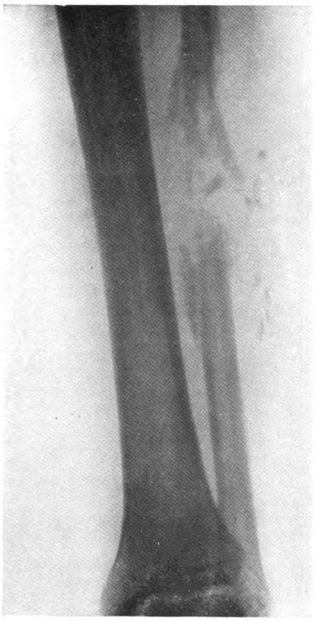


FIG. 9.



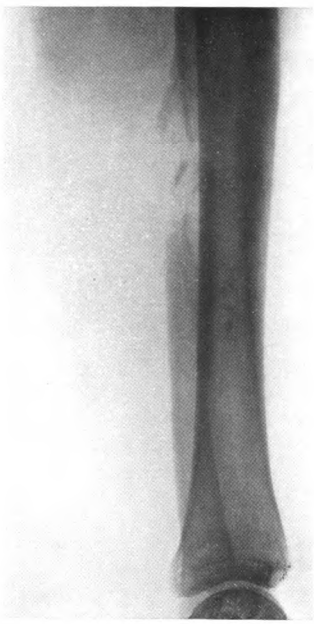


FIG. 10.—Postero-anterior View. Lateral View.

The X-ray Appearances of some Bony Injuries caused by Modern Projectiles.

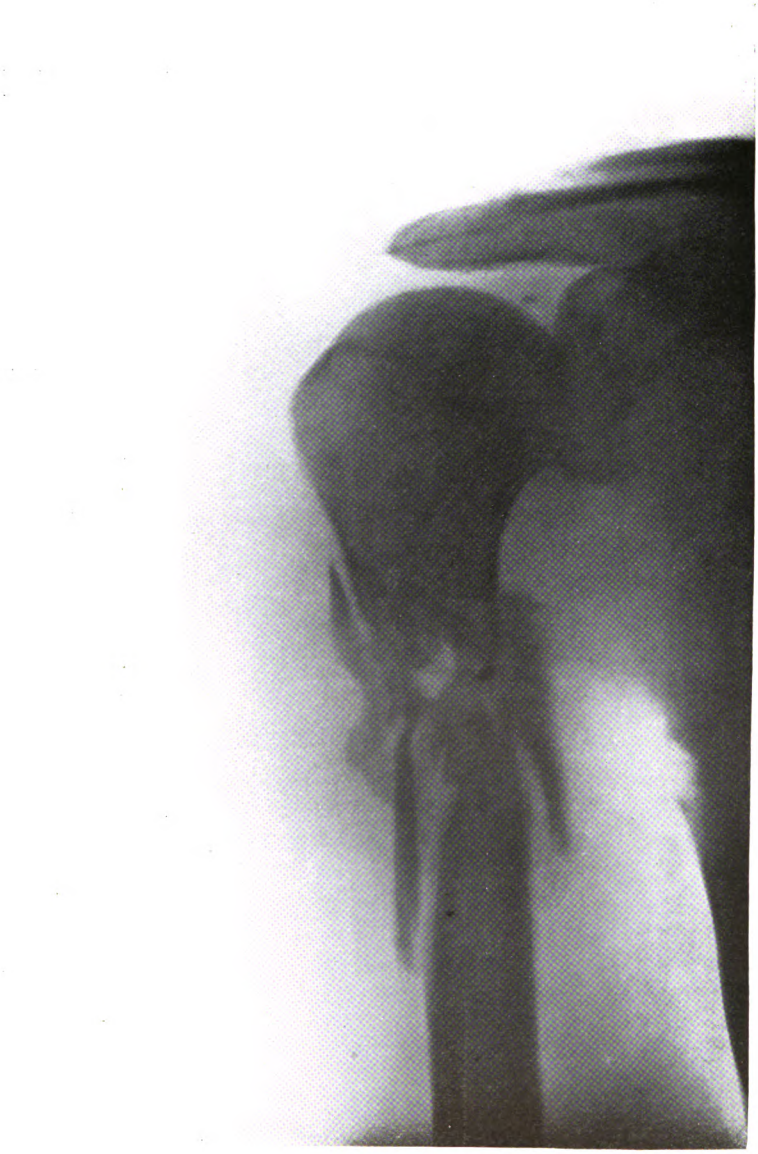


FIG. 11.

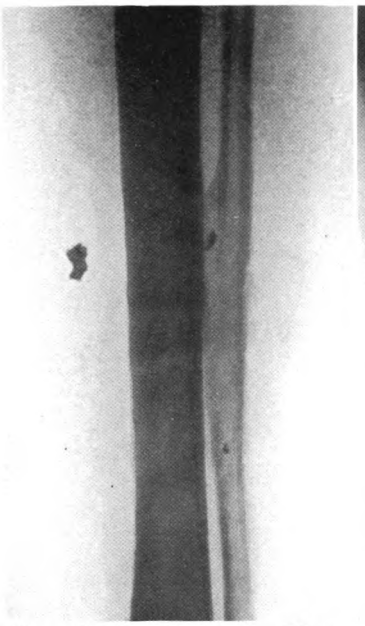
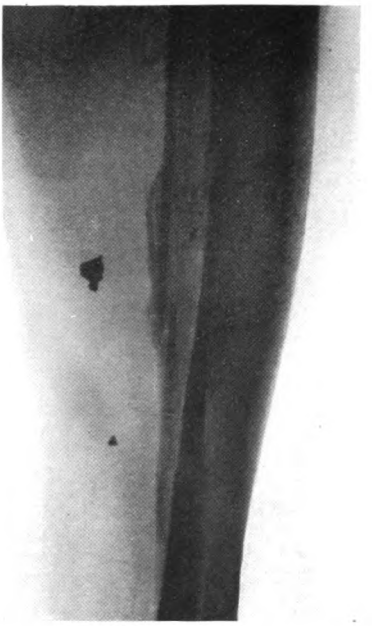


FIG. 12.—Postero-anterior View.



Lateral View.

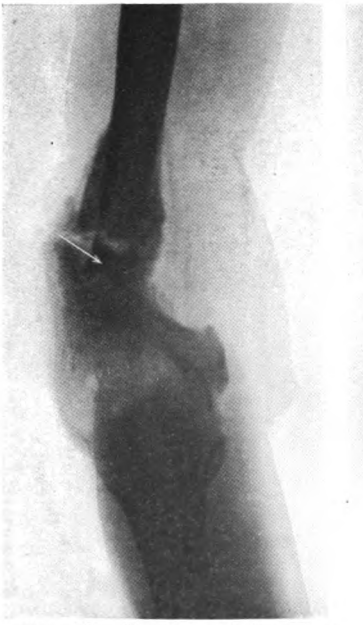
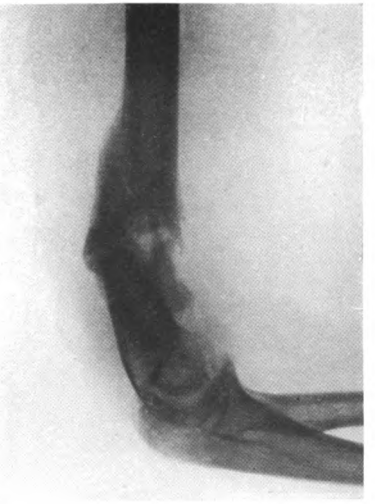


FIG. 13.—Postero-anterior View.
The arrow points to the dead fragment.



Lateral View.

THE NEW LABORATORIES OF THE BACTERIOLOGICAL DEPARTMENT AT GUY'S HOSPITAL.

By

JOHN W. H. EYRE.

A short account of the institution and development of the Bacteriological Department, its personnel, and the scope of its work, may be regarded as forming a suitable introduction to a description of the new buildings which the Governors of Guy's Hospital, with characteristic generosity, have provided to meet the requirements of this youthful but robust offspring of scientific medicine.

Historical.

At a meeting of the School Staff in November, 1888, it was resolved that: "It is desirable to provide instruction in Bacteriology, and that a Committee be appointed to consider the best means of carrying this resolution into effect." This Committee reported in January, 1889, and recommended that the new Pathological Room (the room adjoining the post-mortem room, now part of the Pathological Chemist's quarters) should be used as the site of the proposed laboratory; that the cost of the requisite apparatus and fittings should not exceed £30; that the working expenses of the Department for the first year should not exceed £5; and finally, that a Demonstrator of Bacteriology should be appointed without salary.

The Committee's recommendations were at once adopted, and Dr. Washbourn, at that time a Demonstrator in the Dissecting Room, was appointed Demonstrator of Bacteriology (H. A.

Hunt, the Post-mortem Room Attendant, now Museum Attendant, acting as his Laboratory Assistant); and the subject of Bacteriology was first specifically mentioned in the "Green Book" of the Medical School for 1889–90.

With the re-building of the Petersham Block (1890—92), provision was made for separate laboratories for Bacteriology, one over the Bakehouse and the other in the renovated Practical Chemistry Laboratory on the ground floor; and in 1893 the Bacteriological Department was instituted as a definite unit of Guy's Hospital and Medical School for teaching, diagnosis, and research.

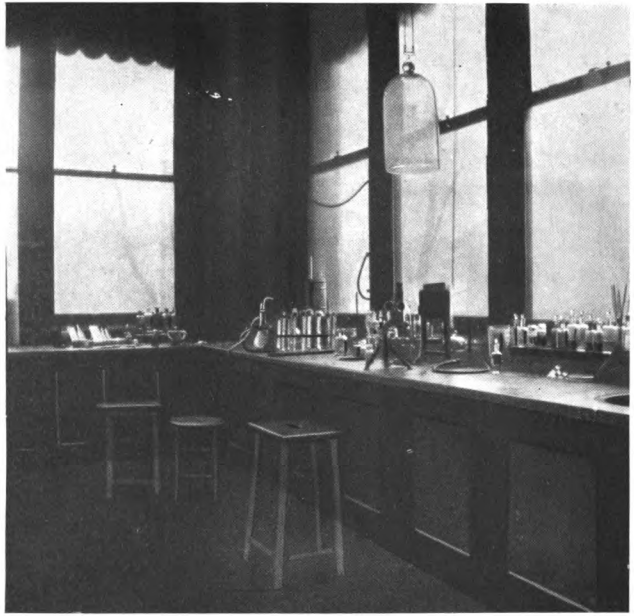
Its Staff then comprised Dr. Washbourn, as Lecturer and Demonstrator, Mr. W. C. C. Pakes, as Student Demonstrator, and a laboratory boy.

The new department so rapidly justified its existence that in July, 1894, the Medical School appointed a Committee to consider the best means of obtaining further assistance in the Bacteriological Department. This Committee reported at the end of the month, advising the appointment of an Assistant Demonstrator, and in the following October, Mr. W. C. C. Pakes was appointed the first Assistant Demonstrator of Bacteriology "in consideration of the increasing recognition of the value of bacteriological methods in the diagnosis of disease."

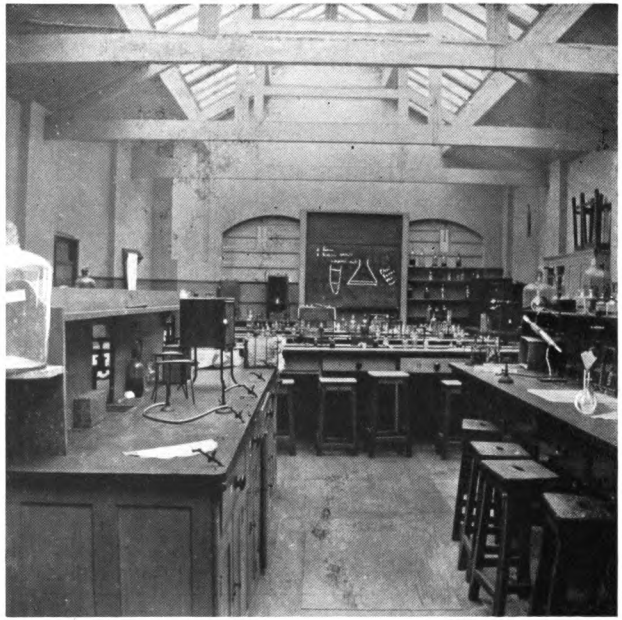
In 1896 Dr. Washbourn was appointed Lecturer on Bacteriology in the Medical School, and Mr. Pakes became Full Demonstrator, whilst the laboratory staff was increased by the addition of a second laboratory boy and a female "cleaner."

