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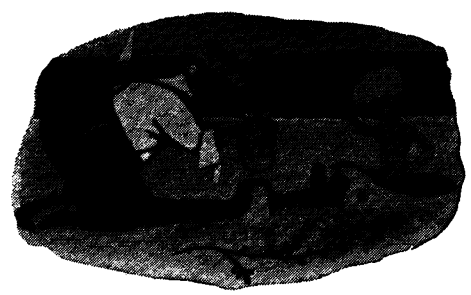

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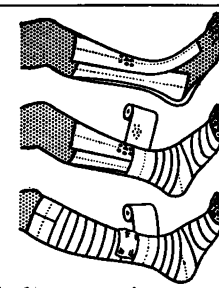

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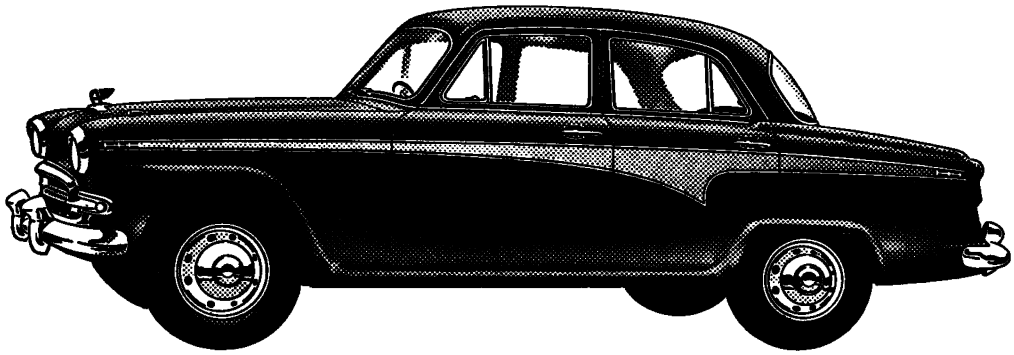

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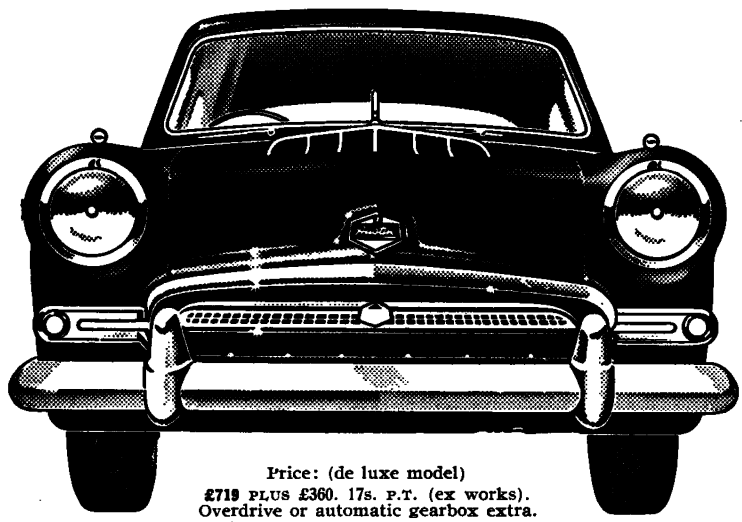

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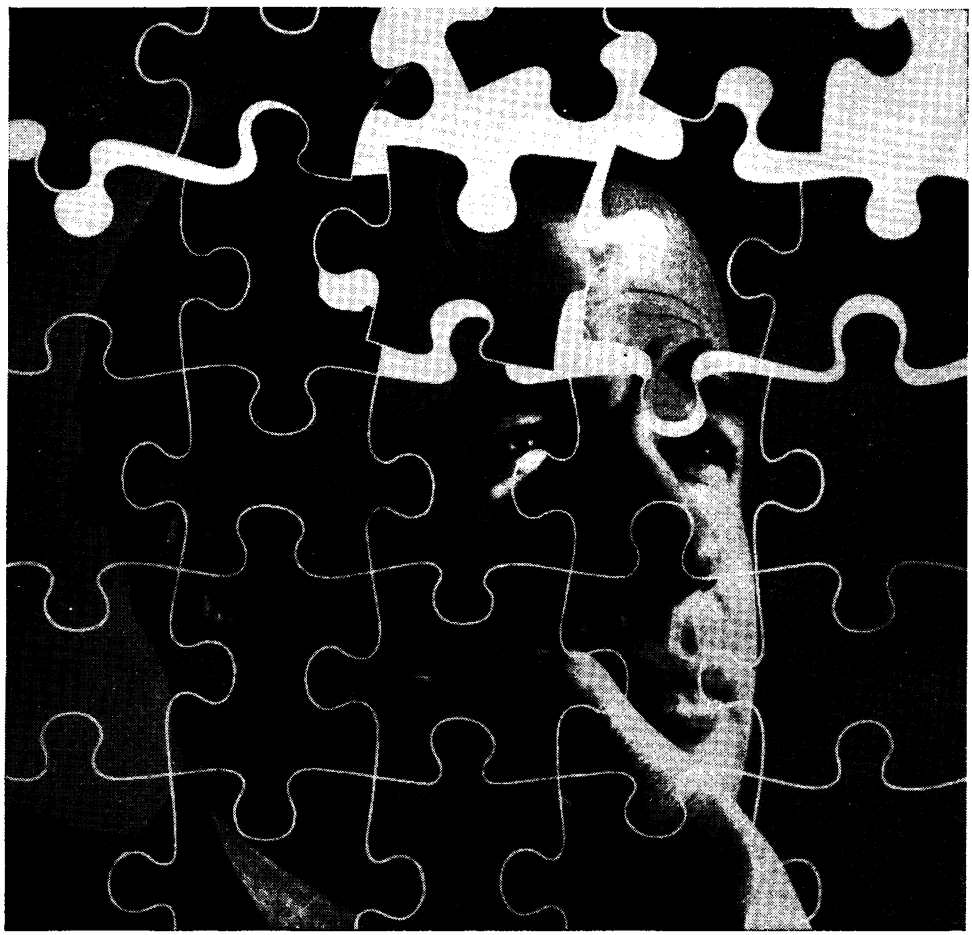

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CONTENTS

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THE ULSTER MEDICAL JOURNAL

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THE
ULSTER MEDICAL JOURNAL

PUBLISHED ON BEHALF OF THE ULSTER MEDICAL SOCIETY

Vol. XXVII 1st MAY, 1958 No. 1

Pædiatrics in this Changing World

By KENNETH H. TALLERMAN, M.C., M.D., F.R.C.P.
Physician to the Children's Department, The London Hospital

Address to the Ulster Pædiatric Society, March, 1958

I BELIEVE that there are times when it is useful and helpful to review certain aspects of medicine, and the changes that have occurred in pædiatrics during the past four decades are such, I think, as to warrant some evaluation of the present position and an attempt perhaps to forecast the future position of pædiatrics within the framework of general medicine.

Certain diseases which were common in infancy and childhood a few decades ago are now infrequently seen; others which were relatively rare are now relatively common. The pattern of disease has changed, so that the course of many of the diseases which we are called upon to deal with has altered. Modern techniques and modes of treatment have brought about profound changes not only in prognosis but in our whole approach to our patients. The place of pædiatrics within the sphere of medicine has changed, its position and influence in the curriculum of a teaching school have altered, and it now has a recognised place of its own. Pædiatricians themselves are of a somewhat different type from that of the past, and even our own speciality has become reorientated. Let us consider these points, and then think about what the future may hold.

Although pædiatrics as a speciality was well recognised in Central Europe and the United States a long while ago, in England this was hardly the case even when I was a medical student. British contributions to the literature of children's diseases had certainly been far from negligible, but it was only in the 1920s that pædiatrics in this country really began to take a place of its own. The general pattern of pædiatric practice in America was then, and always has been, different from that in vogue here. There, although consultants are recognised, they are

relatively few in number, but most parents in U.S.A. would not feel secure or serene without the aid of a pædiatrician who, although perhaps a specialist, is essentially a general practitioner working within a particular age group. Our system, in which the general practitioner, if conscientious and properly trained, can and should act as the medical guide, philosopher and friend of the whole family, calling upon the pædiatric consultant when need arises, is, I think, the better arrangement.

In London about forty years ago, Still, Frew, Edmund Cautley, Hector Cameron, Eric Pritchard, Jewsbury, Wilfrid Pearson and Donald Paterson, were, I think I am correct in saying, the only persons practising exclusively among infants and children, while I believe that outside of London, John Thomson, Leonard Findlay and G. B. Fleming stood apart, for Leonard Parsons in Birmingham, and others in such larger cities practised among adults as well as children. Such outstanding men as Robert Hutchison, happily still with us, John Poynton, Hugh Thursfield and Morley Fletcher, although concerned with sick children who occupied much of their practice, were all actively engaged in looking after adults, who constituted the major portion of their work. In pædiatrics their interest was almost exclusively centred on sick children, and I strongly suspect that most of them regarded as revolutionary the idea that they should devote time and trouble to the care of the normal infant and child with a view to simply preventing disease. It is indeed questionable whether at that time they would have had much hope of success even if they had entertained such an idea, for, apart from vaccination against smallpox, no protective inoculations were available, and social conditions were much against them. They were not particularly interested in the normal or in small deviations from it, and in fact when the Maternity and Child Welfare Act came into force in 1916 it certainly was not the children's specialists who exerted a guiding influence upon the Infant Welfare Centres which then sprang into being. Now, of course, this is all very much a part of our work, and we expect to have charge of the infant nurseries of obstetric units.

How far things have altered is surely reflected in the designation of the professorial chairs. Most if not all of these are now endowed as Chairs of Child Health, and the occupants are no longer simply Professors of Diseases of Infancy and Childhood. The whole emphasis has changed, and I feel that it is not going too far if one regards pædiatrics at present as one of the outstanding branches of preventive medicine, with a very close association with the Medical Officers of Health and their departments. In this connection we have surely only to think of what has come about as the result of prophylactic inoculation against diphtheria and, with less dramatic results, in regard to whooping-cough. Whereas in 1936 the number of cases of diphtheria in England and Wales amounted to 57,729, with a death rate of 3,081, in 1956 cases notified numbered only 53 with 8 deaths. The mortality for whooping-cough has also fallen significantly. B.C.G. vaccination is now surely a well-established procedure, and has succeeded in significantly lessening the risk of tuberculous infection in infancy and childhood, and thereby decreasing the incidence of miliary tuberculosis and tuberculous meningitis, which

are now far less commonly met with. Today immunisation against acute anterior poliomyelitis is a further important stride forward.

When one considers the question of nutrition in childhood the picture also is a relatively bright one. Gross malnutrition in this country is virtually a thing of the past; feeding problems in infancy still present themselves, generally where an ignorant woman is mismanaging things, but the educational value of the Child Welfare movement has made its impact felt, and the Welfare State has, of course, had the effect of raising the general standard of living beyond the dreams of thirty years ago. Infantile rickets and scurvey now rarely occur, for our knowledge of their ætiology has made them preventable diseases. The incorporation of vitamin D in dried milks has had an enormous influence, and the value of this is especially apparent in many cases where the mother fails to take up and give her infant the cod-liver oil which is available to her, but the child still remains free from rickets. The provision of orange juice concentrate, together with educational propaganda in regard to nutrition, has had its effect in banishing infantile scurvy.

Acute infantile gastro-enteritis is at the present time a rarity, and its mortality has considerably diminished. I gave a communication on this condition about twenty-five years ago at the time of the International Pædiatric Congress in London, and reported a mortality rate of no less than 75 per cent. of all cases except those so mild as not to have required any parenteral fluid therapy. This experience was not exceptional, but today the picture is a very different one.

Diphtheria, already mentioned, has now reached almost vanishing point, a total of perhaps 2 to 5 cases being reported in a week for the whole of the British Isles. Congenital syphilis is, of course, another disease which, owing to the modern care and treatment of the mother, has become much less common.

The advances in surgery, especially cardiac surgery, are such that certain types of congenital cardiac abnormalities (approximately one-third of the total cases met with) are amenable to treatment, and as a result not only good health but full physical efficiency may be attained by many patients who in the past would have been doomed to deterioration relatively early in life.

Physiotherapy and occupational therapy, the importance of which is now more fully realised, play a most important part in aiding our patients and hastening their recovery.

Metabolic disorders such as hypercalcæmia and galactosæmia, and other metabolic disturbances associated with amino-aciduria are now recognised, owing to help available from our biochemical colleagues, and to the introduction of paper chromatography, where before they were passed over, and symptom complexes which earlier were recognised but not understood have, with advances in methods of investigation and fuller knowledge, been sorted out. The anæmias of infancy and childhood are nowadays more clearly defined and understood. Von Jaksch's pseudo-leukæmia infantum now no longer concerns us, and what masqueraded under the title of neonatal icterus gravis familiaris is now designated hæmolytic disease of the newly-born, and its ætiology is known and its treatment is

established on a firm basis. The criteria calling for exchange blood transfusion in this disease are well recognised, even if there is still controversy over some border-line cases.

On the debit side we now have fibrocystic disease of the pancreas to deal with, but no doubt this disease was actually occurring before it was finally put on the map by Dorothy Andersen some eighteen years ago, those patients whose symptoms were mainly intestinal being accepted as suffering from cœliac disease, and those with predominantly respiratory symptoms as simply suffering from recurrent attacks of broncho-pneumonia. Then retrolental fibroplasia, now happily on the wane since we have learnt, or think we have learnt the error of our ways. This is surely an outstanding example of a disease resulting from therapeutic enthusiasm acting in a wrong direction. Looking back it is hard to imagine why premature infants who were not cyanosed or suffering from any definite respiratory embarrassment were subjected to oxygen therapy at all. The fact that retrolental fibroplasia is largely a man - made disease, brought about by well - meaning physicians, makes the tragedy of those infants who suffered blindness only the more poignant. And again with hypercalcæmia, how far is its increased incidence directly due to an excessive fortificaton of dried milk with vitamin D, for which we also have to take our share of the blame? In the case of both acute leukæmia and hæmolytic disease of the newly-born, where the incidence appears to have increased so much during recent years, are we also to some extent responsible? Is leukæmia sometimes occurring as a result of a tendency to over-do radiography, and can the increased incidence of hæmolytic disease be attributable sometimes to the too free use of blood transfusions?

In regard to the changing pattern of actual diseases, we have the example of acute juvenile rheumatism, the severity and mortality of which have lessened considerably in the past four decades, as has the incidence of chorea. Acute rheumatism is also, of course, much less common than it used to be, though the line of the graph does not show a continuous fall, and from time to time the yearly totals jump up again. Scarlet fever too is less severe than in former times and its incidence has decreased: in 1936 notifications totalled 104,698, in 1956 the figure was 33,103. Cyclic vomiting, so fashionable under the diagnostic label of "acidosis" thirty years ago is, I think, also seen less often now in its full-blown form. On the other hand dysentery, particularly the Sonne type, has markedly increased or is now much more frequently recognised, and here it is the younger children that are the most frequent victims. Asthma is another disorder which is, I think, more often met with nowadays, and in younger children. The change in age incidence of acute anterior poliomyelitis is striking, and to refer to this as infantile paralysis is now quite unjustified.

Prognosis is, of course, closely bound up with the whole question of treatment, and this in turn is dependent on an accurate diagnosis, since diagnosis must precede treatment. Biochemical and radiological investigations now make possible the diagnosis of conditions which until recently remained obscure, and with the diagnosis established a proper approach can be made to the question of treatment.

It is perhaps in the common infective conditions that the most noticeable results have been achieved. Until recently the main causes of death in childhood were pneumonia, tuberculosis and other infectious diseases. Owing largely to the control of these diseases mortality in infancy has fallen by over 50 per cent., and in childhood by more than 70 per cent. in the past fifteen years.

It is thanks to the use of the sulphonamide drugs and the antibiotics that the outlook in the acute pulmonary infections, tuberculous infections, and in all forms of meningitis has become completely altered. Pneumonia in childhood, except perhaps staphylococcal pneumonia in infancy, today causes relatively little anxiety, and empyema has become an uncommon complication. Tuberculous infection can usually now be controlled, and tuberculous meningitis, in which the prognosis was hopeless only a matter of some fifteen years ago, can be regarded as a curable disease—certainly in any patient with a history of no longer than ten days' duration, and who is still alert at the time when treatment is begun. Meningococcal meningitis no longer really holds any terrors, a most remarkable thing for those who remember the time when daily or twice daily lumbar punctures were called for in this disease, with intrathecal injections of antimeningococcal serum, in the rather vain hope that this would benefit the patient. If life was saved, the sequelæ were then, as often as not, almost as distressing as a fatal outcome.

All this is most heartening, but there are certain aspects of such treatment which should be borne in mind, but which I am afraid are too often lost sight of. Firstly, the only too frequent uncritical use of antibiotics, which may result in some eventual harm to the patient, and an unnecessary expense to the State. Secondly, there is a tendency to forget the feelings of these young patients and the psychological trauma that may be inflicted on them. I say this because one knows how often injections are ordered by junior medical officers, and sometimes by others who ought to know better, without a critical consideration of the situation, because they are either not thinking of the individual patient but of the *case,* or perhaps have not stopped to think at all. There is no real excuse for this. I wonder if we are paying enough attention in our teaching and training of housemen to the importance of instilling the idea that patients should be considered as individual human beings, and that any procedure involving pain or discomfort should be turned over very carefully in their minds in relation to their child patient before it is ordered. I well remember how when I was a house physician to the late Sir George Frederic Still he insisted on this approach, and how he himself had the greatest abhorrence of even ordering a lumbar puncture to be carried out, although, of course, he was well aware of its necessity. On the other hand, there are fortunately many ways in which greater consideration is shown nowadays for the feelings of sick children. In connection with surgical procedures great efforts are made to avoid distressing or frightening children. With proper premedication before anæsthesia a child often becomes oblivious to everything that happens after leaving the ward on his way to the operating theatre. Anæsthetists with pædiatric experience also employ various tricks to divert the child, who then slips into a

state of unconsciousness during the induction of anæsthesia without realising what is occurring.

We are becoming increasingly aware of the psychological disturbance resulting from the admission of a child to hospital, and all possible steps are being taken to meet this and to minimise it. One of the great and most important changes that has come about within the last ten years is in relation to the visits of parents to the children's wards of hospitals. Whereas formerly visiting was kept to a minimum, because it was thought to upset a child and to carry a risk of the introduction of infectious diseases from outside, we now know that children are likely to suffer a great psychological disturbance if not visited by their parents, and that as a rule the fear of infection being introduced is groundless. In fact, one is coming to realise the value of a mother actually coming into the hospital with her child and carrying out as much of his care as an untrained person is capable of. This has the further advantage of relieving the load on the nursing staff, which is far from unimportant.

More sense is now shown in relation to the mother and her newborn infant. The child is allowed to remain at her side in the maternity ward most of the day, instead of being segregated in an infant nursery as used to be customary, and she is encouraged to take an active part in looking after it. So far as the feeding of infants is concerned, this has become far less rigid both in regard to the diet itself and in the matter of fixed times of feeding. This elasticity is to be welcomed as a matter of common sense, and is a revolt against an unnecessarily rigid routine. At the same time, in the case of young infants I myself prefer, for the sake of both the child and its mother, intervals of roughly four hours between feeds rather than the so-called "self demand" régime, and I personally believe that an infant can be over-fed and that one should not always allow it to take all that it will as a matter of course. The introduction of mixed feeding at an earlier age is all to the good, but to begin this before 3½ to 4 months of age seems to me pointless. One can no doubt get away with a semi-solid diet before this age, but with what object? I cannot see that it is really advantageous. For much of the pioneer work that has led to this more enlightened outlook, I feel we owe a great debt to the late Sir James Spence of Newcastle.

The prominence given to psychiatric work in pædiatrics is another change that has come in our field. Hector C. Cameron, whose well-known book "The Nervous Child" is such a delight, was among the first to insist on the importance of the pædiatrician's work in dealing with children who show emotional disturbances and disorders of behaviour. Most of us would agree with this, and I think it essential that in the first instance such children should be seen by a pædiatrician, and only those needing very specialised attention should be referred to a psychiatrist. Of course the child guidance clinics have an important contribution to make in the handling of some of our patients, and not the least important of this may be the associated parent guidance involved. But pædiatricians by the very nature of their calling should, I believe, deal with many of the children who are now sent direct to a Child Guidance Clinic. Joseph Brennermann's paper on "Pædiatric Psychology

and the Child Guidance Movement," published in 1933,[1] which created quite a stir at the time, is still well worth reading, and what he says holds true today; he was under no delusion about the place of psychiatry within the sphere of pædiatrics, and expressed his fears of over-emphasising psychological distrubance in childhood, and insisted on viewing them in proper perspective.

The relative importance of child psychiatry within our sphere admittedly increases rather than diminishes because, with the tempo of modern life, the altering social conditions and customs, and the conquest of so much infective disease, the proportion of patients exhibiting behaviour problems increases. This change in the type of patient we are called upon to handle is reflected in the almost universal decrease in the call upon hospital beds for the admission of sick children suffering from the common organic diseases. If improved social conditions associated with the Welfare State, nutritional care and teaching at the Welfare Centres, the use of protective inoculations, and the widespread use of antibiotics, are all fully exerting their influence, it is hardly surprising that organic disease in childhood is diminishing, and is likely to diminish still further. Ideally, of course, despite the fact that Boards of Management of hospitals are greatly distressed by low bed occupancy, the aim and final achievement of a first-class medical service should be to have relatively empty hospitals.

As I see it, there are other factors which may diminish the work and scope of the pædiatrician and against which we must exert our influence. I have spoken of the tendency of the psychiatrist to make inroads in a field which is primarily ours, but the same is now happening in relation to other specialists, and is becoming more apparent as their particular branches of medicine grow and develop.

At a time when pædiatrics in this country was first establishing itself, it was one of only a few special branches of medicine. With the increasing scope of pure cardiology, endocrinology, thoracic surgery and neurosurgery, more and more children tend to be regarded as patients who should be referred primarily to such specialists rather than to pædiatricians. This I am quite certain is a mistake, and not in the best interests of the child. I agree, of course, that it is arguable that any patient irrespective of age, with a cardiac lesion or an endocrinc disorder for example, might well be referred to a physician specialising in such diseases. Many family doctors too tend to think in terms of disease, rather than in that of the age of their patient, when seeking a consultant's opinion. This, however, is surely wrong, for a child is not just a miniature adult, but is physically, mentally and psychologically a quite different sort of person, and only those who spend their time studying infants and children appreciate how great the differences are, and learn how to tackle the problem of sickness as it affects the child as a whole. This, of course, is well known to us, and the Pædiatric Committee of the Royal College of Physicians of London, in a recent report to the College, dealt with the aspects of this matter relating to the care of children in hospital. The thing, however, that strikes me so forcibly in this connection is the very fact that many medical men who are not primarily concerned with children fail to appreciate that there *is* a problem at all. To them a child with cardiac disease or diabetes is

simply a patient suffering from cardiac disease or diabetes, the physical, to say nothing of the emotional and psychological factors that are so important in childhood hardly seem to enter their ken.

It is essential that pædiatricians should take every opportunity of stressing this aspect of their work and of keeping a watchful eye on this tendency, otherwise sick children are likely to be placed at a disadvantage. If medical students have this point of view not only put before them but battered into them, then whether they subsequently enter into general practice or become physicians or surgeons in a special field, they will have absorbed the idea of the pædiatrician's role and will invoke his help; this, I have little doubt, is the course most likely to be of benefit to sick children.

The pædiatrician's work and sphere of action is being increasingly affected by various factors, and resulting from them a decrease in the need for hospital beds for children is now recognised; Douglas Gairdner commented on this recently in a paper before the Section of Pædiatrics of the Royal Society of Medicine.[2] These factors have already been alluded to, but—to recapitulate—they include: higher standards of living; the rising standard of infant care and feeding; protective inoculations; the lowered morbidity of certain diseases such as infantile gastroenteritis, tuberculosis and pneumonia, depending largely on the existence of potent antibacterial drugs and their ready availability to all; the diversion of certain types of disease to other specialists; the more active role of the child psychiatrist. Does this mean that the pædiatrician is on the way out? I most certainly think that this is not so, though he will have to be on the alert and may even at times and in some places have to battle a little.

At the moment, British pædiatrics and pædiatricians are maintaining the highest standards and are exceedingly active. This I believe to be also true of pædiatricians in every other civilised country. With us the British Pædiatric Association has, since its foundation in 1928, gone from strength to strength, and has played an increasingly important role. It is consulted frequently by the Ministry of Health, and by many other Authorities in matters relating to Child Health and Welfare, who look to it for advice and guidance.

The number of pædiatricians has increased greatly since the National Health Service came into being in 1948, for consultant pædiatricians have been allocated throughout all the Regions, as an integral part of the Service. Furthermore, various local and regional pædiatric societies and clubs have been formed, and are very active indeed. These societies exert a great influence, and have a most beneficial effect on pædiatric work throughout the country. It is also worth noting perhaps that a large number of candidates for the Diploma in Child Health continue to come forward to sit the examination.

There is no doubt that the pædiatrician's sphere of activities is still considerable, and apart from medical and scentific research, including the investigation of various diseases and disorders of infancy and childhood as yet imperfectly understood, there are certain clinical fields still wide open to him, in which his training

and experience will increasingly prove invaluable. First of all, antenatal and perinatal pædiatrics is surely an absorbing and fruitful ground for his efforts, for today there are as many deaths in the first month of life as in the whole of later childhood. Here there must be joint responsibility for the fœtus and infant, not obstetric interest and responsibility merely until the infant is born, and pædiatric interest and responsibility only after its birth has taken place. Then, tropical pædiatrics is only beginning to come into its own, and the association that has been effected between the Hospital for Sick Children in Great Ormond Street, and the Medical School in Uganda is undoubtedly a helpful move, and points the way to similar liaisons between pædiatricans and children's hospitals and departments in this country and those in developing countries.

And what about our position in regard to medical education? In this matter I personally believe that we have a really outstanding and even decisive sphere of influence. In the first place, every general practitioner finds himself called upon to deal with pædiatric problems in his day-to-day work, and the teaching of this subject to medical students must be done by pædiatricians. Then we are, after all, general physicians working within a particular age group, perhaps the only true general physicians left, for we see more "general" medical cases than do most of our colleagues who deal with adults, but who tend more and more to specialise in particular groups of diseases. We are therefore able to teach students a great deal of general medicine by the way, and also by comparing and contrasting how the features differ in one and the other age group we are able to stimulate interest and underline the importance of the pædiatrician's knowledge in relation to sick children.

Furthermore, pædiatrics lends itself perhaps more than the other branches of medicine to a very human approach. Apart from other things, one is invariably dealing not with one patient but with two, the mother and the child, and often with a still wider family circle. The pædiatrican with his experience is well equipped to educate students in the right method of approach, and to teach them through consideration of the child's and the parents' feelings, fears and hopes, how to be a first-rate doctor in the widest and best sense. This may not seem of importance from an academic point of view, but it is of vital importance to any patient and the members of his family, and absolutely essential if that patient is a child.