In 1900 the Demonstrator of Bacteriology received the additional title of Bacteriologist to the Hospital.

Mr. Pakes, having been appointed Government Analyst and Bacteriologist to the Transvaal Colony, vacated his post at Christmas, 1901. Shortly after this, Dr. Washbourn was incapacitated by illness, and Dr. R. Beaton was placed in charge of the Department, until April, 1902, when Dr. Eyre, at that time Bacteriologist and Lecturer at Charing Cross Hospital, was appointed Bacteriologist to the Hospital and Demonstrator of



A Corner of the original "DIAGNOSIS AND RESEARCH" LABORATORY of the Bacteriological Department, 1893.



The original "TEACHING" LABORATORY of the Bacteriological Department, 1893.

Bacteriology in the Medical School. Dr. Washbourn, as Head of the Department, retained the post of Lecturer until the fatal termination of his long illness in June, 1902, when Dr. Eyre was appointed as his successor, and also to the newly-created post of Lecturer on Dental Bacteriology; in 1907 Dr. Eyre received the additional title of Director of the Bacteriological Department.

The Department and the Medical School.

In consequence of the rapid growth of the work of the Department, additions to the staff of workers became necessary from time to time; thus, in 1904, an Assistant Bacteriologist was first appointed. This appointment carries with it permission for the holder to apply to the Home Office for a licence for inoculation experiments under the Vivisection Act, to be held during the continuance of the appointment, the details of the application being subject to the approval of the Head of the Bacteriological Department for the time being. In 1905 a third laboratory assistant was introduced, and, at the same time, a woman, working from 6.30 a.m. to 1 p.m. each day, replaced the "cleaner."

In June, 1907, a lady typist was installed in the Department. In 1909 a second or Junior Assistant Bacteriologist was added to the staff. This post does not carry with it liberty to apply for a vivisection licence, but it is left to the discretion of the Head of the Department to make the necessary application for any individual holder of the appointment, should it seem expedient to do so.

Since 1907 the Assistant Bacteriologist has been required to devote his whole time to the duties of his post, but the Junior Assistant Bacteriologist, who is a "part time" worker only, is still permitted, subject to the approval of the Treasurer and the Head of the Department, to hold other appointments in addition.

The post of Assistant Bacteriologist is regarded chiefly as one of educational value to the occupant, the experience thus gained enabling him to obtain a more remunerative appointment in

some other hospital. That this is so is apparent from the records of past Assistant Bacteriologists, of whom the Department has had thirteen in the past eleven years.

CHRONOLOGICAL LIST OF ASSISTANT BACTERIOLOGISTS.

1902. Beaton, R. (temporary), January—April, 1902.

1903. Candler, J. (temporary), August—October, 1903. Pathologist to the West London Hospital; Assistant to the Pathologist to the London County Council; Medical Inspector to the Local Government Board.

1904. Price-Jones, C., January, 1904—January, 1906. Assistant, Cancer Research Department, Middlesex Hospital; Lieutenant, R.A.M.C.

1906. Louisson, M. G., January, 1906—December, 1906. Bacteriologist and Pathologist, Christchurch Hospital, New Zealand.

1907. Wedd, B. H., January, 1907—September, 1907. Bacteriologist to Moorfields; Assistant, Cancer Department, Middlesex Hospital; Bacteriologist to the Tropical Diseases Hospital, Albert Docks; Lieutenant, R.A.M.C.

1908. Leeming, A. N., January, 1908—September, 1908. Private Practice; Lieutenant, R.A.M.C.

1908. Minett, E. P., October, 1908—September, 1909. Bacteriologist to Moorfields; Assistant Government Bacteriologist; Government M.O.H. and Port Health Officer, British Guiana; Captain, R.A.M.C.(T.)

1909. MacAlister, G. H. K., October, 1909—December, 1909. Assistant Bacteriologist, Lister Institute; Assistant Bacteriologist, Welcome Research Laboratory; Pathologist, Imperial Laboratory, Muktesar, India.

1909. Catto, H.; (Junior) October, 1909—December, 1909; (Senior) January, 1910 — March, 1911. Laboratory Director, Clinical Research Association; Assistant Government Bacteriologist, Jamaica.

1910. Browning, S. H.; (Junior) January, 1910—November, 1912. Bacteriologist to Moorfields; and Leathersellers' Technical College; Lieutenant, R.A.M.C.

1911. Goodhart, G. W., November, 1911—March, 1913. Gordon Demonstrator of Pathology, Guy's Hospital Medical School; Captain, R.A.M.C.(T.)

1912. Passey, R. D.; (Junior) November, 1912—March, 1913. Bacteriologist, Nordrach-on-Dee Sanatorium; Lieut., R.A.M.C.

1913. Lucey, H. C., March, 1913—September, 1914. Lieutenant, R.A.M.C.

1913. Keith, S.; (Junior) March, 1913—September, 1914. Captain, 4th Queen's West Surrey (T.)

1915. Sampson, B.; (Junior) February, 1915—April, 1915. Surgeon, R.N.

1915. Munro, A. Watson; (Honorary Temporary Assistant) May, 1915–

As already mentioned, the Department was instituted primarily as a unit of the Guy's Medical School, and during the first years of its existence research was its chief function, and volumes of collected papers were issued from the laboratory in 1897, 1902, and 1907 respectively, containing the published contributions of a number of research workers. Teaching was at first limited to one short course of lectures each winter, given in the Physics Theatre (now part of the enlarged Dental Conservation Room), and to practical instruction to a limited number of enthusiasts.

In 1894 practical instruction was instituted for the benefit of candidates for Diplomas in Public Health (in addition to two annual courses of lectures in General Bacteriology), and from this time, until about 1901, a fairly large number of students took out courses of instruction in Public Health Laboratory work. In 1902 the tuition of post-graduate students for Public Health Diplomas was relinquished and not resumed again until 1905—6.

In 1896 a course in Microscopical Pathology, including both Bacteriology and Morbid Histology, became compulsory upon candidates for Medical Degrees and Diplomas, and classes were instituted in these subjects under the joint direction of Messrs. Bellingham Smith and Pakes, and by 1902 the average number of students attending the class was between 70 and 80. With the declining entries, resulting from the introduction of the five years' curriculum, these numbers fell gradually, and since 1909 the average attendance any one year has been about 53. In 1910

it was considered expedient to devote an equal time (three months) to each of the two branches of Microscopical Pathology, viz., Bacteriology and Morbid Histology, and to alternate these two subjects throughout the entire year, so that two complete courses of Microscopical Pathology are now given in each year.

In 1912, on the completion of the New Pathological Building of the Medical School, all the purely teaching work of the Bacteriological Department was accommodated in that building.

The Department and the Hospital.

From the moment Bacteriology took a place in the curriculum at Guy's it has played a part in the clinical work of the hospital, and the statistics of the Department form an instructive commentary on the value attached by the clinician to the results derived from the bacteriological investigation of pathological material. Before the institution of a special department, the aid of the Demonstrator of Bacteriology was invoked in doubtful or obscure cases, as evidenced by such papers as "Two Doubtful Cases of Tuberculosis," "A Case of Glanders, with the results of cultivation and inoculation experiments," which appeared in the "Guy's Hospital Reports" in 1890, but no Laboratory record of such investigations exists prior to 1893. In that year, however, a manuscript note-book, the earlier part of which is in Dr. Washbourn's handwriting, was set aside for notes upon material submitted for examination from the wards, and this sufficed for a time, for only eight specimens were examined during that year. With the expansion of the teaching in bacteriology and the housing of the Department in its own Laboratories, more thorough organisation of the clinical diagnostic work became necessary, and requisition forms on the lines of those already in use for Museum specimens were issued to the wards. These Report Forms bore, on the one side, printed headings for identification notes and final report, and, on the other, concise directions for the collection of material for examination. The forms have been revised from time to time and their present appearance may be gathered from the accompanying example.

READ DIRECTIONS ON BACK.

O.H.—161

SPECIMEN FOR BACTERIOLOGICAL EXAMINATION.

[EVERY SPECIMEN MUST BE ACCOMPANIED BY A REPORT FORM, PROPERLY FILLED IN.]

Physician or Surgeon ..... *Ward & Bed or No. of Inspection* .....

Name of Patient ..... *Disease* .....

Material? *Source?* .....

Investigation required or organism suspected? .....

Date ..... *Signed* .....

This Form must be duly filled up, and signed by a member of the Staff, the Resident Surgical Officer, one of the Registrars, a House-Physician, House-Surgeon, Out-Patient Officer, Assistant House-Surgeon, Obstetric Resident, or Assistant in the Clinical Wards.

When investigations involving inoculations are required the Form must be signed by a member of the Staff.

REPORT.

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Date ..... *Signed* .....

[See overleaf

INSTRUCTIONS.

1. Directions for collecting material:—

(*a*) Pus and other fluids should be collected in sterile tubes in the following manner:—The cotton wool and lip of the tube should be flamed over a spirit lamp. The cotton wool should then be removed, and a small quantity of fluid collected, the lip of the tube again flamed, and the plug replaced. The plug should be held in the hand during the proceeding.

(*b*) If the quantity of fluid is too small to be collected in the above manner, it should be soaked up with a sterile swab, which should then be replaced in the tube.

(*c*) For cases of suspected **Diphtheria,** *and for these cases only*, boxes containing blood serum tubes and sterile swabs are provided. The technique of making cultivations is as follows:—

Withdraw the swab from the tube and apply it to the affected part so as to remove a little of the exudation. Take care that the swab does not touch the tongue or cheek during the procedure.

Smear the surface of the blood serum lightly with the swab, and replace the cotton wool plug.

Put the swab back in the tube from which it was taken, and re-plug the tube. Take care not to lay down the swab or cotton wool plugs either before or after making the cultivation.

Do not make the cultivation immediately after the throat has been treated with an antiseptic.

2. Sterile tubes, tubes containing sterile swabs, sterile pipettes, boxes containing blood serum tubes, and these forms are kept by—

(*a*). Sisters Clinical, Addison, and Stephen.

(*b*). The Sisters in charge of the Out-Patient Department and the Surgery.

Tubes for the collection of specimens of fæces, sterile flasks for catheter specimens of urine, and other special apparatus may be obtained from the Bacteriological Laboratory.

3. The Bacteriological Laboratory is closed between the hours of 5 p.m. and 9 a.m. During this time tubes inoculated with (?) diphtheritic material should be placed in the incubator in the Surgery; **tubes containing pus or other material should be placed in the cupboard in the Surgery provided for that purpose,** *—and not in the incubator.*

GUY'S HOSPITAL.

Issued December, 1896; *revised January*, 1898; *November*, 1902; *June*, 1904.

Special "Report" books, with printed headings on the pages to correspond with those on the Report Forms, were provided for the reception of the Laboratory notes on the various specimens examined.

During the years 1893—4—5 specimens to the number of 150 were examined for the hospital, but in the immediately following years the marked increase which took place in the numbers of the specimens submitted for examination necessitated a fresh Report Volume of increased bulk each year, until, growing unwieldy in size, the yearly volume in 1904 gave place to a card index system of notes—a plan which still obtains—the main facts relating to each examination being transferred to loose sheets which at the end of the year, together with a typescript index, are bound to form a not unhandy reference volume.

In order to facilitate the collection of the pathological material by clerks and dressers, sterilised test-tubes and sterile swabs were placed in the principal wards from the time the Report Forms were issued. Diphtheria outfits, each consisting of a mounted cotton-wool swab and a tube of suitable culture medium enclosed together in a metal case were also provided, and other pieces of apparatus, such as pipettes for the collection of blood, tubes for fæces, sputum, etc., have been added from time to time. Such apparatus was originally served out from the Laboratory itself, but of recent years the work of distribution has been taken over by the Stores' Department of the Hospital, and the following table shows the nett cost of this service for the past five years :—

Year	Serum Tubes.	Sterile Swabs.	Sterile Tubes.	Widal Pipettes.	Fæces Tubes.	Urine Bottles.	Sputum Pots.	Total Cost. £ s. d.
1910	2052	3060	600	192	—	—	—	49 4 6
1911	2448	3408	660	180	—	—	—	57 7 9
1912	2238	3206	856	144	36	8	107	55 14 6
1913	1692	2724	972	96	108	492	648	52 17 11
1914	1362	2352	900	168	360	504	576	42 17 6

During the period 1902—14 the numbers of specimens examined by the Department on behalf of the hospital increased

rapidly, and during the first eight years of that period—until, in fact, stress of routine work and lack of space rendered it impossible—research upon the clinical applications of bacteriology was vigorously prosecuted. Some idea of the volume of work presented to the Department may be gained from an inspection of the three following tables :—

TABLE I.

TABLE showing the total number of Specimens examined in the Bacteriological Department during each month of the twenty-two years, 1893—1914 inclusive :—

	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1893	—	—	—	—	—	3	3	2	—	—	—	—	8
1894	—	1	—	3	2	1	2	—	—	6	16	8	39
1895	6	4	8	—	22	12	2	6	3	23	14	5	105
1896	24	29	29	19	14	20	46	57	55	67	52	61	473
1897	53	60	63	38	39	48	40	72	67	105	65	58	708
1898	71	81	59	62	60	86	56	58	56	77	83	79	828
1899	86	87	54	67	62	66	90	52	86	84	64	50	848
1900	73	65	79	54	57	58	54	67	75	77	71	53	783
1901	65	68	63	65	83	121	91	117	97	97	90	69	1026
1902	77	71	66	116	126	130	106	125	94	132	125	96	1264
1903	78	88	103	105	96	79	101	73	83	100	92	67	1065
1904	105	92	78	76	50	75	102	126	96	95	108	80	1083
1905	117	94	138	89	124	182	144	182	155	175	271	260	1931
1906	349	300	351	269	359	357	387	429	329	385	409	225	4149
1907	408	445	520	565	594	581	704	535	605	541	457	346	6301
1908	447	411	412	353	408	442	414	342	462	542	443	439	5115
1909	512	429	501	489	503	645	537	430	538	497	398	351	5830
1910	380	409	345	426	472	496	416	476	437	405	559	505	5326
1911	568	551	595	453	640	514	615	568	525	478	536	434	6477
1912	607	530	611	498	585	527	620	629	577	642	650	543	7019
1913	606	525	467	515	505	532	630	579	581	644	582	515	6681
1914	588	632	685	545	790	650	694	455	472	527	402	399	6839

Strenuous efforts have been made to combine economy with efficiency, and that some success has been attained in the former respect may be gathered from the fact that if the total cost of materials and labour is divided equally amongst the specimens examined in any particular year, an average cost per specimen is obtained which, during the past fourteen years, has never exceeded 2s. 6d., and is usually well below 2s. Indeed, in one year (1905) it was as low as 9d. per specimen.

In appraising the value of these figures it must be recollected that the specimens examined in the Department include only cultivations or inoculation investigations, Wassermann and other serum reactions, the preparation of vaccines and the like. In other words, the Department concerns itself with bacteriological and parasitological investigations only (section cutting and chemical analyses being carried out in other departments of the hospital), and until two or three years ago even the microscopical examination of sputum for tubercle bacilli was not regarded as of sufficient importance to engage the attention of the Bacteriological Department, but was carried out in the clinical laboratory attached to the wards.

TABLE II.

TABLE showing the total number of Specimens examined by the Bacteriological Department, classified according to the Department of the Hospital from which each was derived, for each of the years 1893—1914, inclusive :—

	Medical Wards.	Clinical Wards.	Surgical Wards.	P.M. Room.	Out-Patient and Casualty Department (Front Surgery)	Dressings, etc.	Total.
1893	4	—	4	—	—	—	8
1894	12	7	13	—	7	—	39
1895	19	20	16	19	31	—	105
1896	69	76	54	19	255	—	473
1897	115	77	77	26	413	—	708
1898	131	82	112	32	471	—	828
1899	204	160	122	51	311	—	848
1900	215	123	100	66	279	—	783
1901	251	160	102	43	470	—	1026
1902	222	164	158	82	638	—	1264
1903	186	176	181	118	404	—	1065
1904	174	138	139	67	565	—	1083
1905	265	259	287	110	721	289	1931
1906	309	289	537	163	2114	735	4147
1907	396	314	488	151	4057	895	6301
1908	443	352	700	120	2479	1021	5115
1909	604	401	794	194	2818	1019	5830
1910	579	400	794	131	2423	990	5317
1911	705	470	874	177	3270	984	6480
1912	725	545	815	196	3356	1380	7017
1913	687	609	871	119	3045	1350	6681
1914	969	603	956	129	2736	1446	6839

TABLE III.

TABLE showing the Ratio existing between the number of Specimens examined (see Table II) and the number of In-Patient Admissions, Autopsies, and Out-Patient New Cases respectively for each of the years from 1893—1914 inclusive:—

	Medical and Clinical Wards.	Surgical Wards.	Post-mortem Room.	Out-Patient and Casualty Department (Front Surgery).
1893	1 : 535	1 : 905	—	.
1894	1 : 108	1 : 296	—	—
1895	1 : 64	1 : 259	1 : 27	—
1896	1 : 16	1 : 77	1 : 28	1 : 183
1897	1 : 12	1 : 56	1 : 18	1 : 129
1898	1 : 10	1 : 40	1 : 14	1 : 117
1899	1 : 7	1 : 41	1 : 9	1 : 210
1900	1 : 7	1 : 50	1 : 7	1 : 251
1901	1 : 6	1 : 52	1 : 11	1 : 151
1902	1 : 6	1 : 36	1 : 7	1 : 119
1903	1 : 7	1 : 31	1 : 4	1 : 196
1904	1 : 9	1 : 40	1 : 9	1 : 152
1905	1 : 5	1 : 20	1 : 6	1 : 124
1906	1 : 4	1 : 10	1 : 4	1 : 62
1907	1 : 3·8	1 : 11	1 : 4	1 : 32
1908	1 : 3·2	1 : 8	1 : 5	1 : 51
1909	1 : 2·7	1 : 7	1 : 3	1 : 46
1910	1 : 2·7	1 : 7	1 : 5	1 : 57
1911	1 : 2 5	1 : 7	1 : 4	1 : 41
1912	1 : 2·2	1 : 8	1 : 3	1 : 37
1913	1 : 2·1	1 : 8	1 : 5	1 : 36
1914	1 : 1·2	1 : 7	1 : 5	1 : 42

The Department, however, does not limit its activities to diagnostic work alone: it also takes its place in the administration of bacterial remedies. Experiments which were instituted in 1904 resulted in the following year in the tentative treatment of selected cases of bacterial infection with special vaccines prepared in the Department; and in association with Mr. Sichel, the Surgeon-in-Charge of the Actinotherapeutic Department, lupus and other tuberculous infections were treated with various forms of light and tuberculin (T.R.), this work being carried out either in the wards or in the old Light Department on the top floor of the Surgical Building. Other cases were treated actually in the Bacteriological Laboratories.

After four years' strenuous work this Vaccine Out-Patients' Department was considered to have established its claim to official recognition, and was constituted in February, 1908, as a Specialist Reference Department. It is now accommodated in the Obstetric Room at Out-Patients under the charge of the Bacteriologist, assisted by one chief and two ordinary qualified clinical assistants.

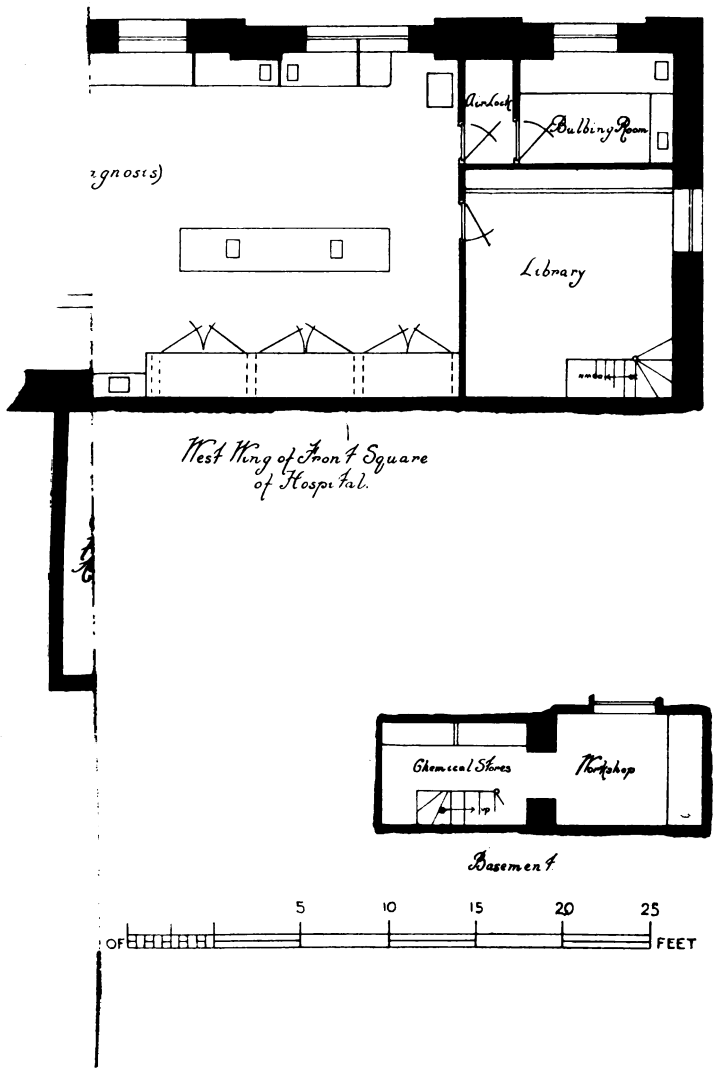
Since 1911 the Assistant Bacteriologist has, as a matter of administrative convenience, been regarded as having a lien upon the post of Chief Clinical Assistant in the Vaccine Department, and the Junior Assistant Bacteriologist upon one of the remaining posts.

Records have been kept of the attendances, etc., from its official institution in 1908, and are summarised in the following table:—

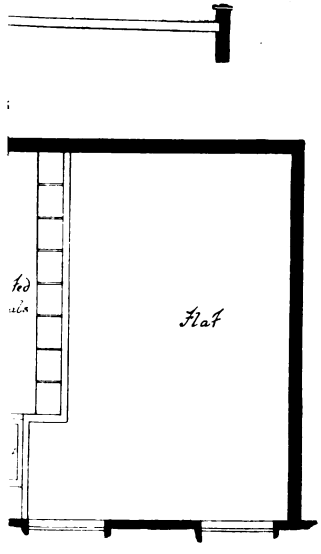
	1908	1909	1910	1911	1912	1913	1914
Out-Patients (New Cases)	200 (10 mths.)	263	234	314	307	424	468
Attendances	1052	1801	2145	2342	2273	2706	2550
In-Patients (in Wards)	164	218	269	243	189	179	158
Autogenous Vaccines prepared	104	211	316	378	408	438	462

The sterilisation of the surgical dressings for the wards and theatres is another matter that comes within the purview of the Bacteriological Department, and a continuous supervision is maintained over the process. Sample packages are included in every batch of dressings sent to the Washington Lyon Apparatus, and these are thoroughly tested in the laboratories, so that in the event of the process breaking down as sometimes happens through mechanical defects in the steam valves, the Sisters concerned are notified and the dressings returned to the steriliser.

The disinfection of mattresses and pillows by a formaldehyde process, which has been in operation for some years past, is also controlled by the Department.