The importance of being a really good doctor, someone, that is, whose constant aim is to help and benefit his patient and not add to his burden of sickness, is too often overlooked in a medical school, although it may receive lip service. The tendency to over-investigate and to over-treat is only too common. Many investigations can be avoided by a person of experience who has taken a careful history, but residents of necessity have limited experience, and the art of taking a history is not always acquired early in a man's career. Over-treatment often results from enthusiasm and lack of judgment, and must be expected in those who lack experience. Discussion of a patient with a senior before embarking on the treatment is the key to proper training of a junior man. In the case of a hospital resident learning his craft, is there any real need for him, except occasionally, to

initiate special investigations until the individual patient has been discussed with his senior? After all, he accompanies his chief round the wards two or three times weekly for this very purpose. It is at such times that the resident should begin to acquire the knowledge of what investigation is or is not necessary, and what is the appropriate treatment called for in each individual patient. This is the time to instil the idea so well expressed by Sir Robert Hutchison in 1953 when he wrote, "From inability to let well alone; from too much zeal for the new and contempt for what is old; from putting knowledge before wisdom, science before art and cleverness before common sense, from treating patients as cases, and from making the cure of the disease more grievous than the endurance of the same, Good Lord, deliver us."

A propos of these words of wisdom, I would like to comment on a problem which seems to me to be a difficult one, and one which requires much thoughtful consideration. I am referring to the position in which we now find ourselves in regard to a child suffering from a serious congenital mental defect, such as mongolism or a primary amentia, who contracts a severe intercurrent infection, bronchopneumonia for example. In days gone by we had no specific treatment available, and such a patient, particularly a mongol with an associated cardiac defect, was likely to succumb. Today, with antibiotics and the sulphonamide drugs at our command, and with oxygen tents at our service, there is every likelihood that the child so treated will recover. Ought we, when faced with such a patient, to intervene, or should we allow nature to take its course? Obviously each case of this kind must be most carefully reviewed, and the patient's particular circumstances weighed in the balance, but I cannot help thinking that it is generally wisest and most kind both for the patient's and for the family's sake to allow nature to take its course. If, however, we act in this way, there is a risk, owing to the modern trend and attitude of mind, of having to face criticism, and even possibly a legal action for negligence in treatment. This, of course, means that a doctor in the discharge of what he believes to be his duty for the ultimate benefit of his patient may be deflected from it by fear of the consequences. This is a dilemma which fortunately did not present itself in bygone years, but we now have to face it. I feel sure that this problem should be put before junior men and discussed with them. It is for each one of us to decide what line of action should be taken in the case of any particular patient, and probably our knowledge of the parents' outlook and feeling in regard to their child will be the deciding factor influencing our course of action.

There is also the difficult problem of whether and when to persist with treatment in a disease such as acute leukæmia, when one knows that the outcome will be fatal, and that remissions, whether natural or brought about by modern treatment, such as the use of cortisone, are relatively short and evanescent. The prolonging of an agonising situation is often more than one feels is really justified in the ultimate interests of the patient and his immediate family. Here again the issue is largely a moral one, and it must, of course, be faced by each one of us in the light of his experience and his belief of what is for the best.

As a last thought, if we may take a very long-term and somewhat philosophical view of what lies ahead, is the pædiatrician, useful though he may be, adding to world problems? With a fall in infant and child mortality and morbidity and with an ever-increasing world population, what are we heading for? One most sincerely hopes that the scourge and horrors of war with its attendant slaughter may be coming to an end, and if that is so, will the production of food keep pace with the numbers requiring it? One is perhaps sometimes tempted to wonder whether, after all, the pædiatrician, with his opposite number the geriatric specialist, is not on the way to creating problems more difficult to solve than those he has up to now succeeded in solving and overcoming.

1. BRENNERMANN, J. (1933). *J. Ped.*, **2**, 1.
2. GAIRDNER, D. (1956). *Proc. Roy. Soc. Med.*, **49**, 974.

REVIEW

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THIS book was written by Mr. Geoffrey Flavell at the request of the medical students at London Hospital. It is written especially for students, housemen, and general practitioners—but even specialists in this branch of surgery and general surgeons would gain much by reading it.

It is surprising that a subject in which most of the major surgical advances in the past decade have been made, and which deals with many of the most common diseases seen in any hospital, should be regarded as a small "special department," and insufficient time given to its study in most teaching hospitals. If all students were to read this book it would help them to realize what a diversity of diseases are found within, or around, the thorax, and to learn much about them.

The book is written in clear, concise English. There is no ambiguity about the author's views, and although the book would probably have benefited by more discussion on alternative lines of treatment, this was omitted in the interests of economy.

It is divided into three parts. Part 1 describes the surgery of the lungs, pleural and thoracic orifices; Part 2 surgery of the œsophagus; Part 3 surgery of the heart and great vessels.

Practically all the diseases which occur in the chest are described—dealing with symptoms, signs, treatment, including pre-operative, operative, and post-operative. Throughout the book are many short case histories from Mr. Flavell's own experience which help to stress or illustrate a particular point. There are many excellent line drawings and over one hundred very good X-ray reproductions. The type is clear and the paper is good. This is an excellent book for all those who wish to learn about the surgical diseases of the chest and their treatment.

T. B. S.

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T. B. S.

Diagnosis and Treatment of Adrenocortical Insufficiency

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Based on a Paper read to the Ulster Medical Society on 20th February, 1958

ONE hundred and three years ago Thomas Addison (Addison, 1855) described the disease which now bears his name. Since his classical description, knowledge of adrenal disorders has widened and increased until it has developed into one of the most exciting and fruitful fields of clinical and biochemical research in the history of medicine. After Addison, many years had to pass before new chemical techniques were available to unravel the complexities of adrenocortical chemistry, but once research workers had mastered these methods, rapid and striking progress was made. In 1927, adrenal cortical extracts were prepared (Hartman, et. al., 1927; Rogoff and Stewart, 1927), which were capable of prolonging the survival time of the adrenalectomised animal. In 1930 the close connection between the pituitary gland and the adrenal cortex was demonstrated (Smith, 1930), when it was shown that hypophysectomised rats developed rapid atrophy of the adrenal cortex which could be avoided by transplants of anterior pituitary tissue, the active principle of which was later named adrenocorticotrophin or A.C.T.H.

In 1936, Kendall discovered Compound E or 11 - dehydro - 17 hydroxy - corticosterone (Mason, Myers and Kendall, 1936) which is now known as cortisone, and shortly afterwards, 17 hydroxycorticosterone called Compound F by Kendall, and now known as hydrocortisone, was isolated. Finally, in 1953, aldosterone, a potent salt-retaining substance, was crystallised by Simpson and Tait in England, and by Reichstein and Wettstein in Switzerland (Simpson, Tait, Wettstein, Neher, v Euw, and Reichstein, 1953).

HORMONES OF THE ADRENAL CORTEX.

The adrenal cortex is essential for life, and three distinct types of steroids are secreted.

1. *The glucocorticoids.* These steroids, of which hydrocortisone is the most important, raise the blood sugar, promote the storage of carbohydrate and fat, induce protein breakdown and inhibit inflammatory and allergic reactions. They have a weak salt-retaining effect. Cortisone, and the new synthetic analogues, prednisone and prednisolone have similar actions, although the salt-retaining property of the synthetic compounds is weaker. The secretion of hydrocortisone is dependant on the action of corticotrophin but there is a delicate interaction between the pituitary and adrenal glands so that the output of corticotrophin is regulated by the level of hydrocortisone in the body. In addition there is a diurnal variation in

the production of hydrocortisone which is at its peak between 8.0 a.m. and 10.0 a.m. and at its lowest level between 10.0 p.m. and midnight. The cause of this variation is unknown and is uninfluenced by light, food, sleep or activity.

The administration of corticotrophin or cortisone for therapeutic purposes has a profound effect on the pituitary adrenal axis. Cortisone inhibits the pituitary gland directly and causes a secondary atrophy of the adrenal cortex. The effect of corticotrophin is similar although it acts in a different way. When it is injected the adrenal cortex is stimulated to secrete more hydrocortisone which in turn inhibits the pituitary gland. When these substances are withdrawn it may take some months for the pituitary-adrenal function to recover fully.

2. *The salt-retaining hormones or electrocorticoids.* Aldosterone is the most active hormone in this group, and is probably the actual hormone formed in the adrenal cortex. It has a profound effect on body chemistry promoting the retention of salt, chloride, and water, and excretion of potassium. Its secretion is regulated mainly by the electrolyte and body water content through mechanisms not yet understood, but it may be partially under corticotrophin control. Deoxycortone acetate (DCA) is a synthetic steroid which has a similar though much weaker action. Recently 9-α-fluoro-hydrocortisone, a synthetic analogue of hydrocortisone, has been introduced (Fried and Sabo, 1954). It is a potent substance, and approximately fifty times more active than hydrocortisone in its electrolyte regulating properties (Thorn et al., 1955).

3. *Sex hormones.* These are of three types; androgens, œstrogens and progesterone. Their effects are uncertain but they probably assist or potentiate the action of the hormones secreted by the gonads. Their secretion is partly under the control of the pituitary but other factors may be involved.

All the adrenal steroids are metabolised to a large extent in the tissues and are then excreted in the urine in conjugated form. The measurement of these substances provides, therefore, an indication of adrenocortical activity, and the methods employed in the clinical assessment of adrenocortical disorders will be described later.

Since the introduction of cortisone (Hench et al., 1949) the diagnosis and treatment of adrenocortical deficiency states has become an important branch of medicine. Now that adrenal substitution therapy is a practical measure, adrenalectomy is becoming increasingly popular for a number of disorders. Total or sub - total adrenalectomy is a standard procedure for Cushing's syndrome (Montgomery and Welbourn, 1957) and total adrenalectomy is being used in the treatment of certain forms of metastatic malignant disease (Cade, 1954) and for malignant hypertension (Van T'Hoff, 1956). Replacement therapy is thus a practical day to day problem for many patients. The use of adrenocortical steroids for the treatment of a number of disorders such as rheumatoid arthritis, ulcerative colitis, asthma and the collagen diseases, increases the liability of an adrenal crisis in the event of severe stress or abrupt termination of treatment and makes the understanding and study of adrenal insufficiency a matter of considerable importance.

Adrenocortical Insufficiency.

Where insufficient amounts of hydrocortisone and aldosterone are produced to meet the body's requirements the syndrome of adrenal failure develops. This may be acute if the output of adrenocortical hormones falls abruptly, either as the result of surgical removal or acute destruction of the glands, or when adrenocortical reserve is impaired by reason of damage, exhaustion, or therapeutic inhibition by cortisone, and unable to respond to the needs of acute stress. Subacute or chronic adrenocortical insufficiency is more commonly seen and implies a more even balance between the hormonal requirements of the body and the adrenal reserve, but the imposition of any stress in these cases, is, of course, liable to result in acute crisis.

The ætiological classification of acute adrenal failure may be described as follows :—

Acute Adrenocortical Insufficiency.

(a) Surgical removal of the adrenal glands for

i. Cushing's syndrome.
ii. Metastatic cancer of breast and prostate.
iii. Malignant hypertension.

(b) Acute adrenal destruction

i. Infective adrenal apoplexy.
(Waterhouse-Friderichsen's syndrome).
ii. Adrenal infarction.
iii. Traumatic apoplexy.

(c) Effects of acute stress in cases with diminished adrenal reserve resulting from

i. Disease of the adrenal glands.
ii. Subtotal surgical removal of the adrenal glands.
iii. Pituitary inhibition by cortisone or corticotrophin.

The symptoms of acute adrenal insufficiency are the result of absolute or relative lack of adrenocortical hormones and are characterised by weakness, nausea, vomiting, apathy, confusion and signs of circulatory collapse with hypotension and tachycardia. If untreated, coma and death may follow swiftly. Symptoms may develop with alarming suddenness after adrenalectomy for Cushing's syndrome or after surgical operations or accidents in patients with latent and unrecognised adrenal insufficiency and are indistinguishable from oligæmic shock. In all varieties of acute adrenal crisis, effective treatment must be promptly administered if lives are to be saved; methods and details are described later.

Surgical operations on the adrenal glands.

It has been known for a long time that symptoms of acute adrenal insufficiency appeared within twenty-four hours after adrenalectomy. This made surgical correction of adrenocortical disorders extremely hazardous, and removal of adrenal

tumours was rarely attempted until the use of cortisone made adrenalectomy relatively safe. At present, patients are prepared for adrenal surgery by both pre- and post-operative hormone replacement, but even with this precaution acute crises still occur. These are commoner following adrenalectomy for Cushing's syndrome, than in patients whose adrenal function was previously normal. Presumably in Cushing's syndrome the patient has become habituated to a high level of circulating hydrocortisone so that operation, even in the presence of cortisone replacement, leads more easily to relative adrenocortical insufficiency.

Acute adrenocortical destruction.

Acute adrenal destruction is a rare condition. It occurs as the result of trauma, fulminating infections often but not exclusively due to meningococcal septicæmia, infarction of the glands of varying ætiology, or as a rare complication of pregnancy and the puerperium. The condition usually occurs in definite age groups.

Acute collapse associated with bilateral adrenal hæmorrhage may be found in the newborn and an incidence as high as one per cent. has been reported in babies dying at or shortly after birth (Forsham and Thorn, 1955). It most frequently follows prolonged and difficult labour. Adrenal apoplexy in the newborn is characterised by hyperpyrexia, tachypnoea, cyanosis and convulsions, but bacteriological and clinical evidence of infection and the purpura and hæmorrhagic manifestations of the Waterhouse-Friderichsen syndrome are absent.

Among children and young adults hæmorrhagic destruction of the adrenal glands occurs most commonly as the result of septacæmic infection. This results in the characteristic Waterhouse-Friderichsen syndrome with hyperpyrexia, cyanosis and diffuse petechial rash. Terminally, signs of circulatory collapse occur and coma and death may follow in a few hours. Occasionally hæmorrhagic destruction of the adrenal glands is caused by severe thoracic or abdominal injury.

Adrenal vein thrombosis with infarction may be a rare cause of adrenocortical destruction, but not a great deal is known about this condition. In some cases it may be a secondary feature of extensive hæmorrhagic destruction and not an initiating process. However, unilateral thrombosis can probably occur, without producing symptoms. This was a finding in one case of Cushing's syndrome, where the right adrenal gland was atrophic and weighed only 2.6 grams. Histological examination suggested that this had been the result of previous infarction.

Latent or unsuspected adrenal insufficiency.

Acute crises occurring in patients with diminished adrenocortical reserve who are subjected to stress are becoming more common (Allanby, 1957; Slaney and Brooke, 1957). Normally stress is met by increased secretion of corticotrophin, and this in turn leads to an additional output of adrenocortical hormone, which is sufficient to meet the body's needs. But his mechanism may break down if the pituitary gland is inhibited by the therapeutic administration of cortisone or corticotrophin, or if the adrenal cortex is damaged and unable to respond effectively to its physiological

stimulus. In such circumstances the response to stress is subnormal and an acute crisis may be precipitated. Emphasis must here be placed on the impairment of pituitary - adrenal function which invariably follows the long - term therapeutic administration of cortisone and corticotrophin. These patients are unable to withstand stress unless additional amounts of hormone are supplied to meet the need.

There are patients who appear perfectly healthy and yet possess only a limited adrenal reserve which may be overwhelmed by surgical operations, accidents or infections. In some, the development of circulatory collapse following an operation, may be the first manifestation of adrenal insufficiency (Welbourn, 1957).

The condition is commonly met with in one of two ways. Firstly, an apparently healthy patient without signs of obvious disease may be suffering from incomplete or suspected adrenocortical insufficiency.

"A woman, aged 50, had a right hemicolectomy performed for carcinoma of the cæcum. After operation her blood pressure remained low and could be maintained only with noradrenaline. After five days, when no other cause for the hypotension could be found, hydrocortisone was given intravenously. Within twenty-four hours the blood pressure remained normal without noradrenaline. Subsequent investigation showed that the patient was probably suffering from mild Addison's disease."

Secondly, it arises in patients who have known Addison's disease or adrenocortical impairment as the result of steroid therapy, who have an acute illness, accident, or who are inadequately prepared for surgery. An example of this was the case of a man aged 40 with Addison's disease, who was undergoing investigation before treatment started. Following gastroscopy under a general anæsthetic he developed acute peripheral circulatory failure and was saved only by immediate treatment with noradrenaline and cortisone.

Diagnosis of Acute Adrenal Insufficiency.

The diagnosis of acute adrenal destruction is rarely made in life, but in children and young adults the condition must be considered in cases of fulminating infection with hyperpyrexia, especially if meningococci are isolated. The presence of an extensive hæmorrhagic rash should suggest the possibillty of complicating adrenal hæmorrhage. Biochemical confirmation of the diagnosis cannot as yet be obtained, since chemical methods to detect the level of circulating 17-hydroxycorticoids are too lengthy to be of practical use, but a raised eosinophil count with more than 50 per cu.mm. is very suggestive of adrenocortical insufficiency. In every case where the condition is suspected on clinical grounds, treatment for acute adrenal insufficiency should be started immediately if irreversible shock is to be prevented and lives saved.

Acute adrenal insufficiency following operation on the adrenal glands themselves is easily recognised, but the rapidity with which a crisis may develop requires the most meticulous post-operative management. Two precautions which are of value are first, the insertion of a polythene catheter into the saphenous vein before the

operation, to provide an immediate channel for the infusion of hydrocortisone and noradrenaline if required, and secondly, the frequent measurement of the pulse and blood pressure until they have stabilised without specific intravenous therapy.

The diagnosis of adrenal crises in patients with unrecognised adrenocortical insufficiency who are subjected to operation or other forms of stress is often difficult. The possibility of adrenal dysfunction should be considered in all cases where the degree of shock is out of proportion to the stress, and particularly in patients undergoing surgery for tuberculous conditions and if there is, in addition, a previous history of vague ill-health, or indefinite gastrointestinal disturbances. A low pre-operative blood pressure, slight patches of pigmentation, and absence or thinning of axillary and pubic hair may point to the presence of adrenal insufficiency and should suggest the diagnosis. In all cases recognition and efficient treatment depends mainly on clinical awareness of the conditions predisposing to adrenocortical failure and the willingness of the surgeon to treat urgently suspected cases without waiting for confirmation of the diagnosis.

The Treatment of Acute Adrenocortical Insufficiency.

This divides itself into two parts. First, the treatment of the established condition, and secondly, the avoidance of acute adrenal insufficiency in those patients with impaired adrenal reserve.

The treatment of acute adrenal crisis.

The patient suffering from acute adrenal insufficiency is in a state of shock and often in a critical condition, so that prompt treatment and careful handling are essential. Principles of treatment consist in the correction of fluid and electrolyte imbalance, provision of adrenocortical hormone, prevention of hypoglycæmia, and the treatment of infection.

Blood is taken immediately for estimation of the serum electrolytes, blood sugar, and blood group, and a "two-way" drip set placed in position, while arrangements are made to record the pulse and blood pressure every fifteen minutes. An infusion of 500 ml. of 5 per cent. dextrose containing 100 mg. of hydrocortisone-free alcohol or 100 mg. of hydrocortisone hemisuccinate is started and run in during the first two to four hours. This may be repeated if there is no response, or continued more slowly if the patient's condition improves. If the fall in blood pressure is profound, or if it fails to rise after starting hydrocortisone, noradrenaline may be given as well. A solution of 4 mg. of noradrenaline in 500 ml. of normal saline is given as fast as necessary to restore the blood pressure. The rate of infusion, which requires careful adjustment every few minutes, is reduced slowly until the blood pressure remains stable without it. Cortisone acetate is commenced as soon as the drip is in place and continued at the rate of 25 mg. intramuscularly every six hours until the patient is able to swallow, when it is administered by mouth. The amount is then gradually reduced until the patient is receiving a maintenance dose of 37.5 or 25 mg. daily. Deoxycortone acetate (DCA) 5 ml. intramuscularly is given at the start of treatment and thereafter daily for the first two or three days,

and may then be omitted provided there is no sodium deficiency. In most cases cortisone is sufficient to maintain a satisfactory sodium level, but occasionally salt depletion occurs, so that the use of a potent salt-retaining hormone such as DCA or 9-α-fluoro-hydrocortisone is required. The use of these steroids, must, however, be carefully controlled, as their injudicious administration may result in hypertension, œdema and even heart failure.

During the first twenty-four hours between two and three litres of intravenous fluid will be required, but the exact volume and composition of the infusion will depend on the electrolyte levels and plasma specific gravity. Additional amounts of glucose may be required in patients with severe hypoglycæmia, but glucose is also necessary to meet calorie requirements until the patient is able to take nourishment by mouth.

When patients are seen in extremis, immediate emergency treatment with concentrated glucose solution intravenously should be given before any other specific therapy is attempted. The early correction of hypoglycæmia may prolong survival long enough to allow time for other therapeutic measures to be employed successfully.

Patients in acute adrenal failure are particularly susceptible to morphia and barbiturates and these drugs should not be administered before treatment with cortisone is started. As a prophylactic measure penicillin is given twice daily, but for specific infections complicating or causing the illness, the appropriate antibiotic is required.

When the patient is receiving a normal diet and hormone replacement has been reduced to a maintenance level, further management is continued on the lines required for chronic adrenal insufficiency.

Early recognition of the condition, coupled with prompt and effective treatment and the use of suitable antibiotics will save the majority of patients with acute adrenal failure. The use of hydrocortisone intravenously in doubtful cases is always justifiable, since its rapid elimination from the body reduces the possibility of untoward effects, when it is used mistakenly in conditions stimulating acute adrenal failure.

Avoidance of acute adrenal crisis.

Careful attention to the routine management of patients with adrenal insufficiency, and the application of knowledge that stress of any kind will require additional amounts of hormone, will do much to reduce the incidence of acute crisis. It is a wise precaution to warn all patients with impaired adrenal function of the early symptons of adrenal failure and to impress on them the importance of taking more cortisone if they fall ill or require an operation. All patients on continuous cortisone treatment, whether for therapeutic purposes or as replacement therapy in Addison's disease, must realise the danger of abrupt termination of treatment, and understand that in the former the drug should only be stopped slowly and under medical supervision. The printed card (figure 1) which is given to these patients embodies this information, and they are urged to carry it with them.

Fig. 1

IMPORTANT

MEDICAL CARD FOR PATIENTS WITH IMPAIRED ADRENAL FUNCTION

NAME.....

Medical Condition: 1. Addison's Disease.
2. Hypopituitarism.
3. Total or sub-total Adrenalectomy.
4. Adrenal suppression following prolonged cortisone therapy.

1. You are suffering from the condition described above. You will remain in good health provided you take Cortisone tablets every day and such additional teatment as may be prescribed. You will get your prescription for the tablets from your family doctor. Your dose is .....tablets (.....mg.) of.....daily.

2. It is essential for you to take the treatment regularly. Make sure you have an adequate supply if you go away from home.

3. The dose may have to be increased temporarily if you develop an illness (e.g. a bad cold or bronchitis), have an accident or have to undergo an operation (e.g. for appendicitis). Show this card to your doctor or to the doctor in hospital at the time.

4. If you feel weak or lose your appetite it may mean that you are not taking enough Cortisone. Try taking an extra half tablet twice a day and see if you feel better. If you don't, contact your family doctor at once or get in touch with .....
at..... Telephone No.....

5. You will be given two of these cards. Carry one with you always and keep the other at home.

It should be remembered too that endogenous adrenal function often recovers very slowly after prolonged suppression by therapeutically administered cortisone or corticotrophin, and that the response to stress may be subnormal for several weeks or even months after treatment has ceased. In such cases it is probably unwise to operate without suitable hormone cover until at least three months have elapsed.

When operations are necessary for patients with impaired adrenocortical function they should be properly prepared for surgery and receive post-operative hormone replacement in the same manner as a patient undergoing adrenalectomy. The following scheme of treatment has been found satisfactory for total or subtotal

adrenalectomy, but various modifications may be required to meet the needs of individual patients (table 1).

Finally, the care of patients with impaired adrenal function imposes a duty on the doctor of explaining to them the nature of their disability and the steps necessary to avoid dangerous complications. In few conditions is the patient's knowledge and co-operation more important; ignorance can be fatal.

TABLE I.

DOSAGE OF CORTISONE AND DEOXYCORTONE ACETATE BEFORE AND AFTER ADRENALECTOMY.

Day	Cortisone Acetate	Deoxycortone Acetate
2 d. before operation	50 mg. 6-hourly, i.m.	
1 d. before operation	50 mg. 6-hourly, i.m.	
Day of operation	100 mg. 3 hr. before operation by mouth, 25 mg. 6-hourly, i.m.	10 mg. 3 hr. before and 5 mg. 6 hr. after operation, i.m.
1 d. after operation	25 mg. 6-hourly, by mouth if possible, otherwise i.m.	5 mg. 12-hourly, i.m.
2-5 d. after operation	25 mg. 6-hourly, by mouth if possible, otherwise i.m.	5 mg. i.m.
6-10 d. after operation	12.5 mg. 6-hourly, by mouth	
11 + d. after operation	12.5 mg. b.d., by mouth	

CHRONIC ADRENOCORTICAL INSUFFICIENCY.

Chronic adrenocortical insufficiency is commoner than the acute variety and may be divided into conditions in which the adrenal gland is primarily involved and those in which the adrenal function is impaired as a result of pituitary dysfunction. The following classification serves to indicate the conditions commonly seen in clinical practice.

CHRONIC ADRENOCORTICAL INSUFFICIENCY.

A. PRIMARY ADRENOCORTICAL FAILURE.

1. *Addison's disease.*
   - (a) Tuberculous destruction.
   - (b) Idiopathic atrophy.
   - (c) Infiltration with carcinomatous deposits or amyloid.
   - (d) Failure of adrenal remnant following subtotal adrenalectomy.
2. *Congenital adrenal hyperplasia.*
3. *Adrenal exhaustion.*

B. Secondary Adrenocortical Failure.

1. *Pituitary Destruction.*
   (a) Pituitary tumours.
   (b) Pituitary infarction—Sheehan's syndrome.
   (c) Pituitary atrophy.
2. *Hypophysectomy.*
3. *Pituitary inhibiton.*

Addison's disease.

Primary adrenocortical insufficiency in the form of Addison's disease is so well known that a detailed description of the condition is unnecessary. During the last five years seventeen cases have been investigated and treated (table II). There were six men and eleven women in the series. In three women adrenal insufficiency followed bilateral subtotal adrenalectomy for Cushing's syndrome, as the result of a gradual failure of the adrenal remnant left behind at operation. Excluding the surgical cases, tuberculosis was the probable cause in eight (57.1 per cent.) metastatic cancer in one, and in the remaining five (35.7 per cent.), a presumptive diagnosis of idiopathic adrenal atrophy was made in the absence of supporting evidence of tuberculosis. These figures must, of course, not be taken too literally, for in only one (the patient with cancer), was post-mortem control obtained; the remaining patients in this group are alive and in good health. Of the fourteen non-surgical cases, ten presented with signs and symptoms which pointed directly to their Addison's disease, but in four the presentation was atypical and adrenal insufficiency was only discovered in hospital after their admission for other complaints. Two of these had associated chronic peptic ulcers; one was diagnosed before gastrectomy, but adrenal insufficiency was only recognised in the second following post-operative collapse and ileus. A third patient was admitted to hospital as a suspected case of gastric carcinoma with acanthosis nigricans, while the fourth, a partial case, was diagnosed in a patient with myocardial infarction. Adrenal crises were the presenting features in five patients, in three of whom the condition was acute and the result of stress in the form of infection in one, the injudicious administration of an anæsthetic in another, and the result of pregnancy in the third. In two other patients the crisis was subacute and did not appear to be due to any particular variety of stress. It seems likely that in these two the adrenal glands were the seat of a progressively destructive process which caused a rapid impairment of adrenocortical reserve. This was almost certainly the case in the patient with adrenal metastases and likely in the other patient, a young man of 26, with tuberculous Addison's disease.