Airlock
Bulbing Room
Library
West Wing of Front Square of Hospital.
Chemical Stores
Workshop
Basement
5
10
15
20
25
FEET

Square of Hospital


Flat

Sets of apparatus supplied to the wards for the intravenous injection of salvarsan are also put up in the Bacteriological Department, which is further responsible for the manufacture of pure distilled water for the necessary saline solutions.

The Bacteriological Department of Guy's, from the time of its inception up to the end of 1913, was administered by the Medical School which, on the one hand, defrayed all costs for materials and wages (with the exception of "cleaning" prior to 1907—8), and, on the other, received all fees for tuition, and, in addition, from 1902 to 1913, an annual subsidy from the Hospital to assist in defraying the expenses incurred in connection with clinical investigations carried out on behalf of patients in the wards, or attending the various Out-Patient Departments, and in the preparation and administration of therapeutic vaccines, etc. The honoraria, however, attaching to the posts of Bacteriologist, and Senior and Junior Assistant Bacteriologist, have always been direct charges upon the Hospital.

At the termination of the 1913 financial year, all Medical School work having been removed from the original Bacteriological Laboratories, this arrangement was completely reversed: the Hospital assumed complete control of the Department and made itself responsible for all running expenses.

New Laboratories.

The laboratories erected in 1893 consisted of a room some 24 feet by 18 feet on the first floor of Petersham House, in which the research work and diagnosis investigations were carried out; and the reconstructed chemical laboratory, 58 feet by 22 feet, which served the varied purposes of media making, sterilising, preparation and class room for teaching purposes. These provided sufficient space for several years, but from about the year 1908 the nature and volume of the work required from the Bacteriological Department rendered the provision of further laboratory accommodation an urgent necessity, which was insisted upon by successive sub-committees. Some relief of the congestion followed the transference of the teaching to the class room

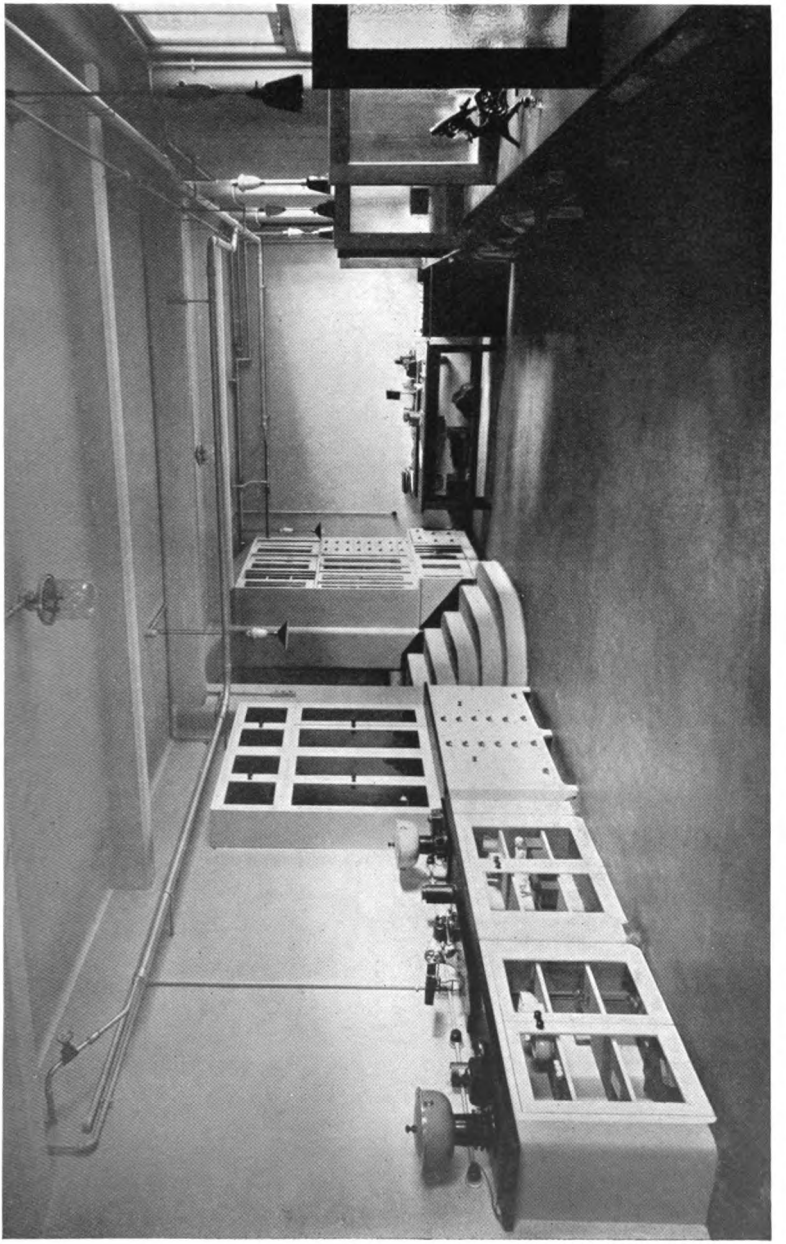
in the first floor of the newly-constructed Pathological Department in October, 1912, it is true, but the improvement was only temporary.

The Governors of Guy's Hospital, whilst fully appreciating the situation, were faced by almost insuperable difficulties in providing an adequate site. Various schemes were put forward from time to time, and in some cases plans were sketched out, but all were rejected in turn, until the moment arrived, towards the end of 1913, when two new floors having been added to the Medical School to provide for the teaching of the early subjects of the curriculum, including Chemistry and Physics, the building behind the Hospital Chapel became vacant, and the Director of the Bacteriological Department was instructed to prepare plans for the adaptation of the site, and in so doing was given an absolutely free hand, subject only to such restrictions as might be imposed by architectural considerations. As a result of this far-sighted policy of the Governors, coupled with the keenly critical supervision of the Superintendent, Guy's now possesses a Hospital Bacteriological Department which is worthy of the premier School of the Metropolis.

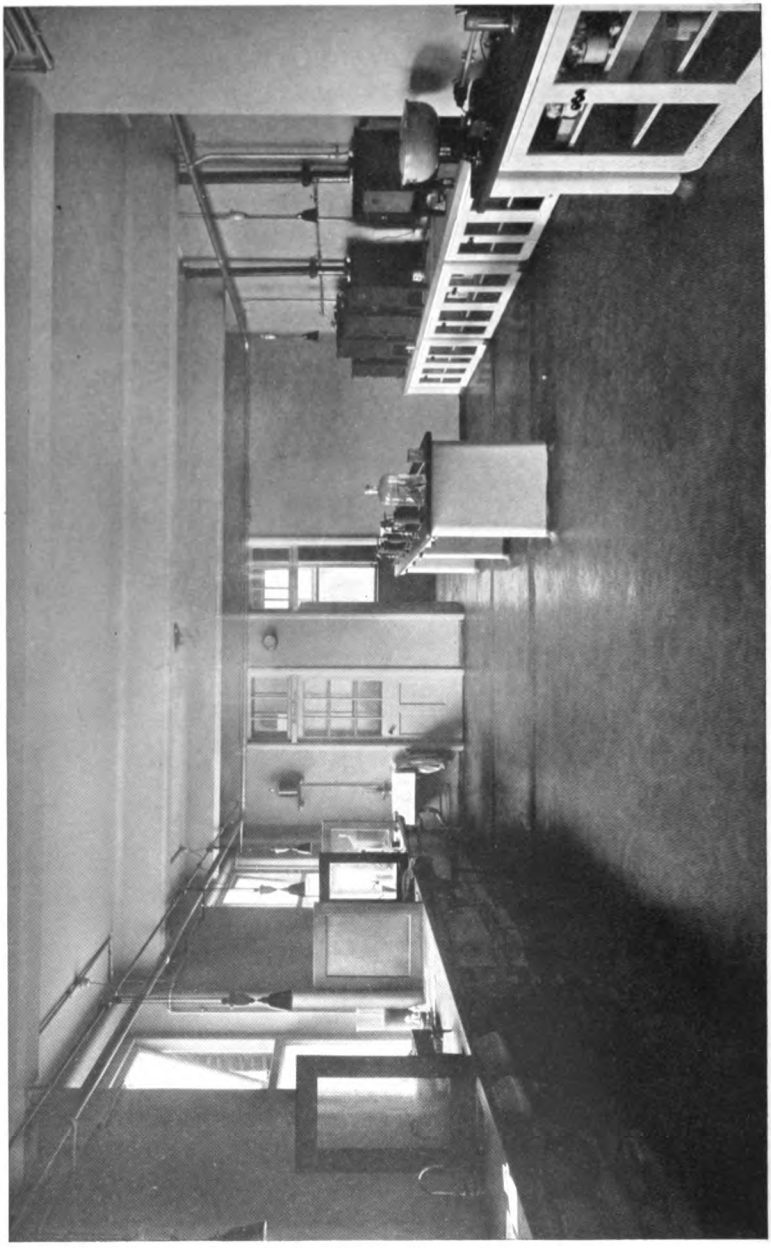
The new Department now occupies the site of the old Chemical Laboratory (afterwards used as a Toxicological Laboratory), the old Materia Medica Room, and the ground floor of the old Chemical Theatre, and is, therefore, situated behind the Hospital Chapel, and between it and the disused graveyard of St. Thomas's Church. The constructional work was actually commenced early in January, 1914, when the premises above mentioned were completely dismantled until only the external walls were left standing.

The original walls thus form the walls of the new Department, although in the case of the old Chemical Laboratory the walls were so riddled with chimneys and flues (for chemical furnaces) that all these various channels had to be filled with concrete before the work could be proceeded with. The new building is arranged on two floors, formed in ferro-concrete, supported on large and small beams (9 inch and 6 inch section respectively)

The New Laboratories of the Bacteriological Department.



Routine Diagnosis Laboratory, looking west, showing on the left steps leading up to the Entrance Corridor.



ROUTINE DIAGNOSIS LABORATORY, looking east. Closed door leads into the BULBING ROOM and open door into the LIBRARY.

of the same material, and a portion of the roof is flat to allow for the housing of animals for experimental purposes.

By the end of January, 1915, the work was if not actually completed, so far advanced that the Department was transferred to its new quarters on February 15th, and a week later was in full working order.

Ground Floor.

Access may be gained to the Department, as to the original Chemical Laboratories, along the narrow passage (by the bake-house) which leads past the Laundry Hostel, but the main entrance is by the door in the Inner Quadrangle, just past the Superintendent's Office, which formerly led to the "Drunk or Dying" Ward. The space available here has been re-arranged as an annexe to the new laboratories, and now provides an entrance lobby where patients sent to the Department await their turn for examination, and two rooms of about equal size, one reserved as a cloak-room for the Female Administrative Staff of the Hospital, the other adapted for the purpose of the Out-Patients' Examination Room. The floor of the last named is asphalted, and the walls are of plaster coated with glossy white enamel paint. In one corner is placed an examination couch, screened off by washable curtains. Hot and cold water are laid on over a large white porcelain sink. Gas and electric light are at hand on the adjacent slate slab, which also accommodates a fair-sized steam steriliser for instruments. A large window in the north wall both ventilates and gives ample daylight, and beneath it is the steam radiator which warms the room in winter. In this room blood for various serological tests is collected or material obtained for the preparation of autogenous vaccines. Thanks to this accommodation, patients no longer enter the Laboratories themselves, where, as often happened in the past, experimental work upon animals was in progress.

Passing through the examination room, the mosaic floored main corridor, running east and west, is reached. This leads to the

laboratories proper. The main laboratory is reached by an archway on the left at the foot of a flight of stairs leading up to the first floor. Another corridor, running off the main corridor to the right opposite the archway, gives access to certain special rooms.

The main laboratory, in which are conducted ordinary routine diagnosis examinations, is on a slightly lower level than the corridor, from which it is reached by a short flight of four steps. It is a room over 50 feet long by 20 feet wide and 9 feet high, well lighted during the day by a row of five windows with north aspect, and at night by three principal ceiling "bucket" lights each containing three 60 c.p. Osram lamps, and twelve single pendant lamps. The walls throughout the Department are plastered, and are at present coated with Duresco water paint, but it is intended in twelve months' time, when thoroughly dry and set, to paint them with some glossy enamel paint like Paripan, which will give a smooth, impervious surface, and which can be easily washed. All corners are rounded in smooth curves to prevent accumulation of dust, and the floor is laid in Decolite —an asbestos wood pulp—with such exceptions as are specially noted.

Immediately within the entrance of the laboratory is placed the desk of the lady secretary, who receives, enters, and numbers the specimens brought for examination, and close to hand are the Post Office, inter-Hospital, and inter-Departmental telephones. A large fitment, comprising glazed cupboards and drawers is erected at this end of the Laboratory for the storage of stationery supplies, the card index, etc.

A long slate working bench extends almost the whole length of the north wall beneath the windows, and is divided into seven unequal portions by glazed screens. The bench is amply supplied with gas, water over small staining sinks, electric light and power plugs, and over each division is a rise and fall pendant light which, running on an overhead rail, has a side to side range of movement of about five feet.. Two of these spaces accommodate the Assistant Bacteriologists, and the remainder pro-

A Corner of the Glass Store Room.

View of the HOT INCUBATOR ROOM (37° C.), looking through the COOL INCUBATOR ROOM (22° C.).

vide definite places for special investigations, such as those involved in the examination of suspected diphtheritic material, complement fixation tests, section work, and the preparation of vaccines, or special pieces of apparatus. On the south wall, just inside the archway, is a large cupboard for media, with drawers below, and beyond that a slate bench for small centrifuges, shaking machines, etc. (driven by electricity), which is provided with half a dozen power plugs, and two gas points for Bunsen burners. Under this bench are glass-fronted cupboards for apparatus.

The space left under the stairs ascending to the first floor has been partly closed in, and the interior walls blackened, to form a dark room for investigations with the dark ground illumination apparatus, or the spectroscope, or the warm stage, and is reached from the interior of the laboratory by a short step ladder. It is well supplied with electric light points and gas, and also contains points served by a heavy power cable, and resistances for reducing the voltage of the current.

Beyond the dark room, also on the south wall, is a York stone bench for a battery of small incubators and sterilisers, running at various temperatures, the space underneath being fitted with glazed doors to form cupboards for the storage of apparatus. Between the incubator bench and the working bench a third slate bench has been built on brick pillars for the use of ward clerks, and others, for the examination of sputum or other material suspected to be tuberculous. This, like the other bench, is provided with electric light and gas, and water is laid on over two staining sinks. Under each of the "seats" of the main bench is fitted a movable drawer unit of five various sized drawers for the personal apparatus of the workers, and two similar units are fitted under the sputum bench.

At the far end of this laboratory are two small rooms, that on the left a bulbing room for vaccines, and that on the right a library. The bulbing room is provided with a small entrance lobby to form an air lock, for into the room itself—whilst the operation of bulbing vaccines is in progress—filtered germ-free

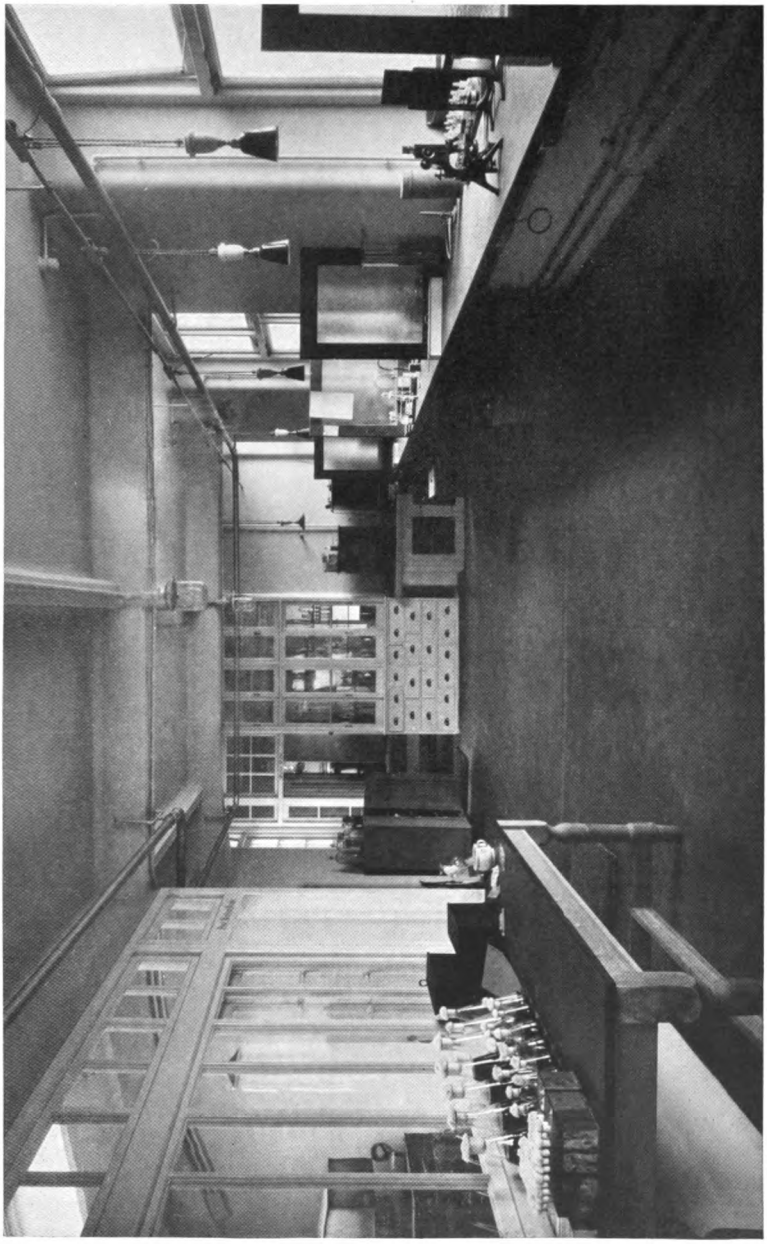
air is pumped by a centrifugal fan, driven by electricity, so that a slight positive pressure is maintained, the object aimed at being to minimise the risk of accidental contamination of the vaccines with air-borne organisms. This room, lighted by electric light, is supplied with gas for Bunsen burners and blow pipes, and a large steam steriliser, arranged on a slate bench. The floor, which is kept moist, is laid in stone mosaic.

The library, in which the Departmental Library and the Records of the Department will be stored, communicates by means of a spiral iron staircase with the Director's Room above, and also down a short flight of stone steps with a basement room, partly underground, for the storage of chemicals; this latter, as it is provided with a carpenter's bench, will also serve as a workshop for the manufacture of various pieces of apparatus. In the area outside the chemical store is fixed the air filter for the bulbing room and its centrifugal fan, the motor being controlled by an electric switch inside the store room.

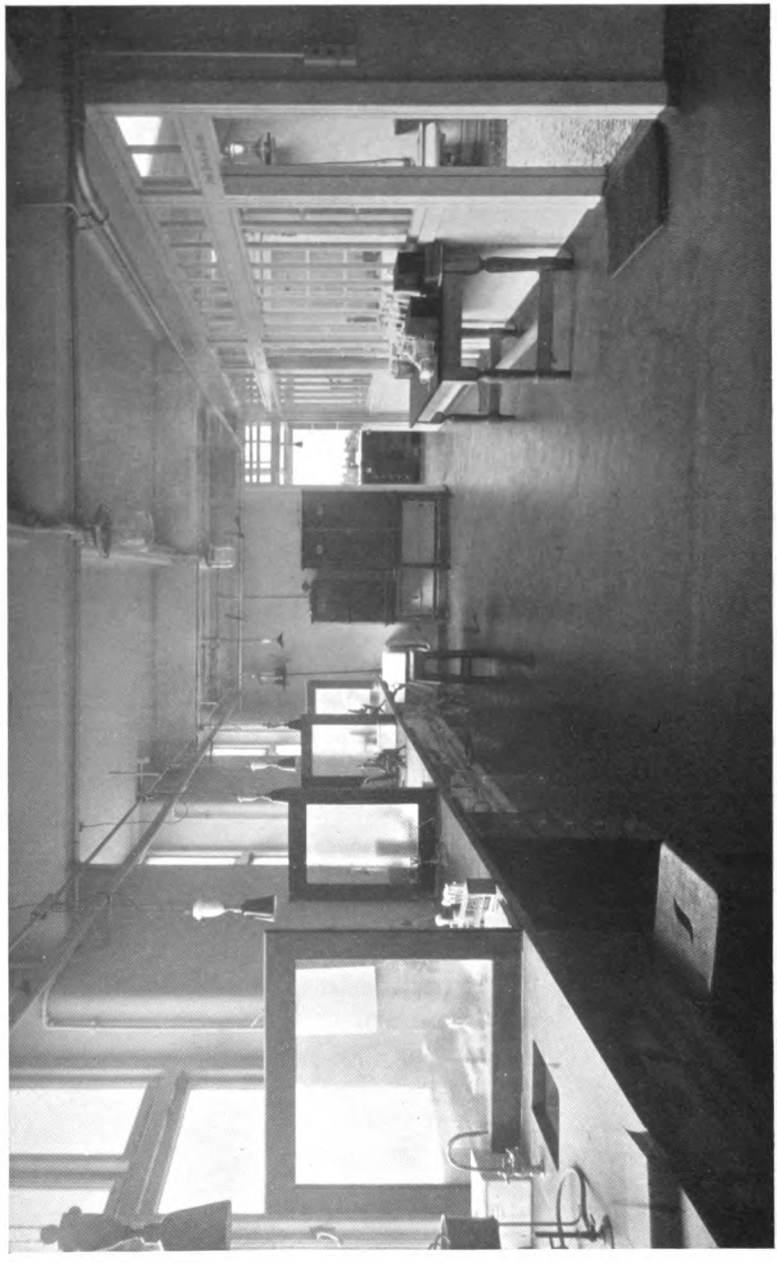
Four other rooms on the ground floor open on to the secondary corridor which runs north and south. First, on the east side is the store room for the stock of glass apparatus, flasks, bottles, etc. Next come two incubator rooms, one opening from the other, the first, to take the stock cultures and to serve as cool incubator, is regulated at 22°C.; it also serves the purpose of an air lock to the second room, which is a seven foot square hot incubator room (37°C.), heated by three electric radiators and regulated by a Hearson's capsule. These rooms, lit by electric ceiling lights, are well insulated by double walls four inches apart, built of thin hollow bricks, the interspace being packed with asbestos wool; outside these walls is a twelve-inch air space, and beyond that an ordinary nine inch brick wall. Both incubators are well ventilated, air taken from the corridor passing through the outer cool room, entering the hot room below a radiator, and passing out through an upcast shaft in the far corner.

Opposite the Incubator Rooms is situated the Refrigerator Room, seven feet square, thoroughly insulated in the same way

The New Laboratories of the Bacteriological Department.



RESEARCH LABORATORY, looking west (MEDIUM MAKING ROOM beyond), showing POST-MORTEM ROOM on the left.



RESEARCH LABORATORY, looking east, with DIRECTOR'S LABORATORY beyond, and showing POST-MORTEM ROOM and OPERATING ROOM on the right.

as the Incubator Rooms, and provided with an adequate air lock. Its floor is of stone mosaic, and the walls and ceiling of white porcelain tiles. This room is not yet actually finished, as it has still to be cooled by brine pipes, connected up to the ammonia compressors in the new engine room under Tabard House.

Next this cool room is a passage in which is now being erected a high-power Delépine centrifuge, to run at 8,000 revolutions per minute, driven by a 5 h.-p. motor (purchased out of a sum of money given to Sir Alfred Fripp by Reg. H. Prance, Esq., to provide research apparatus for the Department). This piece of apparatus will be of inestimable value to the Department. The room nearest the main corridor on the west side is a Photo-micrographic Studio, including a well-fitted Dark Room for developing, with a complete series of Wrattan's safe-light screens fitted to its window. The studio itself is intended to accommodate the photo-micrographic camera, Edinger's apparatus, enlarging and copying cameras, and the necessary outfit for making lantern slides, etc.