Addison's disease may occasionally present in an unusual manner. This was seen in a woman of 46 whose only complaint, apart from weakness, was of severe muscle cramps. Mental symptoms of confusion and persecution were also observed when she was in hospital, but these, like the cramps, subsided quickly when cortisone was given.

TABLE II.

Clinical Features in 17 Cases of Addison's Disease.

Case No.	Sex, Age at Diagnosis	Length of History before Diagnosis	Ætiology	Length of Time under Treatment	Special Features
1	M 46	3 years	T.B.	4 yrs. 7 mths.	Admitted in crisis following tonsillitis.
2	F 39	1 year	T.B.	4 yrs. 6 mths.	
3	F 54	12 years	? atrophy	4 yrs. 4 mths.	Long history of weakness and ill-health before diagnosis. Mild haematemesis on two occasions during cortisone treatment. No peptic ulcer found.
4	M 43	3 years	? atrophy	4 years	Acute crisis following gastroscopy before hormone treatment started.
5	F 64	Uncertain	T.B.	3 yrs. 5 mths	History of dyspepsia, weakness and vomiting for 30 years. No organic cause for dyspepsia discovered.
6	F 38	2 years	T.B.	3 years	Active pul. T.B. treated with chemotherapy and arrested during cortisone replacement.
7	F 46	1 year	? atrophy	2 yrs. 8 mths.	Muscle cramps presenting symptoms. Mental disturbance prominent before cortisone given.
8	F 33	5 years	T.B.	2 yrs. 7 mths.	Two pregnancies with live babies during treatment.
9	M 53	10 weeks	Carcinoma pancreas	3 months	
10	F 69	—	? atrophy	2 yrs. 1 mth.	Diagnosis made following gastroenterostomy for chronic D.U. Post-operative collapse with ileus.
11	M 58	—	T.B.	1 yr. 8 mths.	Addison's disease diagnosed prior to partial gastrectomy for chronic D.U.
12	F 55	—	Adrenal-ectomy	1 yr. 6 mths.	Failure of adrenal remnant 13 mths after adrenalectomy with acute crisis.
13	F 59	—	? atrophy	1 yr. 2 mths.	Mild Addison's disease discovered following admission to hospital for myocardial infarction.
14	F 45	—	Adrenal-ectomy	1 yr. 4 mths.	Failure of adrenal remnant 13 mths after adrenalectomy. Post-operative cortisone discontinued 2 mths before permanent replacement required.
15	F 17	—	Adrenal-ectomy	1 year	Addisonian pigmentation noticed 18 mths after adrenalectomy. Died from basophil carcinoma of pituitary.
16	M 26	9 months	T.B.	6 months	
17	M 35	6 months	T.B.	1 yr. 6 mths.	T.B. hip at age of 17.

Cases 9 and 15 are dead, but all others are well.

Congenital Adrenal Hyperplasia.

A relatively rare, but important variety of adrenal insufficiency occurs in infants with congenital adrenocortical hyperplasia, in which 17-hydroxyprogestrone, a substance with adrogenic effects, is formed instead of hydrocortisone. In these children a variable degree of adrenocortical insufficiency may accur which is manifested by vomiting, diarrhœa, weakness and feeding difficulties. In female infants the abnormal genitalia with hypertrophied clitoris should draw attention to the condition in time for effective treatment to be given, but in males, adrenal failure may supervene before there is appreciable enlargement of the penis to draw attention to the defect. The following case is an example.

"A full-term male infant weighing 6 lb. 5 ozs. (2860 gm.) fed poorly from birth, but was otherwise well for the first four days of life. Thereafter he drank progressively less, became irritable and developed inflammation at the umbilicus. At post-mortem, eleven days after birth, both adrenals were grossly enlarged, and on section showed virilizing hyperplasia."

Adrenal Exhaustion.

Functional adrenocortical exhaustion may occur as the result of prolonged stress, toxæmia and malnutrition. Two such cases have been observed following gastric surgery. The first concerned a female patient of 41 who had a gastrectomy for a large gastric ulcer. The stomach failed to empty for three weeks, during which time she was dependent on intravenous feeding. Towards the end of this period the systolic blood pressure fell to 80 mm. of Hg. and it became increasingly difficult to maintain the serum sodium level. Hydrocortisone intravenously, restored the blood pressure and sodium balance within twenty-four hours. The second patient was a man of 57 with chronic bronchitis, ischæmic heart disease and pyloric obstruction. Following gastrectomy the stomach failed to act and intravenous feeding was continued for three and a half weeks with difficulty. Increasing hypotension, and the appearance of a few patches of buccal pigmentation suggested that he was developing adrenal failure. Cortisone by mouth appeared to initiate gastric emptying and the blood pressure rose to its usual level. Some weeks later cortisone was stopped and he has since shown no further evidence of adrenal insufficiency.

Secondary Adrenocortical Failure.

Pituitary deficiency, from whatever cause, results in a failure of corticotrophin secretion, and as a consequence the adrenal cortex atrophies. The effect of this is rarely as acute or severe as in primary adrenal failure, since in secondary atrophy aldosterone secretion is undisturbed, so that water and salt metabolism is relatively unimpaired. Nevertheless, patients with secondary adrenocortical failure withstand stress poorly and must be protected from its effects in the same way as patients with primary adrenal failure.

Inhibition of the pituitary gland by cortisone and its analogues, or by corticotrophin, is the commonest cause of secondary adrenal insufficiency seen at present, and attention has already been drawn to this. More recently hypo-

physectomy is being performed for metastatic carcinoma of the breast (Edelstyn, Gleadhill, Lyons, Rodgers, Taylor and Welbourn, 1958) and an increasing number of these cases will be seen in the future.

Diagnosis of Chronic Adrenal Insufficiency.

Cases of Addison's disease vary in their severity. In severe cases, diagnosis can usually be made on clinical grounds, which may be confirmed by biochemical tests. In these, the plasma sodium will be low, the plasma potassium high and the blood urea elevated, while the urine will contain large amounts of sodium, despite the hyponatræmia. In such cases, especially if there is vomiting or hypotension, it is dangerous to delay treatment, but subsequently a corticotrophin stimulation test may be carried out to differentiate a salt losing nephritis from true Addison's disease.

In less severe or incomplete cases the diagnosis may be less obvious, for the electrolytes are usually within normal limits. Here diagnosis depends on indirect tests to measure metabolic changes secondary to the adrenal insufficiency, and on direct measurement of the response of the adrenal gland to stimulation by corticotrophin (table III).

TABLE III.

Tests of Adrenocortical Function.

A. INDIRECT

(a) Salt deprivation tests.

(b) Original water load test of Robinson, Power and Kelper superseded by.

(c) Simple water load test of Soffer and Gabrilove.

(d) Eosinophil count and its response to corticotrophin.

B. DIRECT

(a) Measurement of adrenocortical steroids excreted in urine before and after corticotrophin stimulation.

The salt deprivation tests were the first indirect tests of adrenal function to be employed, and consisted in giving the patient a salt-free diet. In Addison's disease there is a characteristic response; sodium is excreted in the urine in excess of the intake, while the blood sodium falls and the blood potassium and urea rises. Such tests are potentially dangerous and are no longer employed, since simpler and much less harmful methods are available.

The water load tests are simple and safe, and are based upon the inadequate water diuresis in Addison's disease. The original Robinson, Power, Kelper test (Robinson, Power and Kelper, 1941) with measurement of blood and urine urea and chloride, has been generally superseded by the simple water load test of Soffer and Gabrilove (Soffer and Gabrilove, 1952). This consists in the administration of 1500 ml. of water by mouth over 15-20 minutes in the fasting state, followed by the collection of urine over a five-hour period. In Addison's disease the volume does not exceed 800 ml. or 55 per cent. of the ingested load. A delayed or impaired diuresis may occur in conditions other than adrenal insufficiency, but it is seldom

improved by cortisone, except when caused by primary or secondary adrenocortical insufficiency. In cases where a poor diuresis is observed it is customary to repeat the test after the adminstration of 50-100 mg. of cortisone by mouth.

The Eosinophil Count.

In adrenal failure the eosinophil count in the blood rises, and an approximate estimation of the adrenocortical reserve may be obtained by observing the fall in the circulating eosinophils after stimulation with corticotrophin. In patients with intact adrenals the fall exceeds 50 per cent. of the initial figure within four hours of injecting 25 mg. of corticotrophin. Thorn (Thorn, Forsham, Prunty and Hills, 1948) was the first to employ this test, which has been extensively used in America. It has not been frequently used here, as the more specific and reliable measurement of adrenal response to injected corticotrophin has been preferred.

Direct measurement of adrenocortical function.

As already mentioned, the adrenal steroids are metabolised in the tissues and excreted in the urine in conjugated form. The measurement of these substances in the urine, therefore, provides a useful method of estimating adrenocortical secretory capacity by comparing the changes in the urine before and after the injection of corticotrophin. In Addison's disease the characteristic finding is a diminished or absent excretion of glucocorticoids (differing fractions of which may be measured as "reducing steroids," 17 hydroxycorticoids, or 17 ketogenic steroids) and androgens which are measured as 17 ketosteroids. In severe adrenocortical insufficency there is no rise in the excretion of these substances following stimulation with corticotrophin, while in milder cases the increase remains below the accepted normal response. A single estimation of the resting 17 ketosteroid output is often of value in confirming impaired adrenocortical function in a suspected case of Addison's disease, but the finding of a normal level renders the diagnosis untenable.

The test which is now used routinely to assess adrenocortical function in the biochemical laboratory of the Royal Victoria Hospital measures the 17 ketosteroid and 17 ketogenic steroids (Norymberski, Stubbs and West, 1953) before and after corticotrophin stimulation (Prunty, 1956). Six successive twenty - four hour collections of urine are made, and from the third to the sixth day, twenty units of corticotrophin gel are injected intramuscularly twice daily. The first two days gives the basal excretion, while the remaining four days show the maximum response to stimulation.

This test is also of value in the diagnosis of secondary adrenocortical insufficiency. The control levels of 17 ketosteroids and 17 ketogenic steroids are usually well below normal in this group, but the adrenal cortex is capable of responding to corticotrophin so that the stimulated excretion will show a rise which may be delayed and smaller than normal, depending on the duration of pituitary failure. Thus the response to injected corticotrophin is a useful method of distinguishing between primary and secondary adrenocortical failure.

Radiology is generally of little help in the diagnosis of adrenocortical insufficiency. Calcification of the adrenals may be seen in cases of tuberculous destruction of the adrenals, but is not commonly found, and was seen only once in the eight tuberculous cases in this series. On the other hand, extensive calcification of the adrenal glands may be seen without clinical or biochemical evidence of adrenocortical failure. Adrenal calcification was a feature in a 29-year-old man who was referred for investigation after his admission to a surgical ward for abdominal pain. On clinical examination there was nothing to suggest Addison's disease and the ketogenic steroid output was normal in both the resting and stimulated phase. Nevertheless, a strong possibility of tuberculosis of the adrenal glands existed in his case, for calcified mesenteric glands were also present, and treatment with streptomycin and P.A.S. was advised and carried out.

Treatment of Chronic Adrenocortical Insufficiency.

Since the introduction of cortisone, the treatment of chronic adrenocortical insufficiency has been placed on a rational basis and patients can now be maintained in a satisfactory state of health. Before 1949, patients with Addison's disease led a precarious existence, and even though they were treated with deoxycortone acetate (DCA) they were subject to hypoglycæmic attacks, while any stress was liable to end in a crisis which was often fatal. When cortisone became available it was soon clear that this substance exerted profound effect; hypoglycæmia was abolished and the patient's increased energy, well-being and gain in weight, with ability to meet stressful situations clearly showed its usefulness.

It has been shown (Beck and Montgomery, 1956) that cortisone alone will maintain patients with Addison's disease in good health. Further experience has confirmed this view, and despite the relatively limited effects that cortisone has on electrolyte metabolism, it seems clear from the practical viewpoint that cortisone can maintain these patients in electrolyte balance and keep the blood pressure at a satisfactory level. Usually 25 or 37.5 mg. of cortisone daily is sufficient. When the larger dose is given, 25 mg. may be taken in a single dose in the morning, for in man adrenocortical activity is then at its peak. With treatment the patient improves remarkably, gradually losing the pigmentation and gaining in weight and vigour. Failure to reduce pigmentation suggests that the dose of cortisone is too low.

Excessive loss of salt may be found in rare cases of Addison's disease or result from excessive sweating in those engaged in heavy work or exposed to high environmental temperatures. Such patients may require additional salt or a compound like 9-α-fluoro-hydrocortisone (0.5 mg.) may be effectively combined with the daily cortisone. Deoxycortone acetate may also be used, either in the form of a depot injection of the trimethylacetate microsuspension, 50 mg. every three or four weeks or in the form of linquets (1 mg.) for buccal absorption.

Of the seventeen cases of Addison's disease in this series cortisone alone has been the method of treatment adopted in all but one of the patients. The exception concerned a farmer who noticed weakness and nausea during vigorous haymaking

in the summer of 1955. This was corrected by additional salt by mouth. Subsequently he was tried on prednisone by mouth, following which he developed evidence of salt depletion and hypotension and required deoxycortone trimethylacetate crystules every four weeks. Since then his condition has remained so good that this treatment has not been altered.

In secondary adrenocortical insufficiency cortisone replacement therapy is given in exactly the same way as described for Addison's disease. On the whole, the management of these patients is easier, for they rarely become salt depleted.

Reactivation of the tuberculous process is a rare complication in patients with Addison's disease who are receiving cortisone replacement in the doses usually employed. It did, however, take place in one of the T.B. cases in this series, who became sputum positive about two years after cortisone was started. Whether this was the result of cortisone treatment or not is difficult to say, but the pulmonary condition responded satisfactorily to chemotherapy without alteration in the dose of cortisone. A case too can be made for a course of treatment with streptomycin for all new tuberculous cases of Addison's disease. This may limit further destruction of the adrenal glands and may also lower the risk of reactivation or spread in other parts of the body when cortisone is given. This policy has been adopted with new cases when practical.

Summary.

An account is given of the varying manifestations of adrenocortical insufficiency and the methods employed in diagnosis and treatment are described. Death from adrenocortical failure, once inevitable, is now a tragedy, and can be largely prevented by the application of knowledge gained in recent years. The important fact emerges that while cortisone has introduced a new therapeutic era, its correct use must be thoroughly understood and mastered if patients are to obtain its benefits and not succumb to its dangers or misuse.

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REVIEW

APPLIED PHYSIOLOGY OF THE EYE. By H. Willoughby Lyle, M.D., F.R.C.S., assisted by T. Keith Lyle, C.B.E., M.A., M.D., M.R.C.P., F.R.C.S. (Pp. vii + 341; figs. 32. 45s.) London: Baillière, Tindall & Cox, 1958.

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Coming from the pens of two such well-known ophthalmic surgeons, it is only to be expected that it would be a book of a high order. This it undoubtedly is, and it should be a great help to those who are working for higher examinations in ophthalmology, such as D.O., F.R.C.S. in ophthalmology, etc.

The optic nerve with the retina is an outgrowth of the brain, and is therefore an integral part of the central nervous system. The complicated cerebral connections are clearly described and show how certain diseases of the C.N.S. produce their signs and symptoms. Again the marked changes which occur in the cardio-vascular system frequently show their effect in the retinal capillaries and prove a valuable aid in diagnosis and prognosis.

Finally, the chapters giving up-to-date, concise knowledge of the endocrine system, vitamins, diabetes mellitus and allergy are well written, and give the book a good balance. J. R. W.

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MY object today is to try, I am sure very inadequately, to tell you something of the life of that illustrious fellow countryman of mine, William Smellie, the Master of British Midwifery, who was born in Lanark in the year 1697.

Smellie lived during the reigns of four sovereigns—Queen Anne and the first three Hanoverian Georges. He saw the union of the Scottish and English Parliaments in 1707, and lived through the two Jacobite rebellions, the '17 and the '45. Lord Lovat, the last person to be beheaded in public, was executed in Tower Hill in 1747 for his part in the battle of Culloden. It is recorded by Smellie that a patient who had an anencephalic fœtus blamed her misfortune on the fact that her husband attended the execution!

Addison, Steele and Defoe were dead. Pope and Swift were old men. Samuel Johnson was a contemporary, but did not achieve fame until late in Smellie's life. Hogarth, one of the earliest English painters and the first cartoon artist, was at work in London. Gainsborough and Reynolds were also climbing to fame. Handel and Bach were both revolutionising the world of music, and it is known that Smellie knew of Handel's work, owned an organ and a number of flutes.

William Smellie attended the Grammar School in Lanark and subsequently entered the Medical profession by becoming an apprentice to an established practitioner. There is a little doubt concerning the identity of his Principal, but the evidence suggests that it was Mr. Gordon of Glasgow to whom he articled himself. Ultimately, in 1720, at the age of 23, he settled in Lanark in practice on his own account. In 1724 he married Eupham Borland. There were no children.

For nineteen years he practised in Lanark, and during this time he improved himself by reading books which were borrowed from Dr. Cullen's library in Hamilton, from Glasgow and from Edinburgh. It is known that he even sent to London to obtain books not available elsewhere.

He attended a number of difficult midwifery cases at the request of various midwives, and from that early date he developed the remarkable habit of writing down in detail all the relevant facts. These case records can be read today and should be read by all of us, not once, but many times.

> "**Case 382.** Primipara: the Shoulder presented; Hæmorrhage; the Fœtus delivered by tearing down the Body with the Crotchet.
>
> 1722—The woman was young and strong. This was her first child; the membranes broke the day before; she had strong pains all night. When I arrived in the morning I found the shoulder forced down to the lower part of the pelvis."

He then goes on to describe his attempts to perform internal version, first with his left and then with his right hand:

> "Till I was at last excessively fatigued and obliged to desist. I rested about half an hour, considering what I should do next, and waiting until I should recover the use of my arms. By these efforts, and the exertion of great force, a considerable flooding was brought on; and this alarmed me not a little, especially as it was one of my first cases, and I had not yet attained that calm, steady and deliberate method of proceeding, which is to be acquired only by practice and experience. I had over-fatigued myself, from a false ambition that inspires the generality of young practitioners, to perform their operations in the most expeditious manner."

Ultimately he delivered the patient by using a crotchet.

After nineteen years in Lanark, Smellie decided that he must seek further knowledge. Therefore, in 1739, he set off for London to pursue this aim. He was particularly anxious to learn more about obstetrical forceps, his interest having been stimulated by papers written by Butters, Chapman and Giffard. He had tried out Duse's forceps and found them "ill contrived," but he seemed to realise their potentialities. It has been suggested that he made the journey to London with his friend Tobias Smollett, and that the journey has been recorded in "Roderick Random," but possibly this is speculation.

His opinion of London may be best put in his own words, "Here I saw nothing was to be learnt," so he journeyed on to Paris and received instruction from the Gregoires—father and son. It was in Paris that he saw a "phantom" used for teaching, and developed the idea of a district midwifery service to be used for the purpose of training students.

Returning to London, he bought a house in the Mall, and within a short time he had a flourishing practice, and decided to become a teacher. First of all he prepared a series of lectures and "by dint of uncommon labour and application" he fashioned a very realistic "phantom" and several manikin dolls, much superior to those used by Gregoire.

He issued a seven-page brochure with a syllabus of the lectures and a note of his fees, and as there were no hospital beds, he set up a district midwifery service and let it be known that he, with his students, would attend gratuitously poor women in their homes. He arranged for students to pay a six shillings fee into a fund out of which he gave charity to necessitous patients. Sometimes his students caused excitement in the street, much in he same way as our Flying Squad does today.

He describes a case of prolapsed arm in a lane off St. Giles, which he attended with twenty-eight students. "Such a number going in had so alarmed the lane that a great mob assembled and began to exclaim that we were trying practices. Some of the women also told us that the parish officers were sent for, who at that time were glad of showing their authority. On these accounts I was obliged to deliver the woman in a hurry. The child was alive and when this was told the mob and that the woman was also safe, they all dispersed."

Altogether he gave 280 courses of lectures and trained 900 pupils.

In 1745 his merit was recognised by the University of Glasgow, by the conferring of the M.D. degree, and he thereupon abandoned the inferior title of Mr. and became known as Dr. William Smellie.

British Obstetrics Prior to Smellie.

It is at this point that a digression should be made to review the state of British obstetrics at the time Smellie started to teach in London. In 1540 the "Byrth of Mankynde," the first textbook of midwifery written in English was published. This book was largely a translation of a German book called "De Partu Homenis," written by Rosslin. It is not certain whether the English author was Dr. Thomas Raynolde, or the translation was printed by one called Thomas Raynolde, a London printer. Ballantyne of Edinburgh has written an interesting monograph on this publication.

The real renaissance of British obstetrics occurred in the seventeenth century. In 1634 the works of Ambrose Paré were translated, followed in 1651 by William Harvey's celebrated work in Latin entitled "De Generatione Animalium," with one chapter "De Partu," the first original description of labour to appear in British literature. Harvey has been described as the Father of British midwifery, but the description is not really applicable, as he was not an obstetrician so much as a physician.

In 1672 Hugh Chamberlen published "The Accomplished Midwife," an English translation of Francois Mauriceau's textbook, and this book was re-edited many times during the next hundred years. In 1733 Edmund Chapman published a short account of the Practice of Midwifery, describing fifty cases. He was the first person to make public a description of the forceps used by the Chamberlens, although at the time of his publication the forceps were no longer a secret, and had been used by Giffard and Drinkwater, as well as others.

In 1741 Fielding Ould of Dublin published a treatise on the practice of Midwifery. Fielding Ould, a great obstetrician, was the second Master of the Rotunda, following Bartholomew Mosse, who was more a philanthropist than an obstetrician. Mosse founded the hospital in the year 1745. Ould's grandfather fought in the Battle of the Boyne, and his father was a captain in the British Army. In the year he was made Master of the Rotunda he received a Knighthood, and this prompted the jingle :—

Sir Fielding Ould is make a Knight,
He should have been a lord by right,
For then each lady's prayer would be,
O Lord, good Lord, deliver me.

Ould was the first obstetrician to realise from his observations that the head entered the pelvis in the oblique diameter and rotated during descent. To aid delivery of the head he advocated pressing the coccyx back per rectum by means of the thumb, followed by the application of traction per rectum on the baby's jaw by means of the forefinger.

Smellie's Writings.

Smellie's first publication was in 1742, when he published "A Course of Lectures upon Midwifery." His most celebrated work, however, was "A Treatise on the Theory and Practice of Midwifery," which appeared in three volumes.

The first volume, published in 1752, contains in the introduction a review of the literature from Hippocrates onwards, followed by the subjects normally dealt with in a standard textbook. In 1754 he next published his famous "Set of Anatomical Tables," containing thirty-nine plates, of which Rymsdyk (a Dutch artist) drew twenty-six and Smellie, assisted by a pupil called Camper, drew the remaining thirteen. Of the original publication, not more than one hundred copies were made. In the same year, 1754, he also published Volume II of his treatise. This consisted of a collection of cases illustrating the points made in Volume I. In 1774, after Smellie's death, Volume III appeared, containing again a collection of cases, all of a so-called "preternatural" character.

The three volumes of his treatise were edited by his friend, Tobias Smollett, the author, and undoubtedly to him we owe a debt for the delightful style throughout. A case of extreme interest to this Society is Case 433; the Cæsarean operation performed with success by a midwife described by Mr. Duncan Stewart, surgeon in Dungannon in the County of Tyrone, Ireland.

> "Alice O'Neale, aged about 33 years, wife to a poor farmer near Charlemont, and mother to several children, in January 1738-39 was taken in labour, but could not be delivered of her child by several women who attempted it. She remained in this condition twelve days; the child was thought to be dead after the third day.
>
> Mary Smalley, an illiterate woman but eminent among the common people for extracting dead births, being then called, tried also to deliver her in the common way and, her attempts not succeeding, performed the Cæsarean operation by cutting with a razor, first the containing parts of the abdomen, and then the uterus, at the aperture of which she took out the child and secundines. The upper part of the incision was an inch higher and to one side of the navel and was continued downwards in the middle betwixt the right osilium and the linea alba. She held the lips of the wound together with her hand till one went a mile and returned with silk and the common needles which tailors use. With them she joined the lips in the manner of the stitch employed ordinarily for the hare lip, and dressed the wound with whites of egg, as she told me some days after when, led by curiosity, I visited the poor woman who had undergone the operation. The cure was completed with salves of the midwife's own compounding.
>
> In about twenty-seven days the patient was able to walk a mile on foot, and came to me in a farmer's house, where she showed me the wound covered with a cicatrice; but she complained of her belly hanging outwards on the right side, where I observed a tumour as large as a child's head, and she was distressed with a fluor albus, for which I gave her some medicines and advised her to drink the vulnerary plants and to support her belly with a bandage. The patient has enjoyed very good health ever since, manages her family affairs, and has frequently walked to market in this town, which is six miles distant from her own home."

Advances in Obstetrics by Smellie.

Most of us remember Smellie from our student days, when we were taught the Mauiceau-Smellie-Veit method of delivering the aftercoming head. The method was in fact first described by Mauiceau in 1681, and again by Giffard in 1734. Smellie

advocated the method in his treatise in 1752 (Veit rediscovered it one hundred years later in the nineteenth century, and really should not have his name attached to the manœuvre at all). In actual fact, Smellie is much more to be commended, according to our present standard of learning, for using the forceps on the aftercoming head, and indeed for inventing a long forceps with both a pelvic and cephalic curve specially for the purpose.

First of all, we must give Smellie credit for breaking away from folklore and refusing to believe anything which he could not prove by personal observation—much of which was very painstaking. It had been taught that the fœtus lay as a breech until term, and then when it got hungry it turned round and crawled out. Smellie dismissed all of that and described accurately the mechanisms of labour using his illustrations, so that we cannot improve on his teaching to this very day. He was the first to describe the rachitic pelvis, and taught how to measure the diagonal conjugate diameter with the fingers per vaginam. He taught conservatism in the third stage and recommended light traction on the umbilical cord.

Forceps.

As indicated above, the Chamberlens' secret was out, and Giffard, Chapman and Butters had all written about forceps. Smellie's first forceps were short and straight and were made of boxwood. As he was no mean wood carver, I would like to think that he made them himself. His reason for using wood was that it made less noise, and therefore was less frightening to his patients. He advocated that the forceps should be concealed in side pockets and then applied secretly under the cover of the sheet, which should be pinned to the chest of the operator. The use of these forceps led William Douglas, one of his harshest critics, to call him "A Wooden Operator." In actual fact, he only used wooden forceps on three cases, and then discarded them in favour of metal ones covered with leather.

In 1754 he mentions forceps curved to one side, i.e., with a pelvic as well as a cephalic curve, but it is uncertain whether he, or one called Pugh, or a Frenchman called Levret, actually invented the pelvic curve. It is generally agreed, however, that Smellie invented the Engish lock, which is universally used today.

By accident he discovered that the head could be rotated from the posterior position to the anterior one by means of the forceps. In Case 258 he describes how in a patient with an R.O.P. position the forceps came off on three occasions :

> "While I paused a little, considering what method I should take, I luckily thought of trying to raise the head with the forceps, and turn the forehead to the left side of the brim of the pelvis where it was widest, an expedient which I immediately executed with greater ease than I expected. I then brought down the vertex to the right ischium, turned it below the pubes and the forehead into the hollow of the sacrum; and safely delivered the head, by pulling it up from the perineum and over the pubes. This method succeeding so well, gave me great joy, and was the first hint, in consequence of which I deviated from the common method of pulling forcibly along and fixing the forceps at random on the head; my eyes were now opened to a new field of improvement on the method of using the forceps in this position, as well as in all others that happen when the head presents."

Smellie's long forceps were invented for use on the aftercoming head, and not for the high forceps operation which he condemned. In fact, he laid down rules for the use of forceps which are almost identical to those being taught today. Unfortunately, after his death forceps were used indiscriminately and with such bad results that they fell into disrepute, and there sprang up a generation of obstetricians who were ultra-conservative. It was this conservatism that led to the Royal tragedy of 1817, when the Princess Charlotte of Wales, after labouring for twenty-four hours in the second stage, with the head showing for six hours, was delivered spontaneously of a stillborn child and died of postpartum hæmorrhage. That child had he lived would have become the King of England.

Croft, the obstetrician in charge, was following the conservative teaching of his father-in-law, Denham, who published his famous book in 1788, in which he advocated that the forceps should not be applied until the head has been on the perineum for six hours. Croft, however, was so upset at his misfortune that he wrote to a friend: "May God grant that you nor any connected with you may suffer what I do at this moment." Three months after the tragedy he shot himself.

Smellie's Critics.

As you well know, it was commonplace in those times for criticism, legitimate or otherwise, to find an outlet in the form of a pamphlet. The laws of libel and slander were not at all as they are today. It was, therefore, not surprising that William Smellie, as he became more famous, received considerable abuse at the hands of several critics.

The first of these, William Douglas, published two scurrilous letters, addressed to Dr. "Smelle," in the year 1748. "Such monstrous hands are, like wooden forceps, fit only to hold horses by the nose whilst they are shod by the farrier, or stretch boots in Cranbourne Alley."

The second attack came from Mrs. Nihell, one of the most famous midwives of that time. She had trained for two years at the Hotel Dieu, and, married to a surgeon apothecary had set up practice in the Haymarket. In 1760 she attacked all man-midwives: "That multitude of disciples of Dr. Smellie, trained up at the feet of his artificial doll, or in short, those self-constituted man-midwives made out of broken barbers, tailors, or even pork butchers, for I know myself one of this last trade who, after passing his life in stuffing sausages, is turned an intrepid physician and man-midwife. See the whole pack open in full cry: to arms! to arms! is the word, and what are those arms by which they maintain themselves but those instruments, those weapons of death."

In referring to Smellie, she said: "The delicate fist of a great horse godmother of a he-midwife, however softened his figure might be by his pocket-nightgown being of flowered calico, or his cap of office tied with pink and silver ribbon."

The next attack came from Philip Thicknesse, who wrote "A letter to a young lady on her marriage." Later he wrote in "Man Midwifery Analysed" that Smellie's treatise was "the most bawdy, indecent and shameful book which the Press ever brought into the world."

Fortunately, in a pamphlet written by an actor called Foote, Thicknesse himself was informed that "He had the stupidity of an owl, the vulgarity of a blackguard, the obdurate heart of an assassin, and the cowardice of a dunghill cock."

Lastly, amongst his critics was Dr. John Burton of York, who gained immortality as the original Dr. Slop in "Tristram Shandy" (the famous novel written by Laurence Sterne). In 1753 Burton published a letter to William Smellie, M.D., in which he harshly criticised Smellie's treatise. The letter ran to 233 pages and dealt with every aspect of the treatise. He caught Smellie out with lithopædus senonensis—a petrified child—which Smellie thought was an ancient author, but in the main his criticism can be discarded by posterity.

Smellie did not refute any of the attacks made upon him, but several of his pupils published letters in his defence.

Smellie's Death and Posthumous Appreciation.

Smellie left London in the year 1759, at the age of 62, after nineteen years of strenuous professional life. His practice was taken over by Dr. John Hanna, a pupil who had married a niece of Mrs. Smellie. Returning to Lanark, he settled in retirement in a small estate which was called Smellum, and there, on the 5th March, 1763, he died. He was buried in the kirk yard of St. Kentigren in his parents' grave. In his Will he left books, his writing desk and his "smoaking little chair" to the School of Lanark.

Appreciation of William Smellie was voiced in France and elsewhere abroad, long before it came to this country. At his death very little appeared in the Press, but at that time it was not usual for obituary notices to be published in Medical journals. Furthermore, he had been away from London for a number of years, lost and to some extent forgotten, in the remoteness of Scotland. After all, he had not been on the staff of a lying-in hospital, nor had he any reputation as a society obstetrician.

Slowly, however, his greatness began to become evident. In 1788 Professor Thomas Young of Edinburgh edited Smellie's treatise, and added a fortieth plate to the anatomical tables. At the end of the century, the great Jean Louis Baudelocque classified Smellie along with Mauriceau and Levret as three obstetricians who had exerted vital influences on the progress of midwifery. He put this down to the fact that they had all written "out of the very bosom of practice."

In 1876 the New Sydenan Society chose Dr. Alfred McClintock of Dublin to act as Editor of their new edition of Smellie's works. This publication, occurring as it does half-way between Smellie's time and the present, makes most interesting reading, and I commend it to you.

It commences:

> "As a teacher, author and practitioner, there is no British obstetrician—certainly none of the eighteenth century—who deserves so high a place in our estimation as William Smellie. Nay more, under whichever of these several aspects we may regard him, he scarce has an equal Whilst of all the men, British and foreign, who have most

largely contributed to the advance of sound obstetric knowledge, Smellie may justly stand in the foremost rank. No accoucheur, ancient or modern, unfolded so many of the principles of true obstetric science and in his practice so consistently acted up to them."

In 1894 Professor John Glaister published his great biography, and it is to him that I owe much for the contents of this paper.

In 1927 Spencer published "The History of British Midwifery from 1650-1800," and he was the first to speak of Smellie as the "Master of British Midwifery."

He wrote :

"In concluding this brief account of the greatest of British obstetricians, one must make a tribute of admiration for his great achievements in circumstances which would have discouraged a less heroic man. Without powerful friends to help him, without the advantages of a hospital clinic, but attending and teaching in the homes of the poor, by sheer devotion to his art he raised himself to the foremost position in his profession, which he enriched with many original contributions.

Since then Professor R. W. Johnstone of Edinburgh has brought Smellie's biography up to date by publishing, in 1951, "Wm. Smellie—The Master of British Midwifery."

Professor Miles Phillips of Sheffield, who is one of the most devoted admirers of Smellie in this generation, wrote :

"It is my belief that no other man ever advanced in his own lifetime knowledge of the theory and practice of midwifery to an extent in any way comparable with that achieved by Smellie. The light he shed on the mechanism Nature employed in the passage of the fœtus through the pelvis, and on the wise management of labour, both natural and abnormal, and on the training of midwives and students, was widely recognized in his own time in other countries as well as this. Several of those other countries, including Holland, Denmark and Sweden, have continued to use the methods he advocated, and to extend their practice on the same principles."

In conclusion, may I quote Shakespeare, to sum up all that was good in William Smellie :

"Some men never seem to grow old. Always active in thought, always ready to adopt new ideas, they are never chargeable with fogyism. Satisfied yet ever dissatisfied, settled yet ever unsettled, they always enjoy the best of what is and are the first to find the best of what will be."

I am very much indebted to Mr. Alistair Gunn for much helpful advice on the preparation of this address.

Post-Traumatic Epilepsy and Brain-Stem Signs

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Based on communication given to the Association of British Neurologists at Belfast, April, 1957

BRAIN-STEM lesions following head injuries have been reported by Hutchinson (1876), Bollinger (1891), Duret (1920), Symonds (1932) and Kremer, Russell and Smyth (1947). These authors do not mention associated post-traumatic epilepsy. I have seen in recent years three young men who, following a minor head injury, had attacks of altered consciousness, in two frankly epileptic, and on examination there were signs suggesting a lesion in the brain-stem.

Case 1, J. G., aged 23, sustained a head injury on 3/12/50, when he slipped in the snow and fell on the back of his head. He was dazed but there was no loss of consciousness and he managed to find his way home. Three days later he was drinking coffee when his hands began to shake and the coffee spilled. He felt dizzy, got up from the table, but remembers nothing more until the following morning. He was later told by a friend that he staggered like a drunk man, was very quarrelsome and his speech was slurred. Since then he had several attacks of altered consciousness with ataxia and diplopia. On 15/3/51 he was ataxic and dysarthric following one of the attacks. He complained of double vision but not of headache. There was slight clouding of consciousness, horizontal nystagmus, more marked on left lateral gaze and also present on upward gaze, the right pupil was greater than the left and reacted poorly to light and convergence was weak. There was a slight right-sided facial weakness. The abdominal reflexes were difficult to obtain, but the tendon reflexes were normal and there were no abnormal sensory findings. An EEG. was abnormal with bilaterally synchronous outbursts of high amplitude slow activity. (Fig. 1). He was admitted to the ward where I witnessed a major fit which appeared to have no focal onset. In hospital his mental state improved and he became clearly orientated although there was amnesia for the day prior to and the day of admission. The physical signs cleared up except that the pupils remained unequal and there was slight nystagmus. X-rays of skull were normal, blood sugar normal, C.S.F. normal. He was put on phenobarbitone gr. 1 twice daily. He attended once as an out-patient six months later and stated that he had had further attacks of altered consciousness, but the phenobarbitone had been omitted. There had been no further convulsions. Since then I have been unable to contact the patient.

Case 2, F. G., aged 16, had always had slight visual difficulties and a latent squint. On 17/7/56 he was climbing a tree when the branch broke and he fell

about three feet on to the back of his head. According to his friends he got up but promptly fell unconscious. He was admitted to the Tyrone County Hospital and was unconscious for two hours. There was a hæmatoma in the occipital region but no fracture of the skull. The blood pressure was 140/60, pulse varied from 90-96 and he was discharged two weeks after admission to hospital.

Since this accident he complained of drowsiness. On 8/9/56, approximately two months later, he was riding his bicycle when he felt a "lightness in the head" and noticed that he was steering to the left, and the next thing he remembers was finding himself in the ditch at the side of the road. He was re-admitted to the Tyrone County Hospital and I was asked to see him about a fortnight later on account of drowsiness—he would tend to go to sleep at any time, even over meals.

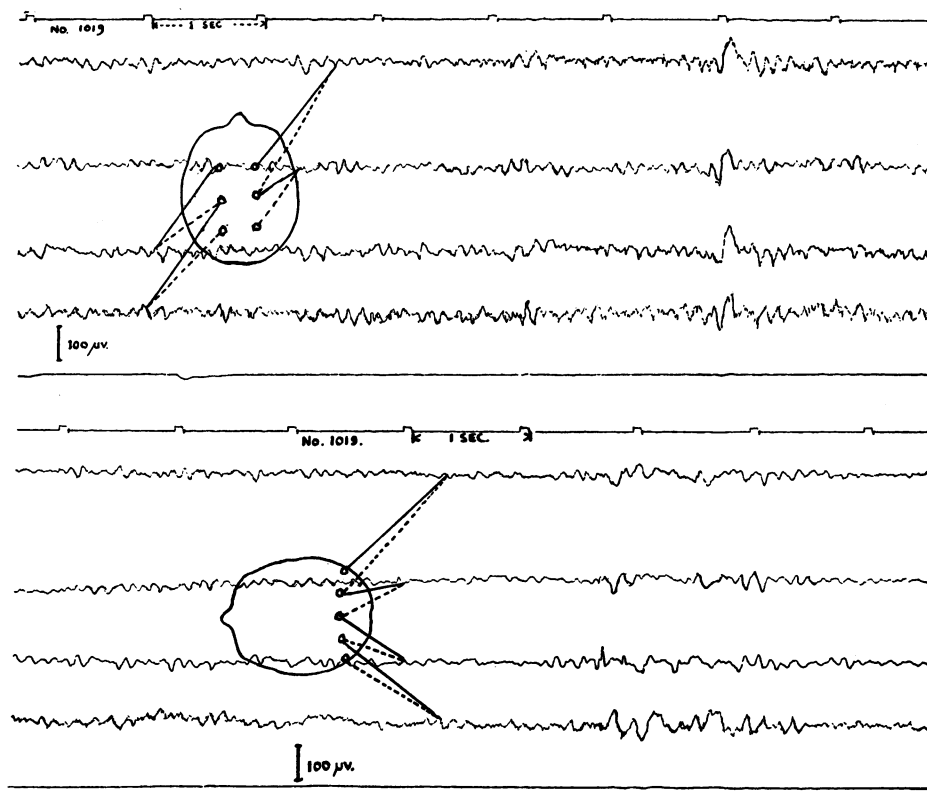

Fig. 1 (see text)

When I saw him he complained of a right frontal headache and double vision. On examination at that time he showed very gross nystagmus. On left lateral gaze the nystagmus was more marked in the left eye and there was diplopia to the left due to a paresis of the left external rectus. Abdominal reflexes were absent, the tendon reflexes were present but depressed and the plantar reflexes flexor. He was transferred to Claremont Street Hospital for observation. On admission there was

monocular nystagmus to right and left and he was observed to be drowsy and to sleep for periods during the day. On 24/9/56, EEG. was normal. He slowly improved and was transferred to the convalescent hospital on 4/10/56. On 8/10/56, a series of fits was observed by the house-physician and matron. The nature of some of these was doubtful, but at least two were considered to be organic in that he went stiff and turned a blue colour, the corneal reflexes were absent and he was incontinent of urine. He was transferred back to Claremont Street Hospital. EEG. was repeated and was again normal, and he slowly improved on anti-convulsants.

Case 3, H. E., aged 17, was mounting his bicycle on 25/10/56, when the gear slipped when his foot was in the toe clip. He fell sideways and knocked his head against a lamp post. There was no loss of consciousness. He re-mounted his bicycle and proceeded to a hall where he played table tennis. When riding his bicycle he was very unsteady and when playing table tennis he played poorly, missing the simplest shots. Later he felt dizzy and went outside the hall where he vomited and lost consciousness. With assistance, however, he was able to walk home and was admitted to the observation ward of the Royal Victoria Hospital. He did not regain full consciousness for about one hour. The next morning he felt well and the X-ray skull showed no fracture. He was allowed to go home, but the following day about 9.30 p.m. he complained of headache and double vision and lost consciousness again. According to his mother he looked dazed and his eyes had a staring "far-away" look. He plucked at his mouth and tongue in a strange manner. He was re-admitted to hospital and at that time there was diplopia to the left, horizontal nystagmus on right and left lateral gaze, impairment of light touch and pinprick sensation over the left side of the face and absent knee and ankle jerks. The following day he felt well, free from headache, fully orientated and he remained quite well until 7 p.m. on 5/11/56, nine days following his re-admission. He complained of a headache and when the ward sister went to see him he was scratching his head violently. He did not appear to know where he was and had a blank expression. He started to paw the screen about his bed, and heave himself up and down in the bed, pulling the bedclothes over his head. When I saw him for the first time the next day he was mentally normal again. He was transferred to the Neurological department on 9/11/56. No further attacks of altered consciousness or peculiar behaviour were witnessed. He still complained of diplopia on left lateral gaze, which he stated had improved, and there was slight horizontal nystagmus on right and left lateral gaze. He was treated with phenobarbitone gr. $\frac{1}{2}$ twice daily. On 10/11/56, EEG. was normal and he was discharged from hospital on 13/11/56, since when there have been no further attacks of altered consciousness, and at present there are no abnormal physical signs. The nystagmus has cleared up.

Regarding these three men, there was no history of previous fits or family history of epilepsy. It seems unlikely that the head injury was the first fit, as there was an adequate cause for the injury in all three and in only one was consciousness lost. It is of interest that the brain-stem signs largely cleared up when the periods

of altered consciousness ceased or were brought under control. There was no evidence of bradycardia during the periods of altered consciousness.

Other Cases:

Recently I have reviewed the case histories of 386 epileptic patients. There were only a further 8 in whom epilepsy could be attributed with certainty to a head injury. Of these 5 were seen within a matter of weeks of the injury and of these 3 showed signs pointing to brain-stem damage. The signs were slight, nystagmus in one and unequal pupils in the second; in the third, in addition to unequal pupils, the larger pupil did not react to light. This last case was interesting in as much that he had been examined for another reason prior to the accident, and then the pupils were normal and also the EEG. Following the accident, however, the record showed bilateral theta activity. In 6 patients the EEG. showed bilaterally synchronous theta activity without a focus or lateralisation. (Fig. 2). Bilateral

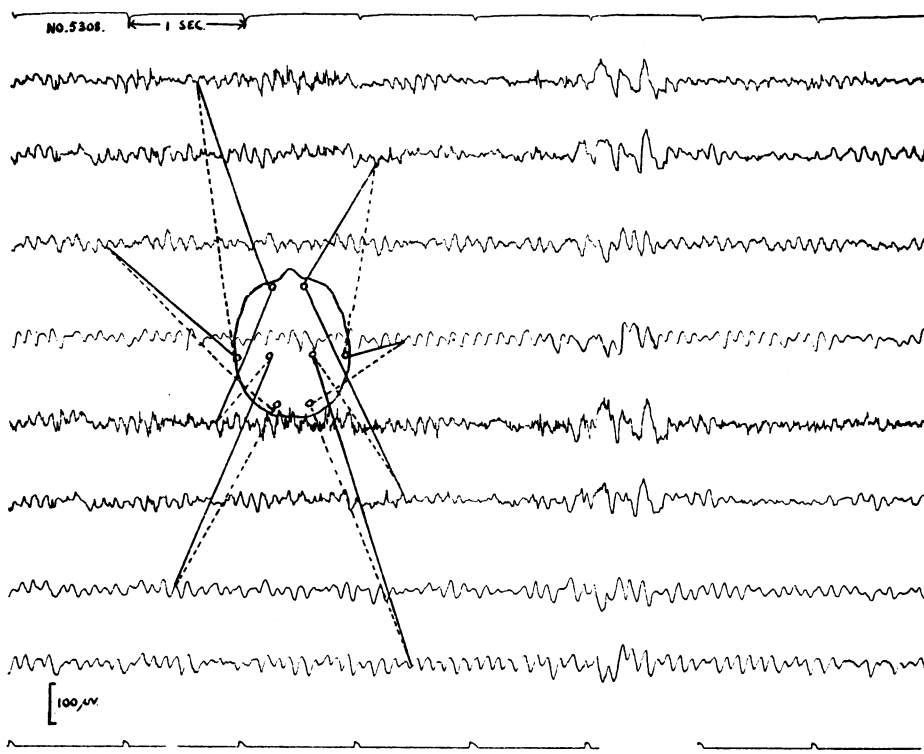

Fig. 2

EEG. carried out on a 20-year-old male one year following a severe head injury, with post-traumatic amnesia of nine days. Major fits began one month following the accident, well controlled by pheno-barbitone. On examination horizontal nystagmus on right and left lateral gaze and also on upward gaze.

paroxysmal activity in the EEG. has frequently been described in post-traumatic epilepsy suggesting a subcortical lesion. Jasper and Penfield (1943) described 3 out of 86 cases of post-traumatic epilepsy with bilaterally synchronous complexes of spike and wave type which suggested that the epilepsy had a deep central origin.

Discussion.

Denny-Brown in 1941 described 6 cases which he called "delayed collapse after head injury." In 3 the collapse was accompanied by loss of consciousness and in 1 a confused state with deviation of the eyes. Four cases had evidence of a sight subarachnoid hæmorrhage and in 4 patients there were signs pointing to brain-stem damage. He attributed the loss of consciousness or collapse to the accompanying slow pulse which in his cases did not fall below 40 and this bradycardia he attributed to a medullary lesion secondary to the trauma. He considered that medullary contusion is a frequent complication of head injuries and that it was not related to increased intracranial pressure.

Following Magoun's classical work on the reticular substance, the loss of consciousness in concussion had been suggested to be due to reversible damage in this area. In a recent paper Foltz and Schmidt (1956) reported their results in experimental concussion in monkeys, in which they observed that in 6 out of 8 experiments no sensory evoked responses appeared in the reticuar formation following concussion. Of the remaining two, a temporary loss of this response appeared in one animal and in the other a delayed but progressive loss was obtained. The prompt loss they explained by direct traumatic neuronal depression in the reticular substance itself. The delayed response they attributed to a pharmacological depression by acetyl choline accumulation as described by Bornstein (1946) and later by Tower and McEachern (1948). However, this excess acetyl choline was not confirmed by Brodie Hughes (1957).

If the lesion in concussion is chiefly in the region of the reticular formation then the brain-stem signs are not difficult to explain and perhaps it would not be stretching the imagination too far to postulate that in some cases, epilepsy following a closed head injury might result from "rebound" in the Sherrington sense (Phillips, 1954)—that is the sudden excessive synchronous discharge of neurones following a period of inhibition in the reticular formation such as was found by Foltz and Schmidt in experimental concussion. There are two objections to this explanation; one is that the fits can be explained by coexisting cortical damage probably in the temporal lobes, and secondly, that other causes of brain-stem damage such as encephalitis, vascular lesions and tumours are not accompanied by epilepsy.

Summary.

The histories of three young men, who had attacks of altered consciousness following a minor head injury, are described. In two major fits were seen and on examination there were signs suggesting a lesion in the brain-stem. There is experimental evidence to suggest that the lesion in concussion is at least partly in the brain-stem reticular substance so that it is not difficult to account for the

brain-stem signs. Reasons are given for and against the possibility that the post-traumatic epilepsy is in some way also related to a lesion in this area.

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REVIEW

STEDMAN'S MEDICAL DICTIONARY. Edited by N. B. Taylor, V.D., M.D., F.R.S.C., F.R.C.S.(Edin.), F.R.C.P.(Cam.), M.R.C.S.(Lond.). Nineteenth Revised Edition. (Pp. xvi + 1,656. 88s.) London: Baillière, Tindall & Cox, 1957.

THIS is probably the best and most consistently revised medical dictionary available. It is, however, of American origin, and it is a more valuable guide to those reading medical literature, and especially to those confronted with unfamiliar American usage, than to those wishing to have a reliable guide to spelling for the preparation of articles for British journals.

The dictionary starts with a useful and clearly presented section on medical etymology.

A feature is the number of anatomical and pharmaceutical tables. The anatomical tables, numbering 52, list ampullæ, arches, areas, etc. It is a pity the only changing feature of descriptive anatomy is nomenclature, and the student will here find the innominate artery but not the brachiocephalic.

Some definition of terms in common usage are short and provide no clear guide as to accepted or debatable usage. Thus "stillbirth—the delivery of a dead fetus" is scarcely worthy of a medical dictionary and "live birth" and "fœtal death" so carefully defined by the World Health Organisation are omitted. Nevertheless, the medical man will rarely seek without finding some enlightenment, and the book is, after all, a dictionary and not an encyclopædia.

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The Leucocyte Count in Weil's Disease

By C. Cotton Kennedy, M.A., D.M.(Oxon)
Consultant Clinical Pathologist, Belfast City Hospital

Many authors have stressed the presence of a polymorphonuclear leucocytosis in Weil's disease (Leptospirosis icterohæmorrhagica), often as one of the distinguishing features from catarrhal jaundice . . . Alston and others (1935), stated that "a polymorph leucocytosis . . . is constantly found in leptospiral jaundice, and is in contrast with a tendency to leucopenia, and an absolute or relative lymphocytosis which have been observed in epidemic catarrhal jaundice by investigators abroad and in the country." McKeon and Brown (1936) and Shapiro and Smith (1956) also emphasized this point. Davidson and Smith (1936) included a moderate leucocytosis among the signs to be expected in the first seven days of illness. Although total leucocyte counts were not given, Swan and McKeon (1938) stated that a marked polymorphonuclear leucocytosis was present in fourteen of their thirty cases. Walch-Sorgdrager (1939) noted that a leucocytosis with an eosinophilia had been reported; also that 2-2.5 per cent. myelocytes or meta-myelocytes had repeatedly been observed. Sladden (1939) considered an increase in neutrophils and monocytes characteristic, with an alteration in the lymphocyte : monocyte ratio in favour of the latter. Johnston (1950), in a review of twenty-one cases of classical Weil's disease in Australia, stated, "Early in the disease neutrophile leucocytosis was a feature." Robertson and Broom (1951) recorded that the leucocyte count is normal in the early part of the illness, but that a polymorphonuclear leucocytosis occurs later. Robertson (1952) considered that a normal white cell count was quite usual in the absence of jaundice, but when jaundice had developed "a polymorph leucocytosis . . . is probably constant." Whitby and Britton (1953) held that a polymorphonuclear leucocytosis occurred during the febrile period. Jeghers (1953) pointed out that there was a leucocytosis in the septicæmic stage, whereas there was often a normal white cell count in leptospiral meningitis.

On the other hand, many observers have noticed that the white cell count remained within normal limits throughout the illness. In Flanders in the First World War, Stokes, Ryle and Tytler (1917) carried out daily blood counts in four early cases, the counts being made on five successive days from the fifth to ninth day of illness. They were unable to demonstrate anæmia or leucocytosis. Buzzard and Wylie (1947) described five cases of meningitis leptospirosa; they noted that the "high serum bilirubin and blood urea and the neutrophil leucocytosis of the textbook Weil's disease were absent." Gauld (1947), who saw thirteen cases of Weil's disease in a military hospital in the Isle of Wight, observed that several had normal white cell counts. In a review of 214 cases in the north-east of Scotland, Smith (1949) reported that in the blood picture there was often little change from normal. Beeson and Hankey (1952) noted in twenty-four cases of

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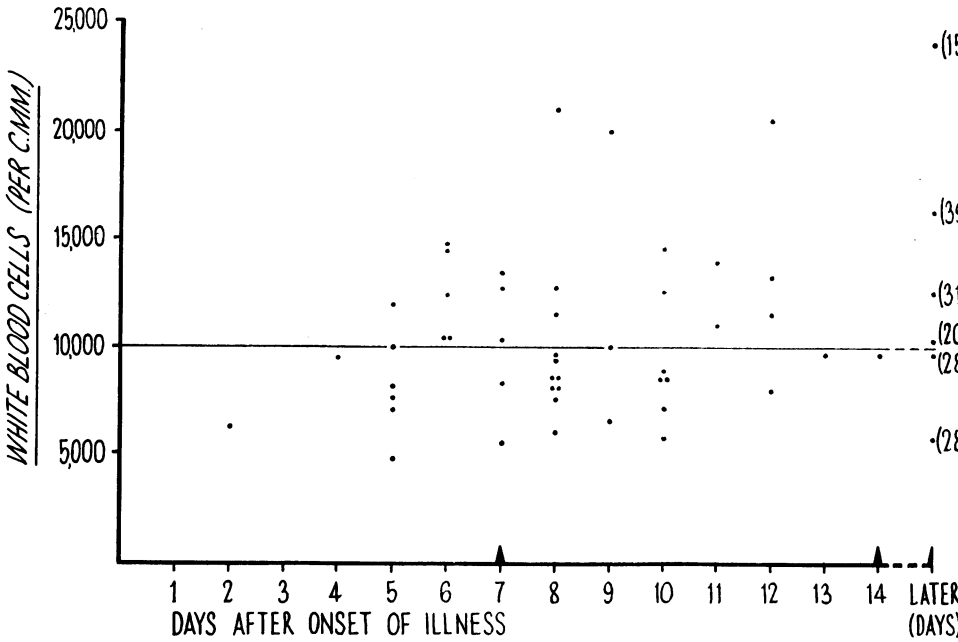
leptospiral meningitis (ten due to *L. icterohæmorrhagiæ*) that the leucocyte counts were normal. Broom and Alston (personal communication to Morgan, 1952) stated that in the first week of the disease a leucocytosis may be absent.