First Floor.

The main or central portion of the first floor is given over to a laboratory for research and special investigations. This is a room 40 feet long by 20 feet wide and 8 feet high, lighted by four north windows and three central bucket ceiling lights and seven pendant lights. It is equipped with hot and cool incubators (cellular and ordinary) of Hearson's manufacture for various temperatures. The long slate bench on the north side, subdivided by glazed partitions, similar to the one in the laboratory below, gives ample space for four workers. Beneath the bench are drawer units similar to those in the Diagnosis Laboratory.

Built against the south wall, and separated from the laboratory by glazed screens, are two rooms well lit by skylights, the one (11 feet by 7 feet) for animal inoculations, equipped with steam steriliser, an aseptic operation table, and an overhead rail for an electrical surgical motor; and the other (13 feet by

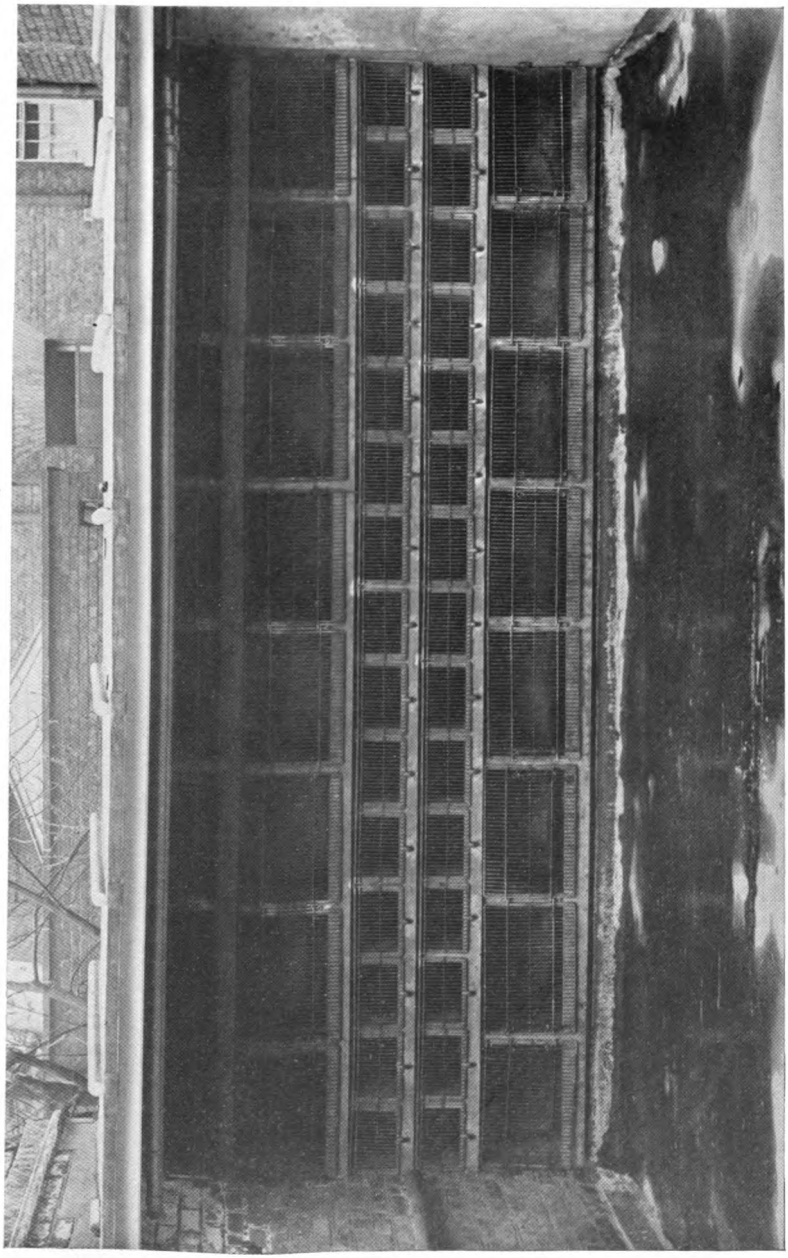
7 feet) for the post-mortem examination of experimental animals, with steam steriliser, and slate bench, and, in addition, a powerful gas incinerator by Fletcher, Russel & Co. for the destruction of the cadavers and other infective material. Both these have stone mosaic floors. Both are provided with hot and cold water supplies laid on over glazed earthenware sinks.

At the eastern end is the private laboratory of the Director of the Department, which communicates by the winding stairs previously mentioned with the Library below. This is a well-lit room, 12 feet by 20 feet, and furnished like the other laboratories with slate benches, incubators, etc.

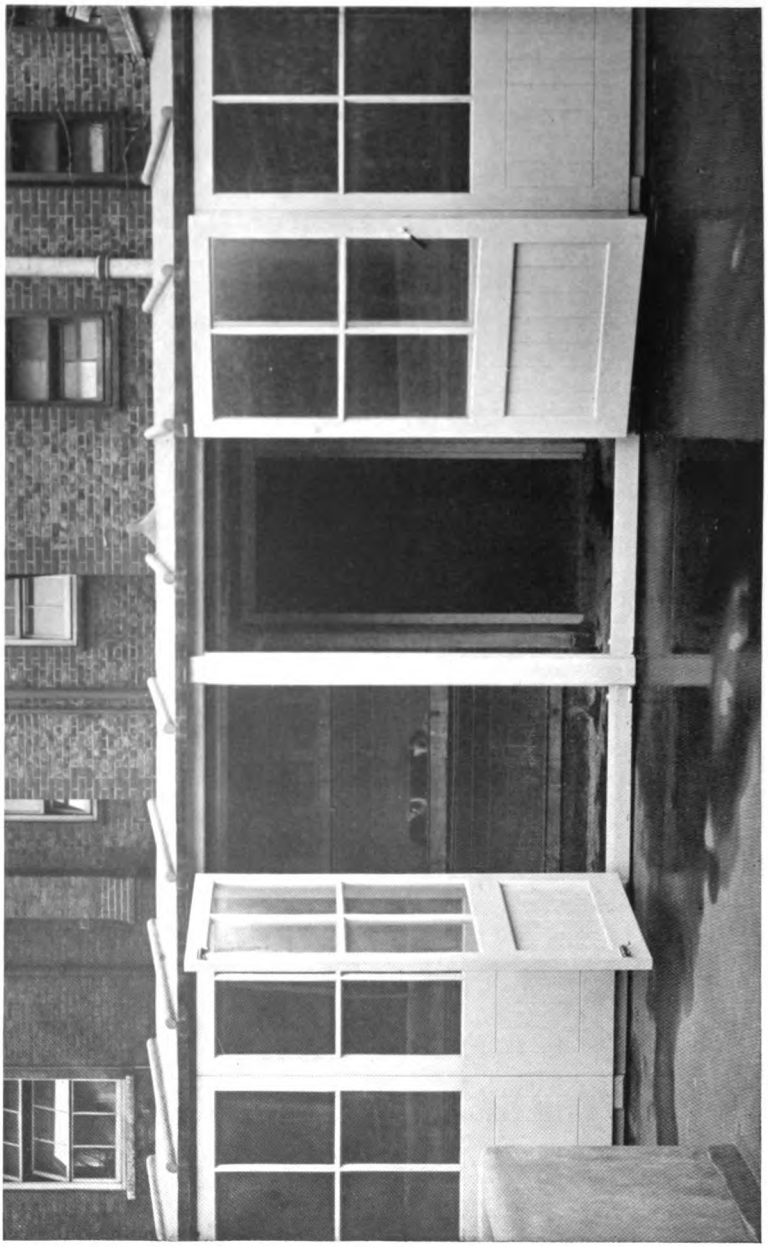
Opening from the end opposite to the Director's Laboratory is the room (15 feet by 10 feet) for the preparation of nutrient media. The wall between this and the Research Laboratory is formed by a cupboard and drawer fitment which is available for the use of workers in either room. In the Media Room, too, is placed the large stone reservoir for distilled water. Beyond the Media Room is the Sterilising Room, in which are housed the large autoclave, and the steam oven for sterilising media, and by the side of the latter is a serving hatch into the Media Room; also the electric hot-air sterilisers and the electric serum inspessator, as well as the large sinks for washing up glass ware and their draining shelves. This room has a mosaic floor, and the walls and ceiling are lined with glazed white porcelain tiles.

Roof.

In the Special Investigation Laboratory, close to the Media Room Door, is another door opening on to a flat between the Laboratory and the Orthopædic Workshops (which occupy the upper part of the site of the old Chemical Theatre, and are, consequently, above the glass store, Incubator Rooms, and Refrigerator Room of the Bacteriological Department). From this flat a flight of steps ascends to the central portion of the roof of the Department. This has been asphalted, and at each end of it has been erected an animal house, one with a south-west aspect, for inoculated animals, the other, facing north-east, for



INOCULATED ANIMAL HOUSE in course of construction, showing arrangement of concrete cages with wire doors, prior to erection of moveable door-front.



"STOCK" ANIMAL HOUSE on roof, completed with moveable door-front.

stock animals. The interval between the two houses on the north side (that is to say, overlooking the disused churchyard) has been closed in by a brick wall six feet high. Beneath the ridge of the roof, and behind the house for stock animals, is a small room for the storage of fodder and bedding (sawdust) for the animals. It also contains the steam-heated apparatus for preparing distilled water which, after condensation, is conducted in tin compo piping to the reservoir in the Media Room below. The animal cages vary in size for different animals, and are constructed in ferro-concrete and finished in cement, and are provided with swinging galvanised wire doors. Each house is closed in with glazed doors on a hinged wooden framework which can be opened out or entirely removed in the hot weather. The houses are lighted by electricity, heated by steam pipes, and adequately supplied with water.

The whole of the constructional work, woodwork, painting, heating, lighting, and plumbing, and the fitting, was carried out very efficiently by the Foreman of the Hospital Works Department, who spared no pains in working out the plans and designs of the Director. The mosaic flooring of the corridors, and the Bulbing, Sterilising, Operating, and Post-mortem Rooms was also laid by the Works Department, but the wood pulp floors (Decolite) of the remaining rooms and laboratories was put down by the Bell's United Asbestos Company.

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, OR OBTAINED

OTHER DISTINCTIONS, DURING THE YEAR 1913.

University of London.

Degree of Master in Surgery.

T. D. M. Stout

Degree of Doctor of Medicine.

G. Dunderdale	A. Neville Cox	H. F. Renton
T. Evans	R. Stout	

Degrees of Bachelor of Medicine and Bachelor in Surgery.

Honours.

W. L. Webb

Pass.

G. Covell	G. T. Foster-Smith	G. S. Miller
A. J. Eagleton	V. Glendining	W. Robinson
J. A. Edmond	J. M. Joly	J. A. Ryle
A. S. Erulkar	S. Keith	A. L. Shearwood
A. L. Fitzmaurice	C. D. Killpack	

Second Examination for Medical Degrees.

March, 1913.

Part I.

Organic and Applied Chemistry.

J. A. M. Alcock	E. N. Glover	R. G. Mayer
H. J. Bensted	H. M. Gray	L. P. Waghorn
E. de Robillard	T. L. Heath	K. M. C. Woodruff

Part II.

Anatomy, Physiology, and Pharmacology.

J. E. Clark	R. O. H. Jones	H. Q. F. Thompson
J. Gaymer Jones	F. A. Knott	

Part I.

July, 1913.

H. F. G. Berncastle	J. Joffe	E. S. Phillips
G. W. Heckels	G. E. Kidman	

Part II.

A W. Cocking	P G. McEvedy	D. M. MacManus

First Examination for Medical Degrees.

August, 1913.

M. Ba Thin	E. C. Hinde	H. N. Prichett
E. F. Deacon	F. R. Leblanc	F. Young
	G. P. Lindsay	

December, 1913.

C. H. Atkinson	J. C. Gie	*F. C. Millar
C. C. Bennett	W. A. Hawes	A. Rathouse
W. A. Flynn	C. G. Haynes	W. H. Steavenson
	N. Kamchorn	

**Awarded a mark of distinction in Physics.*

University of Oxford.