In this present series records are available of leucocyte counts in fifty-nine patients. These were taken from a review of eighty-four serologically proved cases of Weil's disease in Northern Ireland in the three-year period 1950-52 (Kennedy, 1953).

Taking the adult upper limit of normal as 10,000 leucocytes per c.mm. (Dacie, 1950, and others), the total leucocyte counts were as often within normal limits as not. Only twenty-seven patients (46 per cent.) had a leucocytosis. In no case were fully developed myelocytes seen and in high leucocyte counts meta-myelocytes were



LEUCOCYTE COUNTS AT VARIOUS STAGES IN WEIL'S DISEASE
(Horizontal line = upper limit of normal)

not any more common than usual; in fact, the "shift to the left," often mentioned, was not a feature. Also, a monocytosis, described by Rees (1939), did not occur.

The average leucocyte count was 10,944 per c.mm., repeat counts having been excluded. The lowest count was 4,700 per c.mm., and the highest 24,000.

DURATION OF ILLNESS.

In case a leucocytosis tended to occur at a particular stage, the leucocyte counts were plotted on the days they were taken after the onset of illness. The exact dates were known in fifty-three cases. The results are shown in the scatter diagram.

The diagram shows that there are twenty-four values above the 10,000 mark, twenty-seven below, and two at exactly 10,000. All the counts except two were

taken after the fourth day, and in some cases antibiotic treatment had already started. The correlation between the number of days after onset of illness and the total leucocyte counts show that $r=0.18$, D.F.$=51$, $P > 0.1$: i.e., the correlation co-efficient could easily have arisen by chance and providing the leucocyte count is unaffected by treatment, it can be assumed that the data show no evidence of association between the leucocyte count and the number of days after onset on which it was taken.

These findings suggest, therefore, that in the Northern Ireland cases (a) a leucocytosis was not a characteristic of the disease; and (b) a leucocytosis, when it occurred, was apparently not related to a particular stage of illness.

Relative and Absolute Leucocyte Counts.

The white cell counts were examined from two other aspects : a *relative* neutrophil leucocytosis and an *absolute* neutrophil leucocytosis. (Seventy-five per cent. neutrophils is taken as the upper limit of normal in an adult differential white cell count. The normal upper limit of the absolute value for neutrophils is regarded as 7,500 per c.mm.). In 54 cases the differential white cell count was known : in 49 the absolute neutrophil counts could be calculated. The results are set out in the accompanying table.

Neutrophil Counts in Weil's Disease: Relative and Absolute Values Compared.

Neutrophil Counts	Total Cases	Normal	High	Percentage with Leucocytosis
Relative Values -	54	18	36	67
Absolute Values -	49	21	28	57

Total white cell counts were known in 5 of the 7 fatal cases; 4 showed a leucocytosis. The differential white cell count in 6 was recorded and all had a relative increase in neutrophils.

Assuming blood films were accurately spread, the values in the table suggest that the relative neutrophil count is of somewhat greater diagnostic value than the absolute count, but the difference between the percentages is not significant at the conventional level of significance ($P < 0.05$) and more data are necessary to assess the validity of this suggestion.

Icteric and Anicteric Cases.

Hutchison et al. (1946) described 17 cases of Weil's disease in the British Army in Italy. All save one had a polymorphonuclear leucocytosis and were severely ill. There were five deaths. The exception was a mild case. In order to ascertain if severity of illness promoted a leucocytosis a comparison was made of the counts in the two groups in the Northern Ireland cases, (a) those with jaundice (including seven deaths), and (b) anicteric cases, where the illness ran a mild course. Total leucocyte counts were known in 49 jaundiced patients and in 10 without jaundice. Of the 49 jaundiced patients 25 (51 per cent.) showed a leucocytosis, compared with 2 (20 per cent.) among the anicteric cases.

If it can be assumed that the two groups are similar in respect of factors likely to affect their leucocyte count other than the presence or absence of jaundice, these figures offer some evidence of the occurrence of a leucocytosis in those who are severely ill. A difference in the two percentages as great or greater than that observed is likely to occur by chance only once in twenty trials.

Summary.

An examination of the leucocyte counts of 59 patients with Weil's disease in Northern Ireland revealed only 27 (46 per cent.) with a leucocytosis when the upper limit of normal is taken as 10,000 leucocytes per c.mm.

No evidence of significant correlation was found between leucocyte counts and the day after onset at which they were taken.

The observations are consistent with the hypothesis that a leucocytosis is more frequent in severe than in mild disease.

There were insufficient suitable data to assess whether relative or absolute neutrophil counts were the better diagnostic aid.

I am indebted to Professor G. W. Pickering, Regius Professor of Medicine, Oxford University, for permission to reproduce material from my D.M. thesis, on part of which this article is based. I wish to thank Dr. J C. Broom, Wellcome Research Institution, London, for permission to include particulars from cases of his own. I am grateful to Dr. V. D. Allison, Central Laboratory, Belfast, for allowing me access to the laboratory records, and to Dr. E. A. Cheeseman, Department of Social and Preventive Medicine of the Queen's University, Belfast, for statistical help.

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The Virus Reference Laboratory

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In the winter of 1956 plans were laid for the development of a Virus Reference Laboratory in the Department of Microbiology, the Queen's University, Belfast, to be run jointly by the University and the Northern Ireland Hospitals Authority. This has gradually come into operation over the past eighteen months, and a report of the work done by this Reference Laboratory up till the end of December, 1957, is presented in this journal, page 53. It is the purpose of this note to discuss the kind of work a virus reference laboratory may be expected to do.

It should be emphasised at the outset that a virus reference laboratory is not (like most bacteriological laboratories) prepared routinely to attempt virological diagnosis of all clinical diseases suspected to be of viral ætiology. Virology, with the exception of a few conditions, is still in a stage of "research" diagnosis rather than "routine" diagnosis. Experience of many virus reference laboratories has shown that the investigation of a single patient believed to be suffering from a virus disease may take months of detailed work and is often a waste of time and money. On the other hand, investigations of an outbreak of a few cases or of an epidemic are usually rewarding in the diagnostic help which the virus laboratory can give to the clinician or the Medical Officer of Health.

For the investigation of virus infection there are two kinds of test available—virus isolation and serological tests.

Virus isolation provides the most useful rapid result for some diseases such as poliomyelitis (and other enteric viruses such as Echo and Coxsackie viruses). This is the case also, for example, in smallpox where it is of great public health importance to be able to provide an immediate diagnosis. On the other hand, isolation of virus would not normally be attempted in such conditions as influenza, Q fever or infection with adenoviruses. The reasons for this are (a) that specimens from patients suffering from infections with these viruses are often taken after the period of infectiousness and the specimen will contain no virus; (b) the isolation of a virus and its identification may take weeks of work. For such infections, and also for survey work, the less time-consuming serological tests, are more convenient.

Serological tests may be used for diagnosis of a recent virus infection or for the purpose of surveying the prevalence of a disease in the community. For the diagnosis of a clinical infection two samples of clotted blood are required, these are usually referred to as *paired sera*. The first sample should be taken during the acute phase of the illness and the second sample two or three weeks later during

convalescence. The two sera are then tested to find out if antibody has developed or if there has been a rise of antibody in the convalescent serum. It will be obvious that if just a convalescent serum sample is taken from the patient, all that can be said is that some time in the past this individual has had, or has not had, influenza or psittacosis or Q fever or whatever virus infection is being investigated. This is what is done in survey work i.e. the test merely says the patient has at some time had the disease. Sometimes a very high titre or the presence of a particular type of antibody in a single convalescent specimen may be highly suggestive of a recent infection, but diagnosis based on this type of result is never as satisfactory as that based on a rise in antibody with paired sera. Two samples are required in order to show that a *recent* infection has been caused by a particular virus. With a few exceptions such as poliomyelitis etc. and the pox diseases—virological techniques have not been able to provide the rapid answer which can be provided with most bacteriological specimens and at the present time, with a few exceptions, viral diagnosis cannot be rapid enough to influence treatment in the early stages of an infection.

It will be appreciated that there is no value in sending a specimen ot serum to the virus reference laboratory with a request "organisms please," or "viruses please." With a great deal of work it could be shown that any such serum contained antibody to perhaps a dozen or more viruses. Such information would be of no value to the clinician although of interest when considered with several hundred other sera for the purpose of surveying the prevalence of various infections.

For cases of illness in which a presumptive viral diagnosis has been made, the type of specimen which should be sent is outlined in the appendix of this paper. If there is any doubt what specimens should be taken, it is best to telephone the Reference Laboratory and discuss the procedure for collecting and sending the specimens. At the present time there are no virus diagnostic tests for infectious or serum hepatitis nor for infectious mononucleosis (glandular fever).

In general the kind of investigations which the Virus Reference Laboratory can do, and we feel should be doing, may best be seen by summarising some of the investigations with which it has been concerned during the past eighteen months as reported on page 53.

One of the first investigations made was a serological survey of the distribution of poliomyelitis antibodies in the child population of Northern Ireland. This gave information, not only on the prevalence and types of poliomyelitis in the community but also provided a guide to priorities etc. in vaccination programmes. Later on the Reference Laboratory undertook to attempt isolation of virus in all cases of paralytic poliomyelitis and asceptic meningitis. This study has not only given information on the epidemiology of poliomyelitis in Northern Ireand, but also led to the discovery that a member of the Russian Spring-Summer—louping ill group of viruses was producing an illness in Northern Ireland clinically unrecognisable from poliomyelitis. This type of investigation will continue, as there are several viruses which can mimic poliomyelitis infection. Not only will it be possible to find out more about them, but, since these viruses may infect children who have

been vaccinated against poliomyelitis, it is essential to have this kind of study in progress to evaluate the vaccine and to discover when booster shots of vaccine may be required.

Another example of the type of work the Reference Laboratory has done, and should be doing, was the investigation last year of an outbreak of a C.N.S. infection which mimicked poliomyelitis amongst young adults in Maghera (Co. Londonderry). It was possible to say within a few days that the outbreak was not due to poliomyelitis : the cause of this epidemic is still being investigated. Again, when a poliomyelitis case occurred in a hospital, it was possible within a few days to say which of the other children were excreting poliovirus, so that necessary quarantine measures could be established. Similarly, if a case of smallpox occurred in Northern Ireland, we would hope to be able to give a presumptive diagnosis within an hour of receiving suitable specimens which would be confirmed twenty-four to forty-eight hours later.

In the case of influenza there is never great urgency to have a diagnosis as to the type etc. of infecting virus. During 1956 and 1957 we tested a number of sera for evidence of influenza. These tests, as has been noted, demand that an acute and convalescent sample of clotted blood be sent to the laboratory. Athough the laboratory can give no immediate help in the treament of the acute illness, such tests are of great public health importance to the community. By studying these sera from cases of respiratory diseases seen by various doctors, it was possible to show that early in September, 1957, Influenza A had arrived in Northern Ireland. It was *then* worthwhile discovering which strain of influenza was spreading in the community. This necessitated the isolation of virus, which was done from a number of selected patients.

The viruses isolated were shown to be strains of 'A' (Asain) influenza, and were sent to the WHO influenza centre for comparison with strains of influenza virus from other parts of the world. There are many laboratories scattered over the world, which, like the Virus Reference Laboratory in Belfast, are acting as listening posts for WHO, and the success of this arrangement can be seen from the rapidity with which information on the outbreak of Asian influenza was disseminated last year, and the rapidity with which vaccines were made available. It may be of interest to note that the virus of Asian influenza was first reported from Singapore in May, 1957, and pilot batches of Asian influenza vaccine was being made in the Reference Laboratory in Belfast in July, 1957—as a precautionary measure. If the epidemic had proved to be serious, i.e., if there had been a comparable case fatality rate to that of the 1919 influenza epidemic, we wished to be in a position to make vaccine for Northern Ireland if supplies had not been available in time through the usual commercial channels. It is important then that we should be on the look-out all the time for outbreaks of respiratory disease which could well be the starting point of an epidemic.

Among the other types of work which the Reference Laboratory has done was a survey for the presence of Q fever in Northern Ireland. So far the evidence suggests that Q fever is not present, making Northern Ireland one of the few countries in

SPECIMENS REQUIRED FOR INVE

NOTE (a) Keep cold = keep specimen on ice or in a refrigerator but
(b) Serological tests will not normally be done unless paired
(c) If in doubt please telephone the laboratory for information

1 ILLNESS	2 SPORADIC CASES
Influenza. Adeno-pharyngeal-conjunctival fever. "Epidemic conjunctivitis."	(1) 5-10 ml. of clotted blood taken during first few days of illness. AND (2) 14-21 days later 5-10 ml. of clotted blood.
Psittacosis. Q fever. Atypical pneumonia.	As for influenza (see above).
Respiratory diseases of infants and babies.	As for outbreak of influenza col. 3 above.
Poliomyelitis. Aseptic Meningitis. Coxsackie and Echo virus.	(1) Sample of fæces from bed-pan containing no disinfectant. Sent to lab. in waxed container and kept cold. (2) 5-10 ml. of clotted blood taken at same time as fæces sample. (3) If available 2-3 ml. of CSF kept cold. AND (4) 21 days later 5-10 ml. clotted blood.
Encephalitis. Louping ill—Russian Spring Summer encephalitis. Lymphocytic-choriomeningitis. Mumps encephalitis.	(1) 2 ml. CSF kept cold and sent to lab. on ice. (2) 10 ml. clotted blood kept cold and sent to lab. on ice. (3) Sample of fæces from bed-pan containing no disinfectant, sent to lab. in waxed container and kept cold. AND (4) 21 days later 5-10 ml. clotted blood.
Herpes simplex—other vesicular diseases other than smallpox.	(1) Vesicle fluid in a capillary tube or bijou bottle, or scrapings from lesion in a bijou bottle, keep cold and send to lab. on ice. (2) 5-10 ml. of clotted blood. AND (3) 21 days later 5-10 ml. of clotted blood.
Exanthemata *other* than measles, scarlet, rubella.	As for poliomyelitis (see above).
Lymphogranuloma venereum.	(1) Sample of pus in sterile bijou and kept cold and sent to lab. on ice. (2) 5-10 ml. clotted blood. AND (3) 21 days later 5-10 ml. of clotted blood.
Smallpox.	Telephone smallpox consultants and laboratory 30503 Ext. 238.

Undiagnosed disease of possible viral ætiology.

do NOT freeze.

samples of sera are received, i.e., an acute phase and a convalescent serum.

on collecting and dispatching specimens.

3 Outbreak of Several Cases or Epidemic	4 Fatal Cases
(1) Throat swab taken with swab on wooden applicator. Swab broken off into bijou bottle containing 2 ml. of normal saline and 0.5 ml. of sterile broth. This must be kept cold and sent to lab. on ice. (2) 5-10 ml. of clotted blood taken at same time as throat swab. (3) 14-21 days later, 5-10 ml. of clotted blood.	(1) Portion of lung in sterile bottle kept cold and sent to lab. on ice. (2) Swab of bronchi taken as for throat swab in col. 3 above.
Telephone Laboratory 30503 Ext. 238.	
As above.	
As for sporadic cases col. 2.	(1) Sample of fæces as in col. 2. (2) Piece of cortex, mid-brain pons, medulla and cord in sterile bottle and kept cold, sent to lab. on ice.
Telephone Laboratory 30503 Ext. 238.	(3) CNS material for histology to Pathology lab.
Telephone Laboratory 30503 Ext. 238.	(1) Piece of skin or mucous membrane with vesicles. Keep cold and send to lab. on ice. (2) Piece of cortex, mid-brain, pons medulla and cord in sterile bottle. Kept cold and sent to lab. on ice. (3) CNS material for histology to Pathology lab.
As for poliomyelitis (see above).	Telephone Laboratory 30503 Ext. 238.
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Telephone Laboratory 30503 Ext. 337.

the world where it has not been found. Surveys of psittacosis are also being undertaken, and it has been observed, for example, that Belfast pigeons are commonly infected with psittacosis virus.

It will be clear that the type of work in which the Virus Reference Laboratory can be of most use to the community is—

(a) To investigate outbreaks and epidemics of virus diseases.

(b) By serological surveys and epidemiological studies to define the frequency and importance in Northern Ireland of the known virus diseases of man.

(c) On suitable occasions to assist clinicians in the investigation of individual cases believed to be of viral origin.

(d) To assist clinicians and public health workers in research problems of illness of viral origin.

The diagnosis of virus infections has undergone great development in the last ten years and no doubt this will continue until virus and bacterial infections can be diagnosed with equal ease and rapidity. At the moment, with the exception of certain virus diseases, the diagnosis can only be made when it is too late to influence the course of treatment. This does not mean that most investigations are a waste of time, but it does mean that we should regard a Virus Reference Laboratory more as a means of increasing the knowledge of virologists, clinicians and those concerned with public health, rather than simply as a routine diagnostic laboratory.

REVIEW

AIDS TO ORGANIC CHEMISTRY. By George A. Maw, Ph.D., F.R.I.C. Fifth Edition. (Pp. vii + 176. 10s. 6d.) London: Baillière, Tindall & Cox, 1958.

THE author of the present edition states that care should be taken to avoid reducing the subject to a mere catalogue of formulæ. However, a fairly extensive field is presented to the student in such brief compass, that only those with a good practical knowledge of chemistry are likely to be stimulated to any interest in the subject.

the world where it has not been found. Surveys of psittacosis are also being undertaken, and it has been observed, for example, that Belfast pigeons are commonly infected with psittacosis virus.

It will be clear that the type of work in which the Virus Reference Laboratory can be of most use to the community is—

(a) To investigate outbreaks and epidemics of virus diseases.

(b) By serological surveys and epidemiological studies to define the frequency and importance in Northern Ireland of the known virus diseases of man.

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The Virus Reference Laboratory

Department of Microbiology, The Queen's University, Belfast

Report for 1957

by

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(Assistant Lecturer)

D. S. Dane, M.B.(Cantab.)
(Lecturer)

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E. Moya Briggs, B.Sc., J. J. McAlister, A.I.M.L.T., R. Nelson and D. C. Wilson, F.I.M.L.T.

A Virus Reference Laboratory supported by a grant from the Northern Ireland Hospitals Authority was started in the Department of Microbiology, the Queen's University, in 1957. The aims and purpose of this laboratory are described elsewhere in this journal (page 47). This first annual report for 1957 also includes the virus reference work done in the department during 1955 and 1956.

Poliomyelitis, Aseptic Meningitis and Encephalitis, 1955, 1956 and 1957.

Paralytic Poliomyelitis.

The laboratory diagnosis of poliomyelitis has been undertaken in the department since August, 1955. Our aim has been to receive specimens from all notified paralytic cases, and in practice, thanks to the co-operation of the physicians, we have received specimens from over (seems) 95 per cent. of them. Isolation of virus from a patient's fæces is the most rapid and certain method of diagnosing poliomyelitis and this technique has been used routinely. In certain cases serological tests have been done, but these are likely to give equivocal results, are expensive to perform and do not allow such a rapid diagnosis as virus isolation.

The results of attempted virus isolation from the fæces of patients with a diagnosis of paralytic poliomyelitis from August, 1955, until December, 1957, are given in the following table :—

Year	No. of Cases Investigated	No. of Polio Viruses Isolated			No Virus Isolated
		Type I	Type II	Type III	
1955 (Aug. to Dec.)	22	20	1	0	1
1956	25	20	2	0	3
1957	204	174	0	0	30
Aug., 1955-Dec., 1957	251	214	3	0	34

It should be noted that all three types of poliovirus can be isolated with equal ease and that serological surveys have indicated that the three types of poliovirus are about equally common in Northern Ireland, though of course one type may be found more frequently during any one season in one area.

Type I virus is responsible for the majority of cases of paralytic poliomyelitis and for the majority of poliomyelitis epidemics throughout the world. Over the past three years it has been of even greater importance in Northern Ireland than experience elsewhere might have led us to predict. It is worth noting that the Salk-type poliomyelitis vaccines in use at the present time contain formalin-killed virus of all three types. Antigenically the type I component often appears to be the least effective. Any measures which can be taken to make this the most powerful component would appear to be both logical and desirable.

Aseptic Meningitis.

From August, 1955, to December, 1957, specimens were received from 367 cases of aseptic meningitis. The results of attempted virus isolation from these patients are tabulated below :—

Year	No. of Cases Investigated	Number of Viruses Isolated				
		Poliovirus Type I	Poliovirus Type II	Poliovirus Type III	Coxsackie or Echo	No Virus Isolated
1955 (Aug. to Dec.)	24	2	1	2	10	9
1956	40	3	1	1	4	31
1957	303	125	0	0	27	151
Aug., 1955-Dec., 1957	367	130	2	3	41	191

Type I poliomyelitis was isolated from the majority of cases of aseptic meningitis in 1957, but in all three years a few cases of aseptic meningitis were associated with Coxsackie and Echo virus infections. These cases have in general been sporadic and scattered. One small outbreak of aseptic meningitis caused by a Coxsackie Group B virus occurred in Ballynahinch, Co. Down, in October, 1955.

During the past few years a number of different enteric viruses falling into the Coxsackie and Echo groups have now been shown in various parts of the world to be responsible, at times, for cases of aseptic meningitis. Recently typing sera have become available so that these viruses may be identified. It is hoped that during 1958 all enteric viruses so far isolated in Northern Ireland will be typed and that in future they will be identified soon after they are isolated.

In July, 1957, there was an outbreak of aseptic meningitis among adults at Maghera, Co. Londonderry. This was investigated with the help of Dr. K. W. Newell of the Department of Social Medicine. Preliminary results show that the outbreak was not caused by a poliovirus nor by the Coxsackie virus which was recovered from a few juvenile cases of aseptic meningitis in that part of Northern Ireland at that time, and that was possibly due to louping ill virus.

Paired sera of all patients from whom no virus was recovered were tested for serological evidence of recent infection with mumps virus and also lymphocytic choriomeningitis virus (L.C.M.) Five cases were found to have been caused by mumps virus, but none were due to L.C.M. virus.

Louping ill.

Five cases of infection with a virus of the Russian spring-summer/louping ill group were diagnosed in the laboratory in 1957. Of the five cases, four were clinically thought to be acute poliomyelitis and one encephalitis. Infection with viruses of this group may give rise to an illness resembling acute poliomyelitis in either its parayltic or its non-paralytic form, or it may cause encephalitis. These findings have been reported in detail elsewhere (Likar and Dane, 1958).

Further research during the next few years will reveal how common human infections with louping ill virus are in Northern Ireland. This virus may be responsible for the majority of poliomyelitis-like illnesses among adults in rural areas.

Virus Encephalitis.

Specimens from a small number of cases of possible virus encephalitis have been received during the year. A firm laboratory diagnosis was made in two cases. One was caused by type I poliovirus, one by louping ill virus. The ætiology of encephalitis in Northern Ireland will be the subject of detailed research in 1958 and it is hoped that physicans will send specimens to the laboratory from all cases under their care (see pages 50-51).

Special Investigations.

1. *Surveys of poliomyelitis antibody* in population groups in Northern Ireland have been made. A survey in children from rural and urban areas has been published elsewhere (Dane, Dick, Connolly, Briggs and McLeod, 1956). The following table gives the results of similar studies for medical students and for Northern Ireland Fever Hospital (N.I.F.H.) student nurses :—

	Number Examined	Number with Antibody to: One Polio-virus Type	Two Polio-virus Types	All Three Polio-virus Types	Number with no Antibody to any of the Three Polio-virus Types
Medical Students ...	37	9	17	8	3
Student Nurses ...	33	5	16	11	1

2. *The excretion of polioviruses by normal children* was studied in March, 1956. Fæcal specimens from approximately fifty children attending nine nursery schools in the Belfast area were obtained with the help of Dr. W. J. McLeod and his staff. Type II poliovirus was recovered from two children at different schools and type III from a child at another school. In addition, three other enteric viruses were isolated from this group of children.

3. *Belfast sewage* was examined during the early months of 1957 for the presence of polioviruses and other enteric viruses, but even though sensitive isolation techniques were employed, no viruses were recovered. Our hope that long-term examination of sewage might be used in comparing the prevalence of polioviruses before and after mass vaccination against poliomyelitis had to be abandoned.

4. *The isolation of poliovirus from the throats and mouths of clinical cases and their contacts.* With the help of Dr. F. F. Kane and Dr. W. J. McLeod, throat swabs and mouth swabs were obtained from thirty-one cases of type I poliomyelitis and from seventeen of their home contacts. The relative importance of fæcal and oral excreted virus is still largely unknown and this investigation was done because there have been few quantitative studies of throat virus and virus in the anterior part of the mouth. The following table summarizes our findings :—

Clinical Diagnosis	Number Examined	Number with Fæcal Virus	Number with Throat Virus	Titres per Gram of Throat Secretion	Number with Mouth Virus	Titre per Gram of Saliva	Number from whom no Virus Isolated
Paralytic Poliomyelitis	20	19	9	$10^{2.8}$ to $10^{4.1}$	0	—	0
Non-paralytic Poliomyelitis	11	10	3	$10^{2.8}$ to $10^{4.8}$	*1	$10^{2.8}$	0
Family Contacts of Cases	17	Not Tested	1	$10^{3.6}$	0	—	16

*Virus was found in the anterior part of the mouth in the individual with the highest titre of virus in the throat.

5. *Cerebro-spinal fluids* from one hundred patients admitted to the Northern Ireland Fever Hospital with acute poliomyelitis or asceptic meningitis were examined. No viruses were isolated from any of the fluids. This investigation was prompted by reports that Coxsackie or Echo viruses may be recovered from the C.S.F. of some patients with asceptic meningitis. In infections with these viruses isolation of the virus from the C.S.F. would obviously be of more value in deciding whether a patient's illness was actually caused by the virus than if it had only been isolated from fæces.

6. *Serological Tests for naturally-acquired immunity to poliomyelitis.* Because the rate of natural immunisation to poliomyelitis in Northern Ireland is high, vaccination is obviously unnecessary in many people. While vaccine has been in short supply we have been testing the antibodies of individuals at special risk to find out if they were already immune, and therefore did not require vaccination. Sera from 145 individuals have so far been screened in this manner.

7. *The investigation of poliomyelitis in vaccinated indivduals.* Specimens were received from two children in Belfast suspected of having abortive attacks of

poliomyelitis shortly after receiving Salk-type vaccine. No evidence was found that either of these children had had a poliomyelitis infection. There was one case of paralytic poliomyelitis in a vaccinated girl aged 15 years. She had received a single dose of Salk vaccine in the U.S.A. in 1956. A case of paralytic poliomyelitis (in the South of England) following the second dose of Salk-type vaccine was investigated at the request of a physician. It was not possible to show that the association between vaccination and the development of paralytic poliomyelitis was fortuitous.