Degree of Doctor of Medicine.

R. C. Mullins

Degree of Bachelor of Medicine.

F. A. Hampton	W. H. Ogilvie	J. F. Venables

Second Examination for M.B. Degree.

Pathology.

L. R. Broster	C. P. Symonds	T. A. Townsend

University of Cambridge.

Degree of Master in Surgery.

H. L. Attwater	L. Bromley	J. G. Saner

Degree of Doctor of Medicine.

H. L. Duke | N. Mutch

Degrees of Bachelor of Medicine and Bachelor in Surgery.

F. S. Adams	W. C. D. Maile	J. L. M. Symns
W. T. Chaning-Pearce	A. S. Seabrooke	C. Warner
A. C. Clifford	V. F. Soothill	A. M. Zamora

Second Examination for Medical and Surgical Degrees (New Regulations).

Part II.

Pharmacology and General Pathology.

E. S. Taylor.

Second Examination for M.B. Degree,

Part II.

Pharmacology and General Pathology.

P. R. Boswell | E. C. W. Starling

University of Durham.

Degree of Doctor of Medicine (for Practitioners of 15 years' standing).

J. G. B. Coleman | H. G. Dodd | E. Rae Thomas

Degrees of Bachelor of Medicine and Bachelor in Surgery.

Jap-Ah-Chit | G. E. Warner Lacey

Degree of Bachelor of Medicine.

H. H. Elliot | P. Savage

Royal College of Surgeons of England.

Final Examination for the Fellowship.

J. S. Bookless | J. T. Morrison | E. G. Schlesinger
A. H. Todd

Royal College of Surgeons of Edinburgh.

Examination for the Fellowship.

A. S. Roe | R. Stout

Royal College of Physicians of London.

Elected to the Fellowship.

W. H. Maxwell Telling

Passed the Examination for the Membership.

N. Mutch.

Final Examination for the Conjoint Diploma of the Royal College of Physicians of London and the Royal College of Surgeons of England.

January, 1913.

A. C. Clifford
A. B. Danby
G. D. Eccles
N. Garrard
G. W. B. Garrett
H. C. Godding
A. M. Henry
F. C. Hunôt
D. W. John
K. J. T. Keer
H. V. Leigh
L. M. J. Menage
A. G. H. Moore
O. G. Morgan
J. L. Perceval
C. H. G. Pochin
J. A. Ryle
A. L. Shearwood
P. J. Watkin
S. Wickenden
L. D. Wright

April, 1913.

R. S. de C. Bennett
E. W. Blake
F. Collar
A. C. L. Ad'rifat
G. B. H. Jones
E. M. Mahon
W. H. Ogilvfe
D. V. Pickering
R. C. Poyser

July, 1913.

G. W. M. Andrew
J. R. Barrow-Clough
P. H. Berry
R. Creasy
H. W. Evans
C. H. Gould
R. O. Hilton Jones
J. W. Kemp
R. H. Lucas
A. V. Moberly
E. D. Scott
E. S. Taylor
A. Tilbury
W. C. Whitworth

October, 1913.

F. V. Bevan-Brown
F. Cook
W. L. Gwyn Davies
A. J. Eagleton
A. S. Erulkar
A. C. Hancock
J. L. Lauder
J. L. D. Lewis
H. Mather
J. York Moore
C. S. Lane Roberts
W. E. Tanner

MEDALLISTS AND PRIZEMEN,

JULY, 1914.

Open Scholarships in Arts.

Eric Roland Webb, Dean Close School, Cheltenham, £100.
Gordon Frank Peters, Kent Coast College, Herne Bay, £50.

Open Scholarships in Science.

Eric Clark Hinde, Preliminary Science Class. Guy's Hospital, £120.
Frederick Currer Miller, St. Paul's School, West Kensington, £60.

Scholarship for University Students.

Arthur Gerard East, B.A., St. John's College, Oxford, £50.

Open Scholarships in Dental Mechanics.

October, 1913. Hugh Muir Smith, £20.
May, 1914. Richard John Gustavus Handel, £20.

Scholarships in Dental Mechanics for Pupils of Guy's Hospital.

October, 1913. George Dee, £20.
May, 1914. Pierre Jean Hugo, £20.

Junior Proficiency Prizes.

Leonard Snowden Debenham, £20.
Gordon Ernest Lovell Simons, £15.
Jack Joffé, £10.

The Beaney Prize for Pathology.

William Morris Lansdale, £34.

The Michael Harris Prize for Anatomy.

Leonard Snowden Debenham.

The Wooldridge Memorial Prize for Physiology.

Leonard Snowden Debenham.

The Hilton Prize for Dissections (1913).

John Francis Carter-Braine,
James Gaymer Jones, } Equal.

Dental Prizes.

First Year's Prize in Dental Subjects.

Geraint Arthur Pennant.

Second Year's Prize in Dental Subjects.

Donald Clewer.
Maurice George Whitten, Certificate.

Second Year's Prize in General Subjects.

Maurice George Whitten.
Frank Edward Welton, Certificate.
Donald Clewer, Certificate.

Prize for Operative Dental Surgery.

Arthur Edmund Lowein.
Wilfrid George Hollands, Certificate.

Newland-Pedley Gold Medal for Practical Dentistry.

Arthur Edmund Lowein.
Reginald Joseph Ryland, Certificate.

Golding-Bird Gold Medal & Scholarship in Bacteriology.

William Arthur Young, £20.
William Edward Tanner, Proxime Accessit.

Treasurer's Gold Medal for Clinical Medicine.

Cassidy de Wet Gibb.

Treasurer's Gold Medal for Clinical Surgery.

Cedric Sydney Lane Roberts.

THE PHYSICAL SOCIETY.

Honorary President.—Sir James Goodhart, Bart., M.D., LL.D.

Honorary Vice-Presidents.—Sir George H. Savage, M.D., Frederick Taylor, M.D., Charters J. Symonds, M.S.

Presidents.—C. S. Lane Roberts, E. D. Scott, W. E. Tanner, E. S. Taylor, J. York Moore, R. W. P. Jackson, C. P. Symonds, A. S. Liebson, W. M. Lansdale, A. H. Harkness, F. Bevan-Brown, C. J. Cooke, B. Burnside, C. E. Petley, S. Long, and C. Dean.

Hon. Secretaries.—G. T. Mullally and T. B. Heaton.

CLINICAL APPOINTMENTS HELD DURING THE YEAR 1913.

HOUSE PHYSICIANS.

A. C. Jepson	W. E. S. Digby	C. Aldis
W. S. George	P. Smith	T. P. Cole
T. I. Bennett	J. F. G. Richards	W. Robinson

HOUSE SURGEONS.

G. T. Foster-Smith	V. Glendining	C. A. R. Gatley
L. Milton	G. S. Miller	J. M. Joly
J. L. Perceval	R. C. Ozanne	R. A. Fawcus

ASSISTANT HOUSE SURGEONS.

C. Aldis	H. W. Evans	A. J. E. Smith
T. P. Cole	G. S. Miller	J. M. Joly
A. Seabrooke	R. A. Fawcus	R. C. Ozanne
W. R. Reynell	J. F. G. Richards	J. L. Perceval
A. C. Clifford	O. G. Morgan	J. A. Ryle
	E. S. Taylor	

OUT-PATIENT OFFICERS.

O. G. Morgan	W. Robinson	R. C. Ozanne
A. Seabrooke	W. R. Reynell	J. A. Ryle
J. M. Jarvie	C. A. R. Gatley	W. S. George
T. P. Cole	L. Milton	T. I. Bennett
R. A. Fawcus	J. M. Joly	G. S. Miller
	J. F. G. Richards	

OBSTETRIC RESIDENTS.

A. J. McNair	H. R. Bastard	E. W. Blake
W. J. D. Smyth	H. L. James	A. B. Danby

CLINICAL ASSISTANTS.

R. A. Fawcus
W. Robinson
A. B. Danby
J. L. Perceval
A. C. Clifford
C. H. G. Pochin
N. Garrard
W. H. Ogilvie
R. C Ozanne
H C. Rook
G. E. Genge-Andrews
W. R. Reynell
H. W. Evans
A. J. E. Smith
C. H. Gould
E. D. Scott
J. F. G. Richards
A. Seabrooke
O. G. Morgan
J. A. Ryle
E. M. Mahon
E. S. Taylor
F. A. Hampton
L. B. Stringer

CLINICAL ASSISTANTS IN THE MEDICAL WARDS.

J. A. Martin
E. H. Mahon
O. G. Morgan
W. C. Whitworth
W. L. Gwyn-Davies
F. V. Bevan-Brown
C. F. Pedley
W. R. Pryn
E. G. Martin
P. R. Boswell
P. H. Berry
J. L. D. Lewis
G. Covell
A. Tilbury
F. W. Lawson
J. F. Venables
C. H. Gould
C Sherris
D. C. Scott
R. C. Matson
J. A. Ryle
D. W. John
E. D. Scott
E. S. Taylor
H. Sharpe
S. Wilson
F. D. Annesley
E. C. W. Starling
C. R. Smith
C. Dean

CLINICAL ASSISTANTS IN THE SURGICAL WARDS.

E. C. Cline
A. C. Clifford
J. R. Barrow-Clough
F. E. R. Laborda
G. W. King
A. Tilbury
J. W. Kemp
R. P. Ninnis
A. J. Drew
F. D. Annesley
C. S. L. Roberts
S. Wilson

CLINICAL ASSISTANTS IN THE MEDICAL OUT-PATIENTS.

F. C. Newman
T. W. Sheldon
E. L. Jones
G. W. M. Andrew

DRESSERS IN THE THROAT, EAR, CHILDREN'S, AND GENITO-URINARY DEPARTMENTS.

E. C. W. Starling
P. R. Boswell
A. B. Danby
W. D. Galloway
A. Tilbury
H. Elliot
C. E. Petley
W. E. Tanner
S. Wilson
R. J. Hearn
G. Covell
H. W. Evans
P. D Scott
E. D. Scott
L. R. Pickett
J. Stephenson
A. N. Minns
A. C. Clifford
T. R. Trounce
D. R. Jones
J. A. Martin
W. A. Young
J. H. Parry
C. F. Pedley

DRESSERS IN THE THROAT, EAR, SKIN, AND ORTHOPÆDIC DEPARTMENTS.

L. Muir-Smith
A. Abd-el-Al
A. V. Moberly
D. W. Jones
W. M. Lansdale
S. S. B. Harrison
L. A. J. Graham
W. H. Nicholls
D. C. Scott
J. E. Kemp
C. R. Smith
H. Parry-Price
A. L. Anthony
J. S. Cocks
R. W. Cushing
T. W. Sheldon
F. D. Annesley
F. E. R. Laborda
C. de W. Gibb
H. L. P. Peregrine
D. O. Richards
J. E. Davies
C. S. L. Roberts

DRESSERS IN THE EYE AND NERVOUS SYSTEM DEPARTMENTS.

A. Wills	F. W. Lawson	C. Dean
J. L. Perceval	W. E. Tanner	F. C. S. Broome
W. R. Reynell	H. P. Whitworth	H. Gould
F. G. L. Scott	C. M. Ryley	R. Creasy
J. R. Barrow-Clough	W. H. Ogilvie	N. H. W. Saw
R. C. Matson	H. G. Dresing	H. Mather
C. H. L. Harper	O. G. Parry-Jones	R. Sells
J. York Moore	F. Tooth	L. Horsley
A. H. Harkness	T. A. Townsend	H. L. Messenger
A. Tilbury	S. A. Liebson	F. V. Bevan-Brown
	G. L. Attwater	

SURGEONS' DRESSERS.