8. *Poliomyelitis in children's wards.* In September, a case of paralytic poliomyelitis occurred in a children's ward in a Belfast hospital. The child, a boy aged 6, had been in hospital about fourteen days before he became paralysed and the question naturally arose whether other children in the ward were infected. Though at the time he developed paralysis he was in a large ward with sixteen other children, he had previously been in a side ward with only two other children. Fæcal specimens were obtained daily from the children in the ward and all the children and nurses were tested for naturally acquired immunity to poliomyelitis. Within a day the only other child who was infected with the virus had been discovered and isolated. He remained well but continued excreting virus for a few days. No other child developed a poliovirus infection, and six were found to be already immune to the poliovirus type which caused the case (type I). Another child was excreting an enteric virus which was not a poliovirus. She was also isolated as a precautionary measure.

Investigation of children's wards at another hospital where a child had been admitted with poliomyelitis showed a different picture. In the nursery ward fæcal specimens from fourteen children were examined. Two were found to be excreting type I poliovirus and six were found to be excreting other enteric viruses.

Although these investigations were done to help the physicians in charge of the wards, it is nevertheless possible to draw some tentative conclusions.

(a) Given conditions where close contact between children in wards can be prevented and where there are some facilities for isolation of virus excretors, it may be possible to control the spread of poliovirus in a children's ward.

(b) Given conditions where there is close contact between young children in a ward, that ward is likely to become a focus of infection with polioviruses and other enteric viruses. The majority of these infections will probably be inapparent and do no harm, but the potential hazard should be appreciated by physicians. In particular, it should be stressed that during a poliomyelitis epidemic children who might have poliomyelitis should not be admitted to an open ward for observation. Admission to a side ward with barrier nursing until the results of C.S.F. and fæcal examination can be reported, would seem a logical course to adopt if admission to a general hospital is considered necessary. The case of paralytic poliomelyitis reported above, in fact occurred in a children's ward without warning and *not* because of the admission of a child with suspected poliomyelitis, and it was therefore unavoidable.

Respiratory Virus Infections.

Influenza.

In 1956 no cases of influenza 'A' were diagnosed, but in November and December there were two cases each of influenza 'B' and 'C.'

During 1957, 360 serological tests for influenza virus infection were done in cases of influenza-like illness. The results of these tests are summarized below :—

	Evidence of Recent Infection	No Evidence of Recent Infection	Total
Influenza "A" Virus ...	49	91	140
Influenza "B" Virus ...	0	117	117
Influenza "C" Virus ...	2	101	103
			360

One case of influenza 'C' infection was from Ballymena, Co. Antrim, in April and the other case from Banbridge, Co. Down, in July.

The first reports of "Asian 'flu" (caused by a new antigenic variant of influenza 'A' virus) were received from Singapore in May, 1957. The first serologically proven case in N. Ireland was from the R.A.F. station at Aldergrove on 12th September and the first Asian type virus was isolated from a nurse at R.V.H. on the 14th September. Before this date we had failed to isolate influenza 'A' virus from any patients in 1957. During September more strains of influenza 'A' (Asian) virus were recovered from R.A.F. personnel at Aldergrove and Ballykelly, and also from fatal civilian cases. A total of nine strains were isolated and five of these were sent to the W.H.O. Influenza Centre in London for comparison with Asian strains from other countries. The serological evidence of infection with influenza 'A' (Asian) from patients with influenza-like illnesses is summarized below month by month for 1957 :—

	No. Tested	No. Positive
January to August - - -	45 ...	0
September - - - -	71 ...	42
October - - - -	18 ...	7
November - - - -	2 ...	0
December - - - -	4 ...	0
Total - -	140	49

A special study of "Asian 'flu" was made in university students. After the epidemic a specimen of blood was taken from eighty-three students, and they were asked to complete a form giving details of the signs and symptoms of any recent influenza-like illnesses. The object of this investigation was to determine how many students had been infected with the Asian virus and the nature of the illness

it produced. Also we wished to know the proportion of students who had suffered from influenza-like illnesses during the epidemic, but who had not in fact been infected with the "Asian 'flu" virus. The results of this study will be published elsewhere.

Influenza Vaccine.

When the influenza 'A' epidemic was reported from Singapore in May, 1957, it seemed probable that later in the year the epidemic would spread to the British Isles. There were conflicting reports from Asia about the severity of the epidemic, but on the whole the case fatality rate appeared to be low. However, this did not necessarily mean that the epidemic in the British Isles would be mild, because the severe influenza epidemic of 1918-1919 was preceded in the summer of 1918 by a smaller and much less serious one. For this reason, as a precautionary measure, pilot batches of influenza 'A' (Asian) vaccine were prepared in the laboratory during July. If the epidemic had changed its character and become more severe, as in 1918-1919, we wished to be in a position to produce vaccine for Northern Ireland, because the amount available through commercial channels was likely to be insufficient. Subsequently, when the continuing mild nature of the epidemic became apparent, we abandoned our plans for vaccine production.

Psittacosis.

Sera from 238 patients were tested against the psittacosis group of viruses. In only one case was there evidence suggestive of a recent infection.

A preliminary survey of domestic pigeons in Northern Ireland showed that a high proportion had been infected with psittacosis virus and presumably many of these birds were latent virus carriers. This situation gives no cause for alarm; it has been found in other countries and is not associated with a high incidence of psittacosis in the human population.

'Q' Fever.

Sera from forty-one patients were examined for complement-fixing antibodies to Rickettsia burneti (Nine mile strain). In no instance was evidence of past or recent infection found. In countries where 'Q' fever is enzootic in domestic animals it is usual to find evidence of past infection (C.F. antibodies) in abattoir workers, who, by the nature of their work, are particularly liable to contract this disease or to suffer from inapparent infections. For this reason sera from sixty-one abattoir workers were tested for 'Q' fever C.F. antibodies. No positive results were found and we consider this to be evidence that infection with *R. burneti* is at least uncommon among domestic animals in Northern Ireland, and perhaps does not occur at all.

Adenoviruses (Adeno-pharyngeal-conjunctival fever).

Sera from ninety-five patients were tested for recent adenovirus infections. In only one case was a positive result obtained. Thus, in 1957, adenoviruses appeared to be an uncommon cause of respiratory tract infections in Northern Ireland.

Serological Tests for Virus and Bacterial Diseases in the Population of Socotra.

In 1956 an expedition from Oxford University visited the island of Socotra in the Gulf of Aden. Arrangements were made for the Virus Reference Laboratory to test blood specimens collected from the inhabitants for evidence of past infection with certain viruses and bacteria. Ninety-eight serum specimens from adults were available, but because of storage difficulties in transit not all were suitable for all the tests planned. The following table summarizes our findings :—

	No. Tested	No. Positive
Poliovirus I	98	94
Poliovirus II	98	92
Poliovirus III	98	90
Yellow fever Virus	98	3
West Nile Virus	98	0
Psittacosis Group of Viruses	62	0
R. burneti ("Q" Fever)	62	0
Weil Felix OX 19	98	1
Weil Felix OX 2	98	0
Weil Felix OX K	98	1
Salmonella typhi	98	1
Salmonella paratyphi A	98	0
Salmonella paratyphi B	98	0
Salmonella paratyphi C	98	0
Brucella melitensis	98	0
Kahn Test (Syphilis or Yaws)	90	2

The high incidence of infection with all three types of poliovirus suggests that in contrast the clinical disease is probably rare and will occur only in infants.

Only three individuals had antibody which neutralized yellow fever virus. Because of the antigenic relationship of yellow fever to a number of other viruses these results *cannot* be taken to indicate that yellow fever virus infections occur among humans on the island.

We wish to acknowledge the very great co-operation which we have received from many physicians and medical officers of health.

We also wish to thank Mr. Fred Burns, F.I.M.L.T., Senior Laboratory Technician, Department of Microbiology, for his valuable help in all aspects of the work of the Virus Reference Laboratory.

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Superior Vena Caval Obstruction complicating Carcinoma of Bronchus

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OBSTRUCTION of the superior vena cava is a relatively common complication of bronchial neoplasm. The following case history describes its appearance as the presenting sign, of unusually rapid onset, in a patient with an unsuspected carcinoma of bronchus.

CASE HISTORY.

A man aged 74 was admitted to hospital complaining of weakness in his legs and of unsteadiness which had caused him to fall a fortnight previously. Owing to this he had become confined to bed, and the request for admission was made because of his social difficulties and not for a specific medical cause, for he lived alone, dependent on inadequate daily help. He was known to have had diabetes since 1949, complicated by hypertension and albumiuria attributed to chronic nephritis. He had a right cerebral thrombosis in 1952 with good recovery.

On examination, he was an obese, plethoric, elderly man with blepharitis of both eyes and a dry irritating unproductive cough. He was dyspnœic but not cyanosed. Lymph glands were palpable in both axillæ and there was œdema of both wrists and forearms, more marked on the right, with minimal œdema of sacrum and ankles. There were old scars in both antecubital fossæ due to burns. His chest was emphysematous with high-pitched rhonchi and crepitations over both lungs. There were no abnormal findings in the cardiovascular system at first :—the venous pressure was not raised, the pulse rate was regular, 80 per minute, with equal volume on both sides. The blood pressure was 150/70. There were no abnormal findings on abdominal or on rectal examination other than constipation and moderate prostatic enlargement. He was mentally alert and rational, his fundi were obscured by early cataracts and he had minimal residual evidence of the old left hemiplegia.

Investigations:

Hb. 68 per cent.
Blood sugar 208 mgs. per cent.
Blood urea 70 mgs. per cent.
Urine contained a trace of albumin and of sugar.
Specific gravity range test fixed at 1010.
W.R. negative.
Serum electrolytes normal.

The diabetes was controlled by diet alone.

On the second day in hospital a rise in jugular venous pressure was noted on the right side and his face had become cyanosed and suffused. By the fourth day the pitting œdema of the arms was much more marked, extending from the hands to the shoulders, and down the chest wall posteriorly. Distended veins had appeared over the upper chest wall and he was much more breathless. At this time there was no œdema of the lower limbs nor of the sacrum, and there was no ascites. Superior vena caval thrombosis secondary to mediastinal neoplasm was suspected and a course of anticoagulants and antibiotics was started. A chest X-ray showed shadowing extending out from the right side of the upper mediastinum attributed either to a mass in the mediastinum, or to an aneurysm of the aorta. Screening of the chest would have helped but the patient was too ill for this. His condition quickly deteriorated, the jugular venous pressure rising on both sides to the angle of the jaw, the œdema increasing in the upper half of the body, and the distended veins becoming more evident and more numerous over the chest wall. Œdema was present over the lower ribs posteriorly obscuring the basal breath sounds, but there was no œdema below the level of T.12. Persistent crepitations were widespread throughout the chest with some bronchospasm. Urinary output remained poor and the blood urea continued to rise. He died eighteen days after admission.

Pathological Report.

The body was that of an elderly man with subcutaneous œdema of both upper limbs and upper thorax. Each pleural cavity contained a small amount of amber fluid and in isolated areas firm adhesions bound both lungs to the overlying parietal pleura. The thoracic and neck organs were removed together to enable examination of the superior vena cava throughout its whole length.

Arising from the right bronchus, close to the origin of the bronchus to the upper lobe, and ulcerating the bronchial mucosa, a carcinoma was found. This tumour, measuring 8 cms. in diameter, infiltrated the nearby lung and mediastinal tissues irregularly, and displaced the surrounding structures. The superior vena cava was compressed from the point 2 cms. beyond the crista terminalis by the tumour mass which actually invaginated its wall. The occluded portion measured 4 cms. in length and a fine probe (1 mm.) could only be passed into this portion of the vessel with difficulty. No secondary deposits were found elsewhere in the body and microscopical examination of the tumour showed it to be an anaplastic carcinoma. The cells were large with much pale cytoplasm and showed no special arrangement.

The lungs were emphysematous and there was an associated right ventricular hypertrophy, the wall measuring 7 mm. in thickness. The kidneys were pale and granular; microscopical examination of them revealed irregular scarring with a well marked Kimmelsteil-Wilson change.

Discussion.

It was remarkable how quickly this patient deteriorated after the onset of the signs and symptoms of superior vena caval obstruction. He was relatively fit and well nourished on admission and yet within four days the full picture of obstruction of the mediastinal vessels had appeared and he was dead twelve days later. It is

unlikely that the renal failure contributed much towards his death as it had obviously been present in a mild degree for many years.

The increase in the incidence of bronchial carcinoma in recent years is well known, but the frequency of its association with obstruction of the mediastinal vessels may not be so widely appreciated. In 1949 McIntire and Sykes found 250 cases of superior vena caval obstruction reported in the literature over the previous 45 years; Szur and Bromley (1956) described 107 instances from a series of 732 patients with bronchial carcinoma investigated over a period of 4 years at the Bronchus Tumour Clinic of the Hammersmith Hospital, an incidence of 14.6 per cent. (Of these 107 cases diagnosed clinically, 38 were confirmed at post-mortem).

This is an unusually high incidence when compared with the records of some 12,000 autopsies from the pathology department of the Queen's University where, from 315 cases of carcinoma of the bronchus there were only 17 instances of obstructions to the venous return to the heart (5.4 per cent.), and 10 others where such obstruction was suspected in life but not confirmed at post-mortem. Two-thirds of the proven cases were males, and two-thirds of these occurred in the fifth and sixth decades. The superior vena cava was affected in ten instances, one or other of its main radicles in five; on one occasion a large tumour mass caused obstruction in the right auricle, and once the inferior vena cava was compressed. The tumour originated on the right side in fifteen cases, but when the left bronchus was involved obstruction of the left innominate or left subclavian vein only was found. Compression was the only mechanism of obstruction in almost half of the cases, thrombosis having occurred secondary to tumour invasion, or as a terminal event, in the others.

Szur and Bromley stressed the importance of early diagnosis in this condition, because their experience suggested that life can be made much more tolerable for many of its victims by radiotherapy. They recommended 3,000 to 4,000 R units over a period of three to four weeks, and in a series of 107 treated patients 74 were improved, half of them gaining complete relief from distressing symptoms such as intense dyspnœa and œdema. The other 33 patients remained unchanged. They did not claim that radiotherapy prolongs life, indeed the majority of their patients were dead within six months of diagnosis, and radiotherapy naturally may be expected to do more for patients whose vessels are obstructed by compression than it can do for those where thrombosis has occurred.

Summary.

An instance of superior vena caval obstruction due to carcinoma of the bronchus is described. The incidence, treatment and prognosis of the condition is reviewed with special reference to sixteen other such cases on the records of the Department of Pathology of the Queen's University, Belfast.

We are grateful to Dr. G. F. Adams and to Professor J. H. Biggart for access to the clinical notes and pathological records.

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Hospital Bed Accommodation Requirements of a Region

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Read to the Royal Statistical Society (Medical Section) on 26th February, 1957, and to the British Medical Association (N.I. Branch), 13th March, 1958

FROM time to time assessments have been made of the number of hospital beds required per thousand of the population served. In 1950 the Ministry of Health published a Memorandum on the Development of Consultant Services which, on the overall pattern suggests a figure of from six to seven acute beds per thousand, excluding maternity beds.

It is, however, essential to define the particular area, and particular requirements within a Region, and to define very closely the assumptions on which calculations are based.

For example, in an average Region with a University Teaching School and a hinterland of provincial towns and rural areas, certain specialities will naturally be aggregated at the Teaching Centre giving such a picture of six to seven beds per thousand of population in the University City, but, when such specialities are excluded, but local maternity beds included, the figure for acute beds for a Provincial Centre falls to just over five beds per thousand of population served.

A Report of the Department of Health for Scotland in 1946 gave eight beds for a closely-knit industrial area, but this figure excluded maternity beds and included chronic sick other than aged and infirm.

From 1938 onwards, the Ministry of Health, in association with the Nuffield Provincial Hospitals Trust, agreed to arrange for surveys of various hospital areas in England and Wales.

The following recommended standards are taken from these surveys.

Region	Acute Beds per 1,000 Population	Chronic Beds per 1,000 Population
East Anglia	3.5	2.0
Sheffield	4.0*	2.0
Yorkshire	4.0*	2.0
South Wales	5.0*	1.5
South Western	3.4	1.7

*Excludes Maternity.

Sir Ernest Rock Carling and Dr. T. S. McIntosh, who were the surveyors for the North-Western Area, stated—"We do not think it possible at present to arrive at a figure of so many beds per thousand of the population to be adopted as

a standard. The actual rates vary greatly in different districts and this probably reflects (though not accurately) a variation in need."

Since the Nuffield Surveyors reported on Northern Ireland (giving for Belfast itself an approximate figure of 4.1 beds per thousand, including maternity and convalescent beds) certain case-load studies have been undertaken, comparing like areas with like, comparing waiting-lists by areas and by specialities, and assessments have been made of the over-all requirements of the Province.

The present study is thought to take one a little nearer a precise definition and to provide a few ideas of interest to those working in other parts of the United Kingdom.

The following table, relating to actual accommodation available at the end of 1955, shows a rough comparison of standards in Belfast at that date with standards in three large English cities of comparable size with a Medical School associated with the hospitals in question.

BEDS PER THOUSAND POPULATION.

	BELFAST	LEEDS	BRISTOL	SHEFFIELD
General Medicine	1.3	1.0	1.1	1.3
General Surgery	1.6	0.9	1.3	1.2
Unclassified	0.5	0.6	0.3	0.4
	3.4	2.5	2.7	2.9
Gynæcology	0.4	0.3	0.3	0.3
E. and E.N.T.	0.4	0.3	0.5	0.3
Other Acute	1.5	1.1	0.8	1.4
	5.7	4.2	4.3	4.9
Chronic Sick	1.0	1.4	2.7	2.5
Maternity	0.6	0.5	0.5	0.6
Convalescent	0.2	0.3	0.2	–
	7.5	6.4	7.7	8.0

The Nuffield Trust figures are for the critical number of beds and no allowance is made for seasonal variations or for beds out of commission for repairs. Three hospitals (to a total of about five hundred beds) in Belfast have elected to remain outside the Health Service and are not included in the calculation.

Finally, these figures were recorded at a time when under-development of areas outside Belfast was openly acknowledged, and this led to a greater demand on beds in Belfast for people living outside the city than probably applies even now, and much greater than should apply in the future, as we shall see.

In considering our case-load studies we must first of all determine our Bed-Use Factors, and we are indebted to the Hospital Administrative Staff College for the following illustrations.

The three important factors in the calculation of bed occupancy are the Turnover (T), the Average Length of Stay (LS), and the Turnover Interval (TI).

$$\text{Let } T = \frac{\text{Total discharges and deaths per annum}}{\text{Average number of available beds}}$$

e.g., for a 30-bed department this might come to $\frac{540}{30}$ = 18 patients per bed per annum

$$LS = \frac{\text{Total occupied bed-days}}{\text{Total discharges and deaths}}$$

e.g., $\frac{8,640}{540} = 16$ days per patient.

$$TI = \frac{\text{Total available vacant bed-days}}{\text{Total discharges and deaths}}$$

e.g, $\frac{2,160}{540} = 4$ days.

(In practice we have found that a turnover interval of $1\frac{1}{2}$ to 2 days is more realistic, and may be even less in a hospital with nearly the optimum efficiency factor for bed occupancy.)

General Calculation of Bed Occupancy.

$$\text{Percentage Bed Occupancy} = \frac{\text{Occupied Bed-days}}{\text{Available Bed-days}} \times \frac{100}{1}$$

$$= \frac{T \times LS}{(T \times TI) + (T \times LS)} \times \frac{100}{1}$$

$$= \frac{LS}{TI + LS} \times \frac{100}{1}$$

The occupancy of the 30-bed department mentioned earlier would thus be 80 per cent.

$$\text{viz., } \frac{(LS)16}{(TI)4 + (LS)16} \times \frac{100}{1}$$

In considering variability we note that this index of performance would equally well describe a department with TI = 2 and LS = 8, or another with TI = 8 and LS = 32. (Hay, *The Hospital,* March, 1954).

In practice a hospital is found to be busy, but not over-crowded, at a bed occupancy factor of about 85 per cent.

Case-Load Study No. 1

In carrying out a case-load study of comparable areas outside the ambit of the University Medical School, there are several ways of expressing the factors involved.

For example, we might in simple terms say that the number of beds required $= \frac{\bar{d} \times a}{365}$

where $\bar{d}$ = average length of stay in days.

a = estimated desirable number of cases requiring treatment each year,

where "a" is, in general terms, equal to the total discharges plus deaths, plus or minus the increase or decrease in the waiting-list in a special category at the end of the year.

or Critical Number of beds $= \frac{\bar{d} \times a}{365 \times \text{O.R.}}$ (when there is a definite waiting-list).

Case-Load Study No. 2.

In more precise terms assume

W_x = Number on the waiting-list at the beginning of the year x.

W_{x+1} = Number on the waiting-list at the beginning of the year x + 1.

D_x = Number discharged during the year x.

$\bar{d}$ = Average duration of stay in hospital in days.

a_{x+1} = Number of patients who would have been admitted by the physician (exclusive of any on the waiting-list) if beds were available during the year x + 1.

O.R. = Occupancy Rate.

At first glance

B = Number of beds required in the year x + 1.

$= \frac{\bar{d}(W_{x+1} + a_{x+1})}{365 \times \text{O.R.}}$

(We have to estimate a_{x+1} and we must assume that $a_{x+1} = a_x$ when a_x is defined as a_{x+1} except that for x + 1 we read x.)

If in fact there were A_x admissions during the year x and none of the patients making up W_x are included in W_{x+1} then

$$a_x = (A_x - W_x) + W_{x+1}$$

If the number of beds available throughout the year x is constant then

$$A_x = D_x$$

$$\text{so } a_x = (D_x - W_x) + W_{x+1}$$

so that substituting $a_x = a_{x+1}$ in the first equation gives

$$B = \frac{\bar{d}(2W_{x+1} + D_x - W_x)}{365 \times O.R.}$$

if $W_x = W_{x+1}$ then this gives

$$B = \frac{\bar{d}(W_{x+1} + D_x)}{365 \times O.R.}$$

I am advised by Dr. E. A. Cheeseman, who kindly discussed the algebraic expressions with me that it would be better to think in terms of

$$B = \frac{\bar{d}(W_{x+1} + D_z) + (W_{z+1} - W_z)}{365 \times O.R.}$$

where, as with $\bar{d}$, D_z and $(W_{z+1} - W_z)$ are predicted for their trends over a number of years for specific medical conditions, thus reducing to a minimum any possible temporary fallacy due to a "false" waiting-list. A waiting-list can be artificially created through a surgeon going on holiday, or through structural alterations occurring in a hospital, or for several other reasons.

You will see, however, that algebraically we are back to the position rather more roughly defined in Case-Load Study No. 1 above.

Using the simplified formula of

$$B = \frac{\bar{d}(W_{x+1} + D_x)}{365 \times O.R.}$$

the following table was compiled showing the effect of such a calculation on the position of areas where acute general bed need can be closely calculated and compared and where maternity beds were regarded as a local need. All mental, chronic sick and tuberculosis beds are discounted.

Case-Load Study No. 3

Hospital Group	Population	Waiting List	$\frac{\bar{d}(W_{x+1} + D_x)}{365 \times O.R.}$	Beds Proposed	Beds Calculated per 1,000 Population	Beds Proposed per 1,000 Population	Present Beds
A	70,000	205	327	356	4.7	5.1	328
	(Close to Belfast and Larne Development)						
B	30,000	254	160	173	5.3	5.7	173
	(Isolated General Hospital Area not catering for Chronic Sick)						
C	71,000	500	338	380	4.8	5.3	379
	(Agreed that 50 beds excess has been planned here because of poor existing accommodation)						

Clearly B., in making no claim for much additional accommodation, is very well served as it stands, while A. is over-provided in the proposals and C. is over-provided, but the over-provision has been clearly seen and deliberately undertaken in order to erase old and inadequate existing accommodation to the tune of fifty beds as soon as the new accommodation is available.

Consideration of Special Beds.

In considering the local or area needs for special beds, as distinct from general medicine and surgery, one encounters very variable data.

Maternity.

At one time, not so long before the second world war, it was an accepted fact that for ideal care, free from the worry of possible post-obstetric disabilities of various kinds, a lying-in period of two to three weeks was desirable. Averaging this out at sixteen days, together with a two-day turnover interval, each lying-in bed might be expected to deal with twenty patients per annum.

Based on the total number of local births, live and still, during the year, and adding one-third of the total area needs for antenatal work, together with some few isolation beds, it was possible to say how many beds as a maximum figure each area would require.

Maternity beds are very expensive to maintain and, if domiciliary midwifery is entirely devoid of risk, the need for hospitalisation, apart from obstetric emergencies and complications, becomes a social one rather than one of medical or scientific interest.

In Northern Ireland we decided as a matter of policy to allow for 50 per cent. of all births taking place in hospital in every area outside Belfast, and to allow for this plus additional beds to meet with the need for major obstetric complications in the Royal Maternity Hospital, Belfast. The results are quite interesting.

We found that one-third of the total area requirement was unnecessary for antenatal work and that one-sixth was a more realistic figure, plus about one twenty-fifth for isolation purposes. However, we allowed the ideal of twenty patients per bed per annum to remain. (It should also be borne in mind that modern assessment of antenatal requirements would probably allow a slightly larger proportion of beds depending on local need and not on lying-in beds.)

The result was a total need for the Province of something between nine hundred and one thousand beds. In practice, however, two factors were encountered which upset all calculations. Firstly, the Joint Nursing and Midwives Council did not hold fast to the recommended amended lying-in period of ten to fourteen days; and secondly, the obstetricians themselves saw little objection, where conditions were normal and home conditions satisfactory, to discharging a patient at the end of a week.

Thus beds are in places dealing with thirty to thirty-five patients per annum, or in actual fact, coping with 75-80 per cent. of all local births.

A more realistic and acceptable bed turnover would be 24-26 patients per lying-in bed per annum.

Thus the statistical method of assessment is simple, but the basis of assessment requires further definition in the light of modern medical policy.

Tuberculosis.

The position here is quite different. The demand for beds is lessening and total ascertainment would appear to have almost been reached, because notifications of new cases are showing a downward trend, as are mortality rates. Mortality rates in Northern Ireland are the lowest ever recorded at 12.9 per 100,000 population for 1955 (similar to England and Wales), and so we can make certain assumptions about future demands for beds.

If every case notified in a year was admitted to hospital and remained there for one year, the total number of beds would equal the total notifications per annum.

The number of beds required therefore will vary according to the following formula.

B = Total annual notifications × proportion of such cases requiring hospitalisation × average stay in years.

$$= NT \times \frac{NH + NR}{NT} \times \frac{7}{12}$$

where NT = Total new cases of pulmonary tuberculosis in the year.
NH = New notifications which must be admitted to hospital.
NR = Return cases requiring re-admission.

The following two illustrations will test the validity of this.

Northern Ireland notifications for 1955 = 1,167
Total admissions to hospitals for P.T. = 2,120

$$B = NT \times \frac{NH + NR}{NT} \times \frac{7}{12}$$

$$= 1{,}167 \times \frac{2{,}120}{1{,}167} \times \frac{7}{12}$$

$$= 1{,}237$$

= 0.88 bed per 1,000 population.
or = 1,100 beds per 1,000 notifications approximately.

In 1955 total existing P.T. beds = 1,400
Total empty beds = 165*
Effective beds = 1,235
∴ Under-provision = 2 beds

*The reason why a small waiting-list exists in spite of empty beds is because patients now elect to wait until a bed is vacant nearer home rather than accept a bed some distance away, though immediately available.

In the second illustration we noted that experience pointed to 50 per cent. of all new notifications having to be sent to hospital in the year in which notified.

Now, as already seen, in 1955,

$$NH + NR = 2{,}120$$

$$NH = \frac{NT}{2}$$

$$= 584$$

$$\therefore \quad NR = 1{,}536$$

(Does this indicate an excessive break-down rate, even allowing for re-admissions from surgical units and transfers between hospitals? Does it indicate that the length of stay is too short or that the treatment is ineffective, or after-care inadequate?)

New notifications in 1956 = 1,100
Beds now required = 1,210
Effective beds were = 1,270
$\therefore$ Over provision in 1956 = 60 beds

This, in effect, is exactly the present state of affairs and the (General) Hospitals Authority has asked the Tuberculosis Authority to release two thirty-bed units for return to general medicine and surgery.

The position is far from simple, however. Can we estimate the annual run-down of beds over a period of years and forecast the position say in 1964, as with the general medicine and surgical beds?

For Northern Ireland in 1955, 1,237 beds were required against 1,210 in 1956, or a decrease of 27 beds in the year. If this were maintained, our need in 1964 would be for 27 x 7 or 189, or roughly 200 fewer beds. So we have compromised by allowing for one thousand beds for all purposes connected with tuberculosis in 1964.

Many variables exist, and a good deal of further study of this problem could usefully be undertaken.

Orthopædic Beds.

A breakdown of Northern Ireland waiting-lists in 1955 showed that cases could be grouped according to the following classifications :—

1. Short-stay cases—General Surgeon.
2. Intermediate-stay cases—50 per cent. to General Surgeon.
3. Long-stay cases—Orthopædic Surgeon.

A clinical classification of each group was possible into

(a) Traumatic cases—Fractures, Dislocations, etc.;
(b) Non-traumatic cases—Disease, Congenital Defects, etc.

In practice Groups 1 and 2 = Group 3. All of Group 3 and one-half of Group 2 go to the Teaching Centre, leaving one-half of Group 2 and all of Group 1 to be distributed locally on a basis of forty beds per area of 120,000 population, in accordance with N.H.S. Publications on Consultant Services.

Mental, Chronic Sick.

The need for mental beds remains fairly constant at four beds per thousand of population, though variables are the number of voluntary patients seeking admission each year, and the increasing numbers of genetic disorders and senile cases.

Special care cases are difficult to assess and even at 1,000 to 1,500 places for Northern Ireland for 1964 the psychiatrists are far from satisfied.

Chronic sick beds can be reckoned at a fairly steady 1.25 beds per thousand of population, the variable being mainly the actual number of old people requiring hospital care as opposed to care in a welfare hostel. In County Antrim, where a satisfactory balance has now been struck, there are 360 beds in homes and hostels for the old and frail, as opposed to 440 beds in geriatric or aged chronic sick wards of hospitals. The total population of County Antrim of persons aged 60 years and over is approximately 32,000.

At the Teaching Centre the following specialities are provided exclusively to cater for the needs of the entire Province :—

Infectious Diseases, Radiotherapy, Plastic Surgery, Neurology, and Neurosurgery.

Mental health and special care beds and tuberculosis beds are mainly housed in special *ad hoc* hospitals.

Time will not allow of a full description of the calculations of bed requirements for these, but it will be seen that even in the Teaching Centre for general acute beds, leaving out mental, tuberculosis, geriatric, and highly specialised provincial beds, the present demand is said not to be met at 4.3 beds per thousand, but should be met by 1964 at 4.9 beds per thousand of population. The point at issue, in view of our case-load studies, is whether or not we are over-providing a little. Certainly, at no point, do we appear to be under-providing, and an important factor as yet undetermined is provision against obsolesence of present hutted accommodation. Even a saving of 0.2 bed per thousand of population might result in a saving of over £1,000,000 capital outlay and corresponding saving in running costs in a province of the size of Ulster.

I am indebted to Mr. E. H. Jones, O.B.E., Secretary of the Northern Ireland Hospitals Authority, for permission to utilise comparative statistical returns for the hospitals; and to Dr. Eric Cheeseman, of the Department of Social and Preventive Medicine of the Queen's University of Belfast for helpful statistical advice, though I absolve him from all responsibility for my own mathematical shortcomings.

Pituitary Choristoma: A Survey of Thirty Cases

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Based on material accepted for the Finnegan Scholarship of the Queen's University of Belfast

FROM time to time in post-mortem reports, a small tumour of the posterior pituitary stalk, called a choristoma, is an incidental finding. Looking through the reports of the Pathology Department of Queen's University, it was possible to find thirty cases of choristoma over the past twenty years.

The tumour was first noticed by Boyce and Beadle in 1893, but was called "Choristoma" by Sternberg in 1921. It is also called a granular cell myoblastoma.

Histologically, it is a collection of large granular cells, with peripheral nuclei and is now considered by most investigators to be of ectodermal neurogenic origin (Sternberg, 1921; Priesel, 1922; Simonds and Brandes, 1925). A small tumour about 1 mm. in diameter and often multiple, it is generally regarded as being benign, although malignant changes are described (Ross, Millar and Foote, 1952).

Pituitary choristomata have no known function.

MATERIAL AND METHODS.

Tabulating the cause of death in the thirty cases of choristoma :—

TABLE I.

CAUSE OF DEATH	MALE	FEMALE	TOTAL
Malignant Hypertension	1	–	1
Essential Hyptertension	4	1	5
Intracranial Hæmorrhage	–	2	2
Chronic Pyelonephritis	2	2	4
Accidental	1	1	2
Carcinoma of Prostate	2	–	2
Carcinoma of Bronchus	–	2	2
Carcinomatosis	–	1	1
Carcinoma of Cæcum	–	1	1
Syphilis	1	–	1
Aortic Aneurysm	1	–	1
Congenital Cystic Kidneys	1	–	1
Cholesteatoma	1	–	1
Chronic Nephritis	1	–	1
Bacterial Endocarditis	–	1	1
Cushing's Disease	–	1	1
Astrocytoma	–	2	2
Multiple Myeloma	–	1	1
TOTAL	15	15	30

This shows a marked preponderence of hypertension and related diseases (intracranial hæmorrhage, chronic pyelonephritis, cystic kidneys, chronic nephritis and Cushing's disease).

In order to determine the significance of these results, a further series of thirty cases in which choristomata were not found was compiled from the post-mortem reports. A post-mortem performed as closely as possible in time to each choristoma case was selected, provided the age was similar, the sex was the same and both autopsies were carried out by the same pathologist.

In this manner the following control series of thirty cases was compiled. There is no similar hypertensive trend.

TABLE II.

Cause of Death	Male	Female	Total
Intracranial Hæmorrhage	1	3	4
Chronic Bronchitis with Emphysema	4	–	4
Coronary Thrombosis	1	–	1
Acute Nephritis	1	–	1
Bacterial Endocarditis	1	–	1
Carcinoma Stomach	1	–	1
Carcinoma Colon	1	–	1
Carcinoma Uterus	–	2	2
Pleural Endothelioma	1	–	1
Pituitary Tumour	1	–	1
Myelomatosis	–	1	1
Brain Abscess	1	–	1
Meningitis	–	1	1
Bronchiectasis	–	1	1
Pneumonia	–	1	1
Chronic Pyelonephritis	–	1	1
Simmond's Disease	1	1	2
Nephrosis	–	1	1
Hepatic Cirrhosis	–	1	1
Rheumatic Heart Disease	–	1	1
Accidental	–	2	2
Total	14	16	30

A more detailed examination of each of the sixty cases was carried out, obtaining information from both the post-mortem records and the patients' clinical records prior to death. Incomplete information on five choristoma cases (Cases 51 to 55) made it necessary to exclude them from part of the investigation.

Each case was reviewed for any evidence of hypertension. The heart weight and left ventricular size (pathological, clinical and radiographical) were the main criteria upon which selection was made. The incidence of hypertension in both the choristoma and the control series was compared using the "χ^2" test, the actual blood pressure recordings being compared by means of a "t" test.

The age and sex distribution of an average year's post-mortems was compared with that of the choristomata.

Hypertension. RESULTS.

Considering twenty-five choristoma cases (Cases 1-25).

TABLE III.

CHORISTOMA GROUP.

CASE	SEX	AGE	BLOOD PRESSURE MM. HG.	HEART WEIGHT GRAMS	L.V. HYP.	X-RAY	CAUSE OF DEATH
1	M	55	280/170	610	Yes	—	Malignant Hypertension.
2	M	76	110/70	520	Yes	--	Essential Hypertension.
3	M	36	140/80	300	No	—	Accidental.
4	M	70	—	700	Yes	—	Syphilis.
5	M	63	120/60	540	Yes	—	Abdominal Aneurysm.
6	M	35	260/170	500	Yes	—	Chronic Pyelonephritis.
7	M	40	195/125	1,000	Yes	L.V. enlarg.	Polycystic Kidneys.
8	M	28	Normal	250	No	—	Cholestætoma.
9	M	40	230/110	600	Yes	—	Chronic Glomerulo-nephritis.
10	M	75	190/100	550	Yes	N.A.D.	Essential Hypertension.
11	M	52	170/100	425	Yes	—	Essential Hypertension.
12	M	70	165/90	390	No	N.A.D.	Essential Hypertension.
13	M	67	130/85	410	No	—	N.G. Prostate.
14	M	66	140/110	375	Yes	—	N.G. Prostate.
15	F	52	110/90	280	No	N.A.D.	N.G. Bronchus.
16	F	31	160/85	575	—	—	Acute bacterial endocarditis.
17	F	55	210/90	500	Yes	—	Cushing's Disease.
18	F	35	95/50	275	No	N.A.D.	N.G. Bronchus.
19	F	71	160/100	300	No	N.A.D.	N.G. Brain.
20	F	56	270/140	520	Yes	L.V. Hyp.	Chronic Pyelonephritis.
21	F	71	140/65	420	Yes	—	N.G. Cæcum.
22	F	58	120/80	285	No	—	N.G. Brain.
23	F	51	280/150	300	Yes	—	Chronic Pyelonephritis.
24	F	55	110/90	220	No	L.V. enlarg.	Myelomatosis.
25	F	66	190/100	570	Yes	—	Essential Hypertension.
51	F	17	150/90	—	—	—	Accidental.
52	F	59	200/120	—	—	—	Subarachnoid Hæmorrhage.
53	F	77	120/70	—	—	—	Intracranial Hæmorrhage.
54	F	53	150/100	360	No	—	Carcinomatosis.
55	M	71	—	560	Yes	—	Chronic Pyelonephritis with N.G. Prostate.

TABLE IV.

Control Group.

Case	Sex	Age	Blood Pressure mm. Hg.	Heart Weight Grams	L.V. Hyp.	X-Ray	Cause of Death
26	M	54	170/100	1,000	Yes	—	Acute Nephritis.
27	M	77	140/70	500	No	—	Coronary Thrombosis.
28	M	42	95/55	250	No	—	Brain Abscess.
29	M	62	135/90	310	No	Normal	Chronic Bronchitis.
30	M	64	150/90	250	No	Normal	Pleural Epithelioma.
31	M	45	250/130	500	Yes	—	Subarachnoid Hæmorrhage.
32	M	41	150/50	1,000	Yes	L.V. enlarg.	Subacute Bacterial Endocarditis.
33	M	37	110/75	250	No	Normal	N.G. Colon.
34	M	39	140/90	500	No	—	Pituitary Tumour.
35	M	68	135/70	490	No	Normal	Chronic Bronchitis.
36	M	55	145/85	320	No	Normal	Simmond's Disease.
37	M	70	170/105	750	Yes	—	Chronic Bronchitis.
38	M	71	130/80	350	No	Enlarged	N.G. Stomach.
39	M	62	120/80	350	No	Normal	Chronic Bronchitis.
40	F	54	140/90	340	No	—	Rheumatic Heart Disease.
41	F	32	125/85	410	No	Effusion	Acute Nephritis.
42	F	64	160/75	350	No	L.V. enlarg.	Myelomatosis.
43	F	40	125/75	320	No	—	Accidental.
44	F	82	100/85	400	Yes	L.V. enlarg.	N.G. Uterus.
45	F	58	150/90	210	No	Normal	Simmond's Disease.
46	F	77	200/120	400	Yes	L.V. enlarg.	Pneumonia.
47	F	55	160/90	190	Yes	—	Cirrhosis.
48	F	53	140/70	420	Yes	—	Nephrosis.
49	F	62	—	250	No	Normal	Bronchiectasis.
50	F	76	200/110	600	Yes	—	Subarachnoid Hæmorrhage.
56	F	14	150/?	Normal	No	—	Accidental.
57	F	48	160/90	—	—	Normal	Subarachnoid Hæmorrhage.
58	F	75	170/95	—	—	—	Intracranial Hæmorrhage.
59	F	60	180/100	300	No	—	N.G. Cervix.
60	M	80	150/90	400	No	L.V. enlarg.	Meningitis.

TABLE V.

	Hypertensive	Normotensive
Male	9	5
Female	5	6
Total	14	11

Comparing this with twenty-five control cases (Cases 26-50).

TABLE VI.

Using a χ^2 test on the total cases (i.e., male and female) $0.02 < P < 0.05$

	Hypertensive		Normotensive
Male - -	3	...	11
Female - -	3	...	8
Total - -	6	...	19

numbers used were too small for sex differentation in this test.

Blood Pressure.

The means were 180/100 and 150/85 mm. Hg. for choristoma and control groups respectively.

By means of a "t" test—

$0.02 < P < 0.05$ for systolic pressure.

$0.01 < P < 0.02$ for diastolic pressure.

Sex.—There was no significant difference.

Age.—Compared with age distribution of an average year's post-mortems.

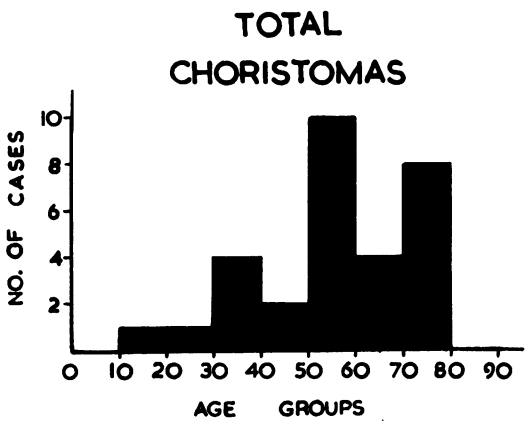

Fig. 1

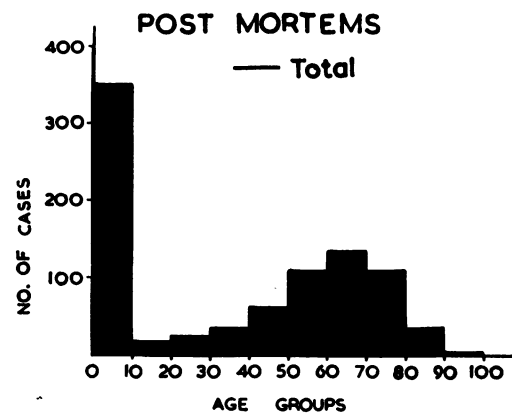

Fig. 2

Discussion.

Although much has been written about the pathology and pathogenesis of choristomata (Shanklin, 1953), there has been no attempt to offer an explanation of their relation to clinical medicine. In reviewing this series this relationship was investigated.

It was only possible to find a small number of choristomata in some 15,000 pathology reports over the past twenty years, but this cannot be regarded as their true frequency. A tumour of this size might easily be missed on routine pituitary section, and in not all post-mortems was such a section taken. It might well be assumed that 6.45 per cent. frequency of Luce and Kernohan (1955) is a more accurate figure.

No sex difference was found, a result confirming the findings of Luce and Kernohan (1955).

The age distribution is interesting. Although the greatest number of post-mortems were performed in the 0-9 age group (almost entirely due to stillbirths and neonates), no choristomata were found here, whereas, the other groups had a proportionate scattering. No case has previously been recorded under 20 years, whereas one in a 17-year-old girl (Case 51) was found in this series.

Statistical analyses of the choristoma and control series show a significant increase in hypertensives in the former, confirmed by a "t" test in the actual blood pressure recordings. However, the number of cases in this survey was small and verification of these results using larger number of cases is required.

The question now arises of whether this tumour is related to the cause of the hypertension, or is simply an effect of it. As seen in Table I, not only does it frequently occur with the hypertensions of unknown ætiology, but it is found more commonly associated with diseases, which themselves give rise to hypertension (chronic pyelonephritis, Cushing's disease, polycystic kidneys, etc.). It, therefore, seems more probable that the tumour results, in a manner not understood, from the effects of long-standing hypertension. Could this tumour have an endocrine function? Microscopically, it appears glandular and might produce some hypotensive agent in an effort to maintain normotension. Clearly, further investigation is required into this problem.

Summary.

Thirty cases of pituitary choristoma were investigated, and their frequency, age and sex distribution, and association with hypertension, are discussed.

I wish to thank Professor J. H. Biggart, C.B.E., D.SC., M.D., F.R.C.P., for use of the pathology files and slides, and for criticism and advice, Professor G. M. Bull, M.D., F.R.C.P., for his help and encouragement, and Miss B. Mager for typing this paper.

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Clinical Trial of Butylphenamide in the Treatment of Certain Varieties of Ringworm

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THIS report concerns the topical use of butylphenamide* in the treatment of some of the common varieties of ringworm of the scalp and body. Laboratory data dealing with fungicidal effect "in vitro" and the absence of signs of toxicity in experimental animals suggested that butylphenamide would be a more valuable fungicide than any of the many others available. Our clinical impression is that this is so.

Ideally all clinical trials should be carried out under strict statistical control, but from a purely practical point of view this is not possible when dealing with local applications which might be of value in the treatment of the various kinds of ringworm. This is so because the duration and response to treatment will vary with the type of fungus responsible and with the part of the body affected, and possibly with the type of skin which the patient has, ringworm being on the average rather more long lasting on a dark "hairy" skin than on a fair skin. The age of the patient may influence results, since we know that ringworm in babies clears relatively quickly. There is also good reason to believe that some fungi tend to lose virulence on transfer from one individual to another—and this will influence treatment results. Again, some patients will produce inflammatory and quickly clearing lesions, while others infected by the same fungus will react with chronic and indolent lesions. Finally, it is rare indeed to see ringworn lesions symetrically distributed on the body, so that using the method of paired comparisons is impractical.

Therefore, no apology is offered for presenting what is little more than a series of clinical impressions regarding the usefulness or otherwise of butylphenamide tincture and ointment. However, the cases included in the trial were limited to those common infections which had been seen so often that one felt that a really beneficial response to treatment would be noticeable when the results of a series were compared with past results—and colour photographs were taken of all cases at each attendance. Only those patients who attended as and when required until their ringworm was quite clear are included.

Derivatives of salicylic acid and of salicylamides are known to possess fungistatic activity. Good results have been claimed from the use of preparations of sali-

*A preliminary report on the use of butylphenamide as a fungicide was read, in co-operation with Dr. John Krafchuk, at the Eleventh International Congress of Dermatology, in Stockholm, in August, 1957.

cylanilide and carbowax for the treatment of Microsporum audouini scalp infections (Schwartz, et al., 1946, Brain, et al., 1948, and Haber, et al. 1949), but Beare and Cheeseman (1951) were unable to confirm these results "when the spontaneous cure rate of M. audouini tinea capitis is borne in mind." A number of substituted salicylamides were synthesized by Jules, et al. (1956). N-n-butyl-3-phenylsalicylamide (butylphenamide) was later screened for its microbiological activity by Hok, et al. (1956). These authors reported that this substance, when compared to other anti-fungal agents commonly used in treatment appeared to be more active. They also produced evidence showing that Trichophyton mentagrophytes and M. audouini did not develop resistance to the substance. The toxicity of butylphenamide in animals has been studied by Seeberg, et al. (1956) who reported that large amounts of butylphenamide applied to the skin of rabbits every day for a month did not cause any significant change in blood, urine or gross or microscopic appearance of tissues. They also reported that very little butylphenamide was absorbed from the gastro-intestinal tract of animals. Krafchuk (1956) and, independently, Keddie, et al. (1956) have reported encouragingly on the use of butylphenamide in skin eruptions due to dermatophytes. Krafchuk, in addition, found an antipruriginous effect in certain skin diseases which were not due to ringworm infections, such as lichen simplex chronicus, atopic eczema and contact dermatitis.

The trial was started in June, 1956, and all cases of ringworm, except infections due to M. audouini, seen from this date to March, 1957, were included initially in the trial, and were given some preparation of butylphenamide. Children with M. audouini infections were excluded because, in my opinion, the essential thing here is to render the child non-infectious at the earliest possible opportunity, and for this X-ray epilation is necessary. Since all our M. audouini infections now come from foreign lands, it is our ambition to keep our area free of this infection. Only those patients who had attended regularly when requested and to complete cure, and who had a common variety of infection, the response to treatment of which we could reasonably compare with past results, were included. Eventually, the final analysis consisted of forty-three cases of infections due to Microsporum canis, Trichophyton sulphureum and Trichophyton discoides. In all these cases the causative fungus had been isolated by mycological culture.

Clinical Results.

(a) Microsporum Canis.

There were eleven cases of scalp infection. One of eight weeks' duration developed a kerion after two weeks' treatment, and butylphenamide was stopped. Another of three weeks' duration developed a moderately severe inflammatory reaction after two weeks' treatment. His condition was clear five weeks later, but butylphenamide was discontinued for ten days at the height of the reaction.. In a third case after five weeks' treatment it was considered necessary to carry out X-ray epilation, since there was no change in the boy's condition and there was some danger of spread of infection to his many brothers and sisters. In a fourth, X-ray epilation had been recommended, but the parents refused to give consent,

and this was after butylphenamide had been used for a period of eleven weeks without clinical change. All these four cases were regarded as failures. Of the other seven cases, one of six weeks' duration showed no inflammatory reaction and cleared in seven weeks, three showed mild inflammatory reaction and cleared in periods of four weeks (initial duration one week), five weeks (initial duration three weeks), and seventeen weeks (initial duration one week). Two showed a moderate inflammatory reaction and cleared in periods of eight weeks (initial duration two weeks), and seven weeks (initial duration three weeks), respectively, and one showed a severe reaction and cleared in four weeks (initial duration three weeks).

TABLE 1.

MICROSPORUM CANIS.

Case No.	Sex	Age	Parts affected	Degree of reaction (0 — ++++)	Duration before treatment (weeks)	Duration of treatment (weeks)	Assessment of result
1	F	10	Scalp	+ + + +	8	2	Failure
2	M	11	Scalp	+ + +	3	5	Failure
3	M	4	Scalp	+	1	5	Failure
4	M	5	Scalp	+	±6	11	Failure
5	F	5	Scalp	0	6	7	Good
6	M	9	Scalp	+ +	1	4	Good
7	F	8	Scalp	+ +	3	5	Good
8	M	6	Scalp	+ +	1	17	Indefinite
9	F	3	Scalp	+ + +	2	8	Good
10	F	3	Scalp	+ + +	3	7	Good
11	M	10	Scalp	+ + + +	3	4	Good
12	F	37	Body and limbs	+ + +	1½	4	Good

In addition, there was one adult patient with a very severe tinea corporis due to M. canis affecting large areas on trunk, arms and legs. Her condition, present for ten days before treatment, cleared completely in another four weeks.

It is suggested that these results in M. canis infections are as good, if not better than one would get from any other type of local application.

(b) Trichophyton Sulphureum.

There were eight cases of tinea capitis. Four non-inflammatory cases we regarded as failed, one (of two weeks' duration) after twenty-six weeks' treatment, when X-ray epilation had to be carried out; the second (of four weeks' duration) after twenty-six weeks' treatment, when the condition was really still active; the third (of one week's duration) after nine weeks' treatment, when X-ray epilation had to be carried out, and the fourth (one week's duration) after six weeks' treatment, when there was no change and X-ray epilation was carried out (an associated tinea corporis cleared within six weeks). One boy who had had a mild infection for five weeks cleared in a further eleven weeks' treatment. Two children had had the

infection for five months; one of these cleared in ten weeks, the other in twelve weeks; the former was non-inflammatory, but after two weeks' treatment developed a moderately severe inflammatory reaction, the second was non-inflammatory at the beginning but later developed a kerion. We would regard these cases as failures. The last case had an infection of thirty-one weeks' duration but had a moderately severe reaction from the start. He cleared with three weeks' treatment (probably good luck, since the infection was likely to be clearing by this time any how).

TABLE 2.

TRICHOPHYTON SULPHUREUM.

Case No.	Sex	Age	Parts affected	Degree of reaction (0 — ++++)	Duration before treatment (weeks)	Duration of treatment (weeks)	Assessment of result
1	M	10	Scalp	0	2	26	Failure
2	M	9	Scalp	0	4	26	Failure
3	F	8	Scalp	0	1	9	Failure
4	F	9	Scalp	0	1	6	Failure
5	M	12	Scalp	+ +	5	11	Failure
6	F	6	Scalp	+ + +	20	10	Failure
7	F	10	Scalp	+ + + +	20	12	Failure
8	M	8	Scalp	+ + +	31	3	Questionable
9	F	11	R. neck	+ + +	1	4	Good
10	F	8	R. arm	+ + +	½	4	Good
11	M	10	L. neck	+ +	1	3	Good
12	M	10	R. arm	+ +	1	4	Good
13	F	8	L. arm	+ +	2	4	Good
14	M	10	R. cheek and ear	+ +	8	4	Good
15	F	12	Front of chest	+ +	6	6	Good
16	F	12	R. neck	+ +	11	3	Good

There were eight cases of tinea corporis of which two had a moderate reaction; they both cleared within four weeks. The other six had mild reactions. One (one week's duration) cleared in three weeks, three (durations one week, two weeks and eight weeks) in four weeks, one in six weeks (duration six weeks) and one in eleven weeks (duration three weeks). We would regard these eight cases as being successfully treated by butylphenamide.

(c) Trichophyton Discoides.

Five cases of tinea capitis all of which showed severe kerions. Three children with durations of one month, two weeks and ten days, cleared in five weeks, six weeks, and two weeks, respectively, and one with a duration of three months cleared in three weeks. The other boy had an exceptionally severe tinea capitis of three weeks' duration. Butylphenamide was only one of many applications used in his long

illness of sixteen weeks' duration, from the first time seen, but it was one of the most useful. These results were considered good.

Three cases of tinea barbæ were included. One of eight weeks' duration cleared in three weeks, one of two weeks' duration cleared in seven weeks, and one of four weeks' duration cleared in seven weeks. All were severely inflammatory. These results are good.

There were eight cases of tinea corporis. One severe reaction in a boy of ten and of four weeks' duration, cleared in two weeks. Five had moderate reactions, and

TABLE 3.

TRICHOPHYTON DISCOIDES.