W. D. Galloway	H. G. Dresing	C. R. Smith
G. B. Pritchard	H. L. Messenger	F. G. L. Scott
F. E. R. Laborda	R. B. Campion	T. R. Trounce
E. R. Bailey	D. M. McManus	H. N. W. Saw
G. L. Attwater	H. L. P. Peregrine	P. D. Scott
W. M. Lansdale	O. G. Parry-Jones	H. Stephenson
S. S. B. Harrison	R. Sells	H. Parry-Price
W. H. Nicholls	C. de W. Gibb	B. Burnside
R. W. Cushing	W. A. Young	A. H. Harkness
R. C. Fairbairn	J. H. Parry	H. L. Messenger
C. E. Petley	J. S. Cocks	J. E. Davies
F. L. Spalding	D. O. Richards	L. R. Pickett
A. S. Liebson	L. Horsley	A. L. Anthony
L. A. J. Graham	K. Westman	J. F. Mackenzie
J. F. H. Stallman	C. H. Medlock	R. J. Quesada
H. Q. F. Thompson	L. S. Fry	W. L. E. Reynolds
R. W. P. Jackson	H. F. T. Hogben	A. K. Day-Lewis

OPHTHALMIC HOUSE-SURGEONS.

A. H. Todd | H. C. Rook

ASSISTANT SURGEONS' DRESSERS.

A. H. Harkness	M. J. T. Wallis	C. E. Petley
J. S. Cocks	D. O. Richards	B. Burnside
J. E. Davies	A. S. Liebson	R. W. Cushing
L. Horsley	J. Stephenson	L. R. Pickett
J. H. Parry	A. L. Anthony	R. W. O. Jackson
C. H. Medlock	W. H. Nicholls	K. Westman
B. Sampson	J. F. Mackenzie	L. S. Fry
W. L. E. Reynolds	H. Q. F. Thompson	R. Quesada
H. F. T. Hogben	A. K. Day-Lewis	S. W. Coffin
C. C. G. Gibson	L. R. Broster	C. P. Symonds
E. J. Cooke	A. C. M. Coxon	W. L. Partridge
J. F. H. Stallman	R. A. Holmes	G. B. Tarring
R. Curle	K. L. Bates	A. F. Rook
N. E. Kendall	G. D. Gripper	V. E. Lloyd
C. W. W. Baxter	A. L. Punch	J. G. Stevens
R. N. Craig	J. E. Clark	G. S. B. Long
	J. Kyle	

DENTAL SURGEONS' DRESSERS.

F. D. Annesley	J. York Moore	L. Du Verge
F. L. Spalding	P. R. Boswell	N. H. W. Saw
J. A. Martin	S. Wilson	

OBSTETRIC DRESSERS AND EXTERNS.

R. O. H. Jones
A. C. Hancock
P. Savage
L. Du Verge
F. V. Bevan-Brown
W. R. Pryn
L. Muir-Smith
S. Wilson
W. E. Tanner
A. N. Minns
P. R. Boswell
Abd-el Al
R. P. Ninnis
C. R. Smith
T. R. Trounce
G. L. Attwater
D. MacManus
S. S. B Harrison
F. G. L. Scott
R. Sells

H. S. Groves
C. S. L. Roberts
J. York Moore
R. C. Matson
J. F. Venables
T. A. Townsend
C. Lambrinudi
D. C. Scott
H. P. Whitworth
C. F. Pedley
C. Dean
E. R. Bailey
C. Sherris
P. R. Chevreau
F. E. R. Laborda
W. D. Galloway
W. M. Lansdale
H. L. P. Peregrine
B. Burnside
H. Parry-Price

F. H. Dodd
A. J. E. Smith
A. J. Drew
F. W. Lawson
D. V. Pickering
C. G. McClymont
A. H. Taymour
F. C. S. Broome
A. Wills
T. W. Sheldon
E. C. W. Starling
L. B. Campion
D. C. Scott
D. R. Jones
H. G. Dresing
R. J. Hearn
F. L. Spalding
N. H. W. Saw
H. H. Elliot
P. D. Scott

CLERK IN THE ELECTRICAL DEPARTMENT.

A. J. Drew

MEDICAL WARD CLERKS AND POST-MORTEM CLERKS.

P. D. Scott
W. M. Lansdale
O. G. Parry-Jones
L. A. J. Graham
H. W. Evans
E. D. Scott
R. J. Hearn
J. F. Mackenzie
B. Sampson
R. W. P. Jackson
K. Westman
L. R. Pickett
F. L. Spalding
J. E. Davies
J. H. Parry
K. L. Bates
A. F. Rook
S. W. Coffin
L. R. Broster
C. P. Symonds
R. O. H Jones
J. York Moore
R. W. Cushing
B. Burnside
R. Quesada
V. E. Lloyd
C. W. W. Baxter
R. M. Craig
D. H. A. Galbraith
C. H. Edwards

F. A. Knott
H. Parry-Price
W. A. Young
R. Sells
H. S. Groves
H. Gould
H. H. Elliot
W. L. Gwyn-Davies
C. H. Medlock
H. Q. F. Thompson
L. S. Fry
A. S. Liebson
A. H. Harkness
J. S. Cocks
D. O. Richards
R. H. Holmes
W. A. Easton
C. C. G. Gibson
A. C. M. Coxon
R. Curle
G. T. Gripper
F. H. Dodd
F. V. Bevan-Brown
L. S. Fry
G. Noott
J. E. Clark
G. S. B. Long
M. J. T. Wallis
V. R. Hirsch
L. H. B. Evans
J. G. Jones

S. S. B. Harrison
C. de W. Gibb
J. F. H. Stallman
A. H. Taymour
D. R. Jones
C. F. Pedley
J. R. Barrow-Clough
W. L. E. Reynolds
A. K. Day-Lewis
H. F. T. Hogben
L. Horsley
W. A. Young
C. E. Petley
A. L. Anthony
W. L. Partridge
N. E. Kendall
E. J. Cooke
M. Chadwick
E. W. Bowell
G. B. Tarring
C. S. L. Roberts
J. Stephenson
W. H. Nicholls
J. G. Stevens
J. Kyle
J. Totton
A. L. Punch
K. N. Purkis
J. J. Conybeare
E. Biddle

SURGICAL WARD CLERKS.

W. L. Partridge	E. W. Bowell	K. Westman
A. C. M. Coxon	M. Chadwick	G. B. Tarring
E. J. Cooke	A. L. Punch	G. D. Gripper
R. Curle	A. F. Rook	K. L. Bates
G. Noott	S. W. Coffin	W. A. Easton
R. A. Holmes	C. C. G. Gibson	J. E. Clark
J. G. Stevens	R. N. Craig	T. Totton
J. Kyle	M. J. T. Wallis	V. E. Lloyd
J. J. Conybeare	J. G. Jones	K. N. Purkis
C. H. Edwards	F. A. Knott	L. H. B. Evans
E. Biddle	D. H. A. Galbraith	V. R. Hirsch
E. A. Leviseur	J. F. Carter-Braine	A. W. Cocking
D. M. P. Whitcombe	A. F. G. Guinness	A. Traill
E. C. de M. Morgan	J. A. Fairclough	R. C. Portway
G. B. Dowling	A. G. East	

DENTAL APPOINTMENTS HELD DURING THE YEAR 1913.

DENTAL HOUSE SURGEONS.

M. Schneider	L. P. Harris	F. W. Lawrance
C. F. Haime	W. G. S. Neely	E. R. Saul

ASSISTANT DENTAL HOUSE SURGEONS.

C. Glover	C. F. Haime	C. H. Edwards
W. K. Fry	L. D. Wright	E. E. Johnson
J. S. Palmer	W. A. Bulleid	K. C. Pitman
D. Clewer	S. Adams	R. L. Donn

CLINICAL ASSISTANTS IN THE CONSERVATION ROOM.

E. R. Saul	R. G. Bradley	G. V. Saunders
L. S. Pilbeam	K. C. Pitman	R. J. Pickett
J. W. Beacock	D. Clewer	P. E. Bernhard
M. G. Whitten	D. A. Bevis	J. M. Barnes

ASSISTANT DEMONSTRATORS IN DENTAL MECHANICS.

W. A. Bulleid	W. G. Lloyd	C. L. Laver
	J. M. Barnes	

ASSISTANT DEMONSTRATORS IN DENTAL METALLURGY.

H. G. James	G. V. Saunders	L. S. Debenham
	W. G. Lloyd	

ASSISTANT DEMONSTRATORS IN DENTAL MICROSCOPY.

C. H. Edwards | L. S. Pilbeam

DRESSERS IN THE GAS ROOM.

E. E. Johnson
H. C. Duggan
M. Pearson
G. F. Charles
W. K. Fry
S. E. Johnson
L. C. Cohen
A. D. Marston
R. J. Pickett
R. H. Rix
N. R. Dyke
H. J. Edwards
W. C. Hammond-Williams
H. L. Smith
A. A. R. Crowe
S. Adams
J. M. Barnes
M. G. Whitten

J. W. Beacock
K. C. Pitman
R. H. Gaverick
L. D. Windermer
R. W. Ballard
J. S. Palmer
E. R. Saul
B. Isaacs
F. H. Morrell
R. C. W. Staley
A. H. Cole
R. G. Bradley
A. B. G. Underwood
G. L. Cutts
J. M. Stebbings
C. H. Oliver
D. A. Bevis
P. E. Bernhard
W. G. Lloyd

W. A. Bulleid
S. Stevens
C. F. Haime
C. E. Thomas
L. Alabone
E. C. Rycroft
A. S. Clarke
W. O. Roberts
L. S. Pilbeam
D. Clewer
A. A. Brown
R. G. Cowell
L. D. Wright
P. King
C. J. Crocker
H. Millett
M. G. Henry
P. T. Pearce

DRESSERS IN THE EXTRACTION ROOM.

W. A. Turner
M. F. Hopson
W. G. Hollands
F. E. Welton
A. Bulleid
W. G. Lloyd
C. H. Laver
C. A. E. Cook
P. T. Pearce
E. H. Bryant
F. A. Stiven
J. A. S. Wright
C. R. Cade
L. H. Cross
H. Wallis
J. L. Oates
C. M. Bullpitt
H. L. Thorn
J. L. Garrard

A. H. Cole
W. J. McB. Allan
S. A. Withers
B. P. Richards
C. L. Curle
K. F. McAlpin
G. H. Howe
F. E. Rudd
P. E. Bernhard
P. E. Jessop
T. D. Corke
H. L. Smith
E. G. Browne
H. Curtis
J. F. Batsford
W. J. R. Viljoen
G. W. Wheldon
J. E. Wright
C. Bethencourt

A. D. Marston
K. C. Pitman
W. E. Coe
A. C. Dean
J. J. S. Hill
D. G. Pearce
E. F. Llarena
G. A. Johnstone
R. J. Ryland
A. E. Lowein
E. A. Dawson
F. D. Neal
P. L. van Schalkwijk
L. Machin
J. W. Beacock
J. L. Mugford
H. V. Stebbings

CASUALTY DRESSERS.

R. Cowell
V. R. Dyke
M. G. Whitten
D. A. Bevis
J. M. Barnes
G. L. Cutts
R. J. Ryland
C. A. E. Cook
W. E. Coe
W. G. Lloyd
L. P. Richards
W. G. Hollands
J. A. Johnstone
K. F. McAlpin

C. J. Crocker
A. D. Marston
D. Clewer
M. G. Henry
E. C. Lewis
F. E Rudd
A. C. Dean
D. F. Small
H. Millett
F. E. Welton
H. V. Stebbings
J. L. Mugford
F. D. Neal
W. K. Fry

R. C. W. Staley
L. D. Wright
H. J. Edwards
W. C. Hammond-Williams
P. E. Bernhard
P. T. Pearce
P. E. Jessop
A. E. Lowein
D. G. Pearce
J. W. Beacock
J. F. Batsford
W. G. McB. Allan

GUY'S HOSPITAL.

MEDICAL AND SURGICAL STAFF.

1914.

Consulting Physicians.—Sir James Goodhart, Bart., M.D., LL.D.; F. Taylor, M.D.

Consulting Surgeons.—Thomas Bryant, M.Ch.; 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 Anæsthetists.—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.D.

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, M.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., M.S.
F. E. SHIPWAY, M.D.	A. H. TODD, M.B.
H. T. DEPREE, M.B.	A. J. MCNAIR, M.B., B.C.

Bacteriologist.
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