Case No.	Sex	Age	Parts affected	Degree of reaction (0 — ++++)	Duration before treatment (weeks)	Duration of treatment (weeks)	Assessment of result
1	M	5	Scalp	+ + + +	4	5	Good
2	M	9	Scalp	+ + + +	2	6	Good
3	M	6	Scalp	+ + + +	1½	2	Good
4	M	8	Scalp	+ + + +	12	3	Good
5	M	4	Scalp	+ + + +	3	16	Useful in a difficult case
6	M	18	Beard	+ + + +	8	3	Good
7	M	35	Beard	+ + + +	2	7	Good
8	M	42	Beard	+ + + +	4	7	Good
9	M	10	R. shoulder R. leg	+ + + +	4	2	Good
10	M	9	L. knee	+ + +	3	2	Good
11	F	2	Back of trunk	+ + +	3	2	Good
12	F	17	L. shoulder L. arm	+ + +	1	4	Good
13	F	12	R. shoulder R. back	+ + +	1	4	Good
14	F	13	Upper back	+ + +	3	3	Good
15	M	22	L. back of hand	+ + + +	2	3	Developed pompholyx
16	F	19	Face	+ + +	2	3	Good
			R. arm R. shoulder	+ + +	1	2	(did not treat face initially)

two of these, each of three weeks' duration, cleared in two weeks, two, each of one week's duration, cleared in four weeks, and one of three weeks' duration cleared in three weeks.

Two further cases of tinea corporis were of some interest. One of two weeks' duration, affecting the back of the hand, seemed to do very well after one week's treatment, but then the patient developed a pompholyx reaction on both hands.

This settled in a further fortnight. And another patient, with ringworm of one week's duration affecting the face, the right arm and shoulder, applied tincture of butylphenamide to the arm and shoulder and after one week there was much improvement. The face, however, had became worse and on questioning it turned out that she had not put butylphenamide on to her face, having mistaken the instructions which she was given. After a further three weeks of treatment her condition was completely clear.

Conclusions.

1. *Microsporum Canis* : Lesions of the non-hairy skin clear quickly and, I believe, quicker than they would do so spontaneously. Lesions of the scalp cleared quickly in about half the cases treated, but in the other half there did not appear to be any beneficial effect.

2. *Tirchophyon Sulphureum* : There was nothing gained from the treatment of the scalp infections, which in this particular series were all non-inflammatory. However, lesions on non-hairy skin cleared quickly.

3. *Trichophyton Discoides* : Here there were most useful results, and the duration of these inflammatory infections appeared to be substantially shortened. All the scalp infections in the series, except one, cleared in six weeks, and in the case which did not clear, butylphenamide was the most useful fungicide of several used. The beard infections cleared within seven weeks, and the lesions of the non-hairy skin within five weeks.

Adverse Effects : There were no examples of epidermal sensitivity nor of primary irritation in this series, nor indeed in any of the other patients treated with butylphenamide but not included in those analysed here. Krafchuk (1957) has not yet seen any example of sensitivity. However, the tincture occasionally stings and young children may object to its application.

Unfortunately, butylphenamide causes a greenish-blue fluorescence in Wood's light, which makes examination of scalp infections due to microspora difficult but, of course, this fluorescence is not limited to the hair.

Summary.

Butylphenamide is a most useful fungicide without any adverse effects. One's clinical impression is that it is superior to any of the older preparations in common use. However, there was no evidence obtained from this trial that it is likely to have any part to play in the treatment of non-inflammatory scalp ringworm.

My colleague, Dr. Ivan H. McCaw, kindly referred some of the cases; Dr. Jacqueline Walker, of the London School of Hygiene and Tropical Medicine, did all the mycological work and identified the fungi; Mr. Ronald Woods, Medical Photographer, Royal Victoria Hospital, took a great many photographs; Dr. John Krafchuk, of New Orleans. U.S.A,, suggested the trial and arranged with Dr. Edwin McLean of Cutter Laboratories Limited, U.S.A., that supplies of butylphenamide be sent. The preparation was supplied under the trade name "Bynamid" (Bynamid Ointment is a five per cent. mixture of Bynamid in a carbowax-polyethylene glycol base; Bynamid Tincture is five per cent. solution of Bynamid in a mixture of isopropyl alcohol, acetone prophylene glycol, and Aerosol).

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REVIEW

AORTOGRAPHY: ITS APPLICATION IN UROLOGICAL AND SOME OTHER CONDITIONS. By W. Barr Stirling, Ch.M., F.R.C.S.(Ed.), F.R.F.P.S.G. (Pp. vii + 291; figs. 155. 50s.) Edinburgh and London: E. & S. Livingstone, 1957.

In this volume the author has summarized his personal experience of five hundred aortograms performed for the investigation of various renal conditions.

The opening chapters give an historical survey of the methods used, a discussion of the surgical anatomy of the renal arteries, and a description in detail of the particular technique used by the author.

The book is admirably illustrated with aortograms showing the abnormal vascular patterns produced in various lesions. Interesting chapters are given on the interpretation of the films and of the hazards and complications that may result from these investigations.

The book gives a very comprehensive survey of the subject, and it certainly should be in the library of every surgeon, especially those surgeons interested in urological work. J. M. M.

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An Evaluation of Dextrotest Method of Blood Sugar Estimation

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DEXTROTEST is a simple, side room method of blood sugar estimation produced by Messrs. Ames Ltd. It is basically a convenient form of a copper reduction technique, measuring the total reducing substances in blood. It consists, in brief, in adding 1 ml. of blood to 2 ml. of water in which a tablet containing sulphosalicylic acid has been dissolved. The proteins having been precipitated, the mixture is filtered, and to 1 ml. of filtrate is added a second tablet containing copper sulphate and sodium hydroxide. Boiling ensues without the application of external heat, and a colour develops, comparison of which with a scale allows an assessment of the blood sugar to be made.

In the following experiments, all of which were personally performed, distilled water was used as a solvent, and a rubber bung used to occlude the tube in which protein precipitation takes place. This tube is shaken gently to ensure thorough mixing before filtration. A control estimation of the blood sugar of each specimen was performed by the staff of the Biochemical Laboratory of this Hospital, using the method of Folin and Wu (1920).

During a series of twenty preliminary tests, it was noted that the final colour developed through a sequence of colour changes and accordingly the possibility of elaborating the Ames Scale envisaged. Standard Colours (B.S.1.) representing intermediate stages were obtained, and an attempt made to assign to each a numerical value by means of the control estimations. It soon became apparent that this was not feasible, as two blood sugars whose actual values were within five per cent. of each other frequently gave quite different final colours by Dextrotest. It was decided at this stage to record all future Dextrotest estimates as :—

$$<100,\ 100,\ 100+,\ 150,\ 150+,\ 200 \text{ and } 200+ \text{ mg./ 100 ml.}$$

the estimate being made by watching the colour sequence develop, while gently agitating the tube after boiling had ceased. This is of assistance in deciding whether the 100, 150 and 200 mg./100 ml. values have been reached or passed. In all cases, the final colour had developed within thirty seconds of the cessation of boiling, and it is important that the final reading should not be taken earlier than this, otherwise the result may be too low.

Consideration of the above experiment raised the question of whether repeated Dextrotest estimations of a single specimen would give consistent results. To ascertain this, six Dextrotest estimations were performed on each of five specimens

of blood. In no case was there any difference in the estimated Dextrotest values of a particular specimen, although small differences were apparent in the final colours.

A grey colour, reported by Davies and Paley (1957) and Osborn (1957), has been seen on four occasions. It was thought that the use of fluoride anticoagulant in the specimens may have been responsible for this abnormality. To investigate this, a series of sixty samples was examined. A Dextrotest estimation was carried out on 1 ml. of each, and anticoagulant added to the remainder, which was subsequently evaluated both by repeat Dextrotest and by the method of Folin and Wu (1920). In this experiment a grey colour appeared on only one occasion, and then in the absence of anticoagulant, whereas on the other three occasions anticoagulant had been present in the samples, giving rise to a grey colour. On this minimal amount of evidence it would appear that the presence of fluoride anticoagulant is not a factor in the production of the grey colour, and indeed no explanation has yet been advanced for its appearance. It may be that the necessity, as stressed by Moss, for the use of completely fresh tablets, plays a part in this abnormal behaviour of Dextrotest. As a result of the above experiment, it was, however, possible to conclude that the presence of fluoride anticoagulant has no effect on the estimate of the range of blood sugar provided by the Dextrotest technique.

A total of 106 specimens were evaluated both by Dextrotest and the method of Folin and Wu and the results compared. A summary of these is given below. It is possible to state that a blood sugar is less than 100 mg./ 100 ml., but further discrimination within this range is impossible. Here a blue colour is seen which has no green tint, and is easily recognisable. Twenty specimens whose control values were over 200 mg./100 ml. were tested by Dextrotest using .5 ml. of blood and 2.5 ml. of water, thus giving a 2X dilution, making estimations of 200, 300 and 400 mg./ 100 ml. possible. The accuracy of the test at this dilution appears to be comparable to its accuracy in its usual form, and is adequate for practical purposes.

	Dextrotest mg. 100 ml.				
Folin and Wu mg. 100 ml.	<100	100-150	150-200	200+	
<100	26	7	1	1	35
100-150	1	25	9	–	35
150-200	–	–	18	2	20
200+	–	–	4	12	16
					106

Statistical correlation shows no change in the accuracy of the Dextrotest technique from range to range.

Discussion.

It is recommended that Dextrotest be performed strictly in accordance with the maker's directions, taking care to ensure accurate measurement of the fluids used. The accuracy of Dextrotest increases with the operator's experience in its use. It

is inevitable that any procedure which relies on human judgment can never be as accurate as one in which this limitation does not exist, but if one bears in mind its limitations, Dextrotest is a useful addition to the physician's armamentarium. It will be noted that the above table is based on four blood sugar ranges, viz., less than 100 mg./ 100 ml., 100-150 mg./ 100 ml., 150-200 mg./ 100 ml., and 200+mg./ 100 ml. With dilution, this can be extended to include the ranges 200-300 mg./ 100 ml. and 300-400 mg./ 100 ml. One's experience of the test is that attempts at greater accuracy than this are hopeful rather than accurate. It will readily be seen from the table that there is a real danger that one may estimate by Dextrotest a blood sugar value of 150-200mg./100 ml. where, in fact, the actual blood sugar may be over 200 mg./100 ml. For this reason it is suggested that on all specimens, whose Dextrotest value is over 150 mg./100 ml., a repeat estimation should be performed using .5 ml. blood and 2.5 ml. of water. Specimens whose Dextrotest values are over 200 mg./ 100 ml. will normally be repeated using this dilution in any case. In the differential diagnosis of hypoglycæmic coma, Dextrotest may confirm a clinical opinion, but it will not pick out the patient who develops hypoglycæmic reactions when his blood sugar is in fact 80-90 mg./100 ml.

Conclusions.

(a) If one accepts that if a patient's blood sugar is within the 100-150 mg./100 ml. (Folin and Wu) range, his diabetes is adequately controlled, Dextrotest can be used for the long-term control of diabetes, e.g., by general practitioners in remote regions, as the test is fairly accurate within this range.

(b) Dextrotest is very useful for the emergency treatment of diabetic ketosis and hyperglycæmia, when laboratory services are not immediately available, giving one the confidence to prescribe large and frequent doses of insulin without the risk of creating hypoglycæmia. One does not have this confidence when working from urinary sugar estimations alone.

(c) Only in the complete absence of laboratory facilities should Dextrotest be used as a screening test for the diagnosis of diabetes.

(d) Subject to the above limitations, it is the author's opinion, that the control of diabetes could be more accurately effected using this test, than by methods based on estimations of urinary sugar.

My thanks are due to Messrs. Ames Ltd., for a supply of Dextrotest reagents; to Dr. J. A. Smyth, Physician-in-Charge, Sir George E. Clark Metabolic Unit, Royal Victoria Hospital, Belfast, for his encouragement and permission to obtain blood samples from his patients; and to the staff of the Biochemical Laboratory, Royal Victoria Hospital, Belfast, for the performance of the control estimations. My thanks are also due to the Statisticians of the Department of Social and Preventive Medicine, Queen's University, Belfast, for their assistance.

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REVIEWS

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There are four sections:—

1. General considerations, concerned with anæsthesia in any field.
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In section 1 there are chapters on mortality rates, pre-operative preparation of the patient, pre-anæsthetic medication and ancillary drugs, pulmonary ventilation during anæsthesia, post-anæsthetic care, and medico-legal considerations. The author is meticulous in the detail of his descriptions. He has arrived at certain conclusions which seem unnecessarily strict, e.g., ". . . It (thiopentone) can never be used safely without assistance to pulmonary ventilation"; ". . . The growing use (of hypotension) for . . . fenestration seems inexcusable." It is interesting to note that in the United States the anæsthetist may be liable in law for wrongful acts or omissions on the part of the surgeon, which he may observe and permit without objection!

Section 2 deals with techniques of general anæsthesia for tonsils and adenoids, the paranasal sinuses, the ear, larynx, and trachea, and for endoscopy and bronchography. Some of these are difficult to accept. "Tonsillectomy is one of the common forerunners of lung abscess." This surprising view (surely not true in this country) has largely prompted the development of the technique used in the Johns Hopkins Hospital for dissection of tonsils and adenoids. A technique, of which the following quotations are descriptive, seems to leave something to be desired:—

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". . . (Vomiting) . . . is likely to occur at least once" (i.e., during tonsillectomy).

Section 3 is most useful, and is written by one who has obviously first-hand experience of the methods he describes. There appears to be no E.N.T. procedure which cannot be done under local analgesia, and the author favours the method in some of them. "S.M.R. should always be done under local anæsthesia." He also considers it the method of choice in bronchography. These techniques are described in detail.

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ANÆSTHESIA: No. 1, VOLUME 14, of BRIT. MED. BULL. (Pp. 1-74; illustrated. 20s.). London: British Council (Medical Dept.), 1958.

THIS number of the British Medical Bulletin is a symposium on anæsthesia. Since the previous symposium twelve years ago there have many advances in anæsthesia, and it would be an impossible task to survey completely the whole field of progress in the small space of this journal. The editors have therefore wisely concentrated mainly on the basic, physical, biochemical, and pharmacological problems associated with the changed pattern of anæsthesia during this period. There are sixteen articles by experts in each field on problems that have been exercising the minds of anæsthetists in recent years. The treatment of respiratory inadequacy, carbon dioxide hæmostasis, biochemical disturbances and electrographic monitoring in anæsthesia are some of the subjects dealt with, and give an idea of the work covered.

This journal is recommended to all anæsthetists from senior house officer to consultant.

W. M. B.

AIDS TO ANÆSTHESIA. By M. V. Goldman, F.F.A.R.C.S., D.A., L.R.C.P., M.R.C.S. Fourth Edition. (Pp. vii + 360; figs. 92. 12s. 6d.) London: Baillière, Tindall & Cox, 1957.

THE fourth edition of this book, so popular with students, is very little different from the previous one published in January, 1954. There is a more detailed and more accurate diagram of the bronchial tree. Three new drugs are included—buthalitone sodium, trimetaphan, and fluothane, the last-mentioned being dealt with in literally one word. W. M. B.

CLINICAL NEURO-OPHTHALMOLOGY. By Frank B. Walsh, M.D., F.R.C.S., D.Sc. Second Edition. (Pp. xvi + 1295; figs. 441. 231s.) London: Baillière, Tindall & Cox, 1957.

THE first volume of Clinical Neuro-Ophthalmology by Professor Frank B. Walsh was published in 1947. It was the result of ten years' work on clinical diagnosis with colleagues in several departments of the Johns Hopkins Hospital and the Baltimore City Hospital. It was so well received that two reprints became necessary in the intervening years.

The new edition (1957) of over 1,200 pages is an exceptionally fine book, and deals exhaustively with neuro-ophthalmology. A good account of the literature is to be found with each section, and this enables the author to go into great detail in his discussions. It is essentially a reference book, and is made especially interesting by the inclusion of case histories, clinical findings, treatment and, if necessary, post-mortem findings.

In this second edition all the modern aids to diagnosis, such as air encephalography, angiography, etc., are set out in their proper place in relation to the straightforward clinical examination. A good description is given of the newer drugs, and the part they may be expected to play is clearly set out.

This book should certainly be in the library of all departments of ophthalmology and neurology. J. R. W.

INTRODUCTION TO DENTAL ANATOMY. By J. H. Scott, D.Sc., M.D., L.D.S., and N. B. B. Symons, M.Sc., B.D.S. Second Edition. (Pp. xi + 344; figs. 218. 42s.) Edinburgh and London: E. & S. Livingstone, 1958.

I READ this book with a pleasure that was heightened by the knowledge that both the authors are Queensmen—J. H. Scott being the Reader in Anatomy for Dental Students at Queen's, and Symons Senior Lecturer in Dental Anatomy and Histology at St. Andrew's University. This is the second edition of a book that first appeared in 1952 and, unlike many second editions, it has been extensively rewritten, the order changed to improve the subject sequence, and some fifty new photographs and diagrams added. Written for students, it retains its original wide scope, but greater emphasis is placed on the structure and development of the human dental tissues and on the functional anatomy of the oral cavity, whilst much of the descriptive information relating to comparative anatomy has been relegated to an appendix—an arrangement that will find much favour with the student reader. The style of writing is lucid, if somewhat terse and intentionally dogmatic; the illustrations, which are mainly of the authors' own material, are well selected and clear, although a few might be improved by the use of "arrowed legends," e.g., figs. 78 and 161. Other criticisms are few: in one place (page 168) a structure is referred to—the granular layer of Tomes—but not explained until page 170, a small point, but one that is liable to worry the student reader; the definition of secondary dentine (page 162) is not in agreement with the one usually given in textbooks of dental pathology. At the end of most chapters there is a short section on clinical considerations that should be of the utmost value to the pre-clinical student—linking his basic study with his future clinical work.

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CHROMATOGRAPHIC TECHNIQUES, CLINICAL AND BIOCHEMICAL APPLICATIONS. Edited by Ivor Smith, B.Sc., Ph.D., F.R.I.C. (Pp. xiii + 309; figs. 85. 45s.) London: W. Heinemann, 1958.

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AN INTRODUCTION TO PHARMACOLOGY AND THERAPEUTICS. By J. A. Gunn, with the assistance of J. D. P. Graham. (Pp. ix + 327. 18s.) London: Oxford University Press, 1958.

THIRTY years ago Professor Gunn prepared the first edition of this book as a short and comprehensive guide for his students so that in his lectures he would feel free to deal in greater detail with certain subjects either of special interest or illustrating certain important principles. This ninth edition of his book continues to fulfil its original purpose admirably. As one who has to give an introductory course in pharmacology and therapeutics, I sincerely hope that it will find its way into the hands of many of my students. It is written clearly and competently, it is up to date, it is remarkably comprehensive, and it is the sort of book a student will wish to keep on his bookshelf as a reference long after he has finished his introductory course. O. L. W.

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ESSENTIALS OF CHEMICAL PATHOLOGY. By D. N. Baron, M.D. (Pp. xi + 247; 25s.) London: The English Universities Press, 1957.

AMONG the ever-appearing publications which deal with medical and allied subjects it is a relatively uncommon event for a volume devoted entirely to chemical pathology to appear and to those interested in this subject something of an event. The present volume differs in some essential respects from most of those already on the market in that, to quote from the author's preface, "This book has been designed to meet the needs of medical students and of house officers and registrars for an adequate knowledge of chemical pathology and to enable them to follow developments in this rapidly advancing subject." Within these self-imposed limits the author has succeeded and his book should meet with considerable success.

The format of the book is conventional, the initial chapters being devoted to the general biochemistry of the body constituents, water and electrolytes, carbohydrates, proteins and lipids. The succeeding chapters deal with disturbances of biochemical function on a systemic basis and the investigations of these abnormalities. The book is essentially concerned with interpretation and consequently data concerning the actual performance of laboratory procedures is limited to the minimum required for the understanding of the results. There are three appendices; No. 1 deals with normal values, No. 2 with methods of routine ward testing, and No. 3 selected examination questions on chemical pathology. Intentionally the author has limited his references to standard textbooks on the subject, and one cannot help feeling that rather more wide indication of useful additional reading would have been an advantage.

Among the chapters of which special mention may be made are those dealing with the proteins, which includes a useful up-to-date account of differential protein analysis, and with the endocrine glands.

The price of this book must compare very favourably with present-day prices for medical publications, and on the whole it can be warmly recommended not only to that group to whom it is directed, but would be found very useful by junior members of biochemical and laboratory establishments. R. A. N.

CHRONIC SCHIZOPHRENIA. By Thomas Freeman, M.D., D.P.M., John L. Cameron, M.B., Ch.B., D.P.M., and Andrew McGhie, M.A. (Pp. x + 158. 21s.) London: Tavistock Publications, 1958.

IN this monograph the authors begin by laying before their readers a theoretical framework which they then proceed to apply to the behaviour and utterances of a group of schizophrenic patients between 20 and 40 years of age who had been in the chronic refractory wards of the Glasgow Royal Mental Hospital and who had been diagnosed as schizophrenic more than two years previously. They do not make it clear how long the patients had to be in these wards before they qualified for selection, nor do they state whether all patients found there who fulfilled their other requirements were used for the study. It would have been useful to know whether any were left out and the reasons for their omission.

It is not until chapter 10 of this eleven-chapter monograph that, as Miss Anna Freud says in her introduction, "Finally, readers are introduced to the painstaking methods by which the patients are induced—wherever possible—to re-establish contact with the environment." Prior to this there is a detailed presentation and consideration of the psycho-analytic theory of mental functioning and the psychopathology of schizophrenia. Having provided their own theoretical spectacles, they then utilise isolated clinical examples to illustrate the phenomena they observed under such headings as "confusion of identity," "disturbances in perception, of thinking and of memory." They justified their use of such examples by avoiding complex psychopathological inferences that would have called for long accounts of group interviews to support them. The examples are interesting in themselves, but when they are used only in support of a theory, and never to draw attention to gaps in that theory, some readers will perhaps think that "faith" would be a more appropriate descriptive term. The authors, however, express surprise at how coherent occasional remarks of a seriously disturbed schizophrenic can be. In their conclusions the authors make it clear that they have deliberately

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The first chapter deals with accommodation, staff and equipment, including in the latter recommendations on methods of sterilization, by autoclave, hot-air oven, and infra-red heat. The standard method is the hot-air oven, fitted with a fan, and autoclaving is not approved for the sterilization of syringes. The choice of syringes is discussed and figures are given for the relative cost of replacements. Equipment for washing, handling and packaging of syringes and needles are all reviewed in detail.

In the second chapter procedures and techniques are described for the use, care, and sterilization of syringes, including ward procedure, processing of syringes, cleaning and sampling of needles, arrangement of the assembly bench, storage and issue, and the keeping of records. No detail of procedure is omitted from this chapter and many new ideas are incorporated, including the ultrasonic method of cleaning needles, the use of aluminium foil capsules for sealing syringe containers, and pirn-boards for holding syringe barrels.

The third chapter is devoted to capital cost of equipment and running costs, according to the daily turnover of syringes and the scope of the service to be given. For a service providing about 500 syringes a day, the capital outlay would be about £2,000, and the running costs, including syringe and needle replacements, £3,725 a year, or about 5d. a syringe.

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THE EARLY DIAGNOSIS OF THE ACUTE ABDOMEN. By Sir Zachary Cope, B.A., M.D., M.S.(Lond.), F.R.C.S.(Eng.). Eleventh Edition. (Pp. x + 188; figs. 35. 18s.) London: Oxford University Press, 1957.

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POLYMYOSITIS. By John N. Walton, M.D., M.R.C.P., and Raymond D. Adams, M.D. (Pp. x + 270; figs. 47. 32s. 6d.) Edinburgh and London: E. & S. Livingstone, 1958.

It has only been in comparatively recent years that the position of muscular dystrophy as a clinical entity has been seriously challenged by the discovery that a somewhat similar clinical picture may be seen in polymyositis, dermatomyositis, and scleroderma. The authors, who have based their monograph on a comprehensive review of the literature (some twelve pages of references are given) and upon a series of forty cases of their own, note that before the year 1948 in no instance was a pathological diagnosis of polymyositis made in a patient without clinical evidence of skin involvement.

Classification presents a difficulty for a syndrome in which muscular wasting and weakness may be uppermost in one case, skin changes prominent in another, and in a third both features and more pronounced evidence of collagen disease seen. They propose that these cases should be grouped under four headings: 1, Polymyositis, sub-acute and chronic, occurring in childhood, early or late adult-life (in this group the disease is confined to muscles); 2, polymyositis with associated collagen disease or dermatomyositis, in which skin changes are minimal; 3, severe collagen disease with only light muscular weakness; 4, polymyositis in association with malignant disease, e.g., primary bronchial carcinoma.

In six of their cases pseudohypertrophy of muscles was present. In distinguishing polymyositis from muscular dystrophy they emphasise the clinical frequency of dysphagia and involvement of the neck muscles in the former condition.

The book is well illustrated with colour photographs of skin lesions and the pathological section is very fully dealt with by Dr. Raymond Adams. There are chapters on investigation by laboratory and electromyographic procedures and a short chapter on treatment. Evidence, however, that successful results follow cortisone or A.C.T.H. treatment is not very convincing, but the inclusion of full clinical details of the forty cases in the appendix is a valuable point, and the book serves a useful purpose in reviewing the present state of knowledge in relation to these diseases. R. S. A.

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THIS is the second of the three volumes of the twelfth edition of this popular manual to be published. The revised order of dissection now used calls for little change in the present volume. The use of the Paris Nomenclature (1955) is continued, but the more familiar terms in common British usage are retained where thought desirable. For example, the "innominate" vessels are referred to as such in the text, with the term "brachiocephalic" as an alternative. This effort to maintain continuity of usage is very welcome. Despite the sad loss of the editor, whose death earlier this year marks the end of an association with this manual extending back to the ninth edition in 1935, it is to be hoped that the publishers will have Volume II on the thorax and abdomen on sale before the start of the coming Michaelmas Term. This would allow the complete edition to be used throughout by students starting anatomical studies at that time, and would simplify their problems by giving them a single terminology to master. The present volume can be recommended to all wishing an up-to-date manual on the anatomy of the head and neck. W. R. M. M.

AIDS TO MEDICAL DIAGNOSIS. By G. E. Frederick Sutton, M.C., M.D., F.R.C.P. Eighth Edition. (Pp. viii + 400. 12s. 6d.) London: Baillière, Tindall & Cox, 1958.

THIS small book is a mine of information and maintains the high standard of the Aids series. It has been completely brought up to date and certain sections, such as "Diseases of the Heart," are outstanding in their logical and compressed knowledge. In a small book of this type it is obvious that there must be sections where one would like to have more complete material but, on the whole, this book maintains an extremely high standard. It can be thoroughly recommended to the student, the general practioner, and the physician as a delightful revision of knowledge and presentation of clear thought. A. P. G.

THE TISSUES OF THE BODY. An introduction to the study of anatomy. By W. E. LeGros Clark, F.R.S. Fourth Edition. (Pp. xii + 415; figs. 127. 40s.) London: Clarendon Press, Oxford University Press, 1958.

THIS book is not a shortened version of a comprehensive text on human anatomy. It is rather an attempt to make the basic facts on tissue anatomy, so commonly neglected by the medical student, as attractive as their importance demands, and also to stimulate the reader's intellectual curiosity so that he will ask for more. This is achieved by constantly drawing attention in the text to recent researches, and by means of frequent footnotes to guide the reader to original papers on the subjects under discussion. A certain amount of rewriting has been necessary in order to bring the work up to date, but the general size of the book has not been altered. The author has fully achieved his aims, and this edition can be recommended to all medical and dental students on account of the ease with which it can be read and assimilated, and because of its wide-ranging viewpoint. W. R. M. M.