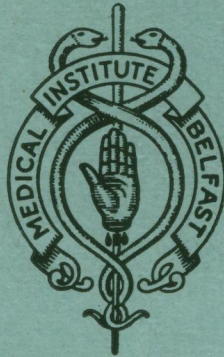


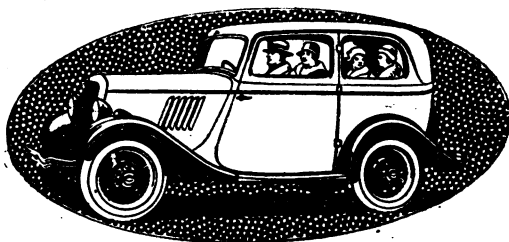
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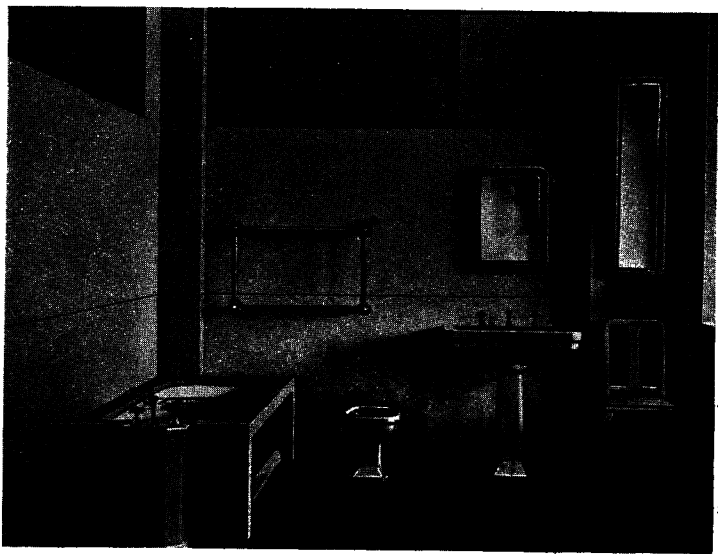
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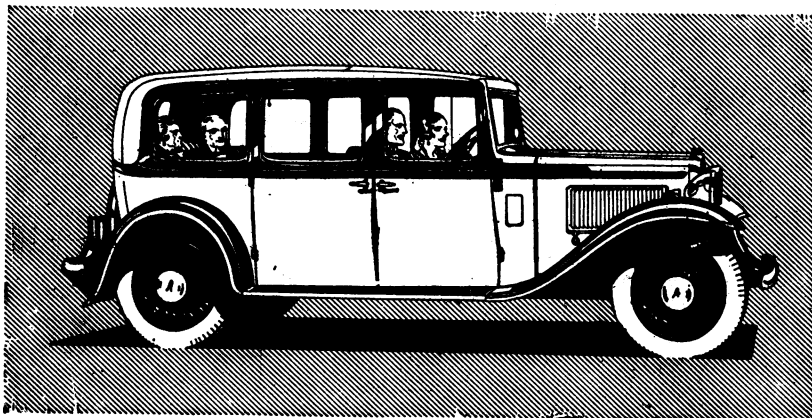
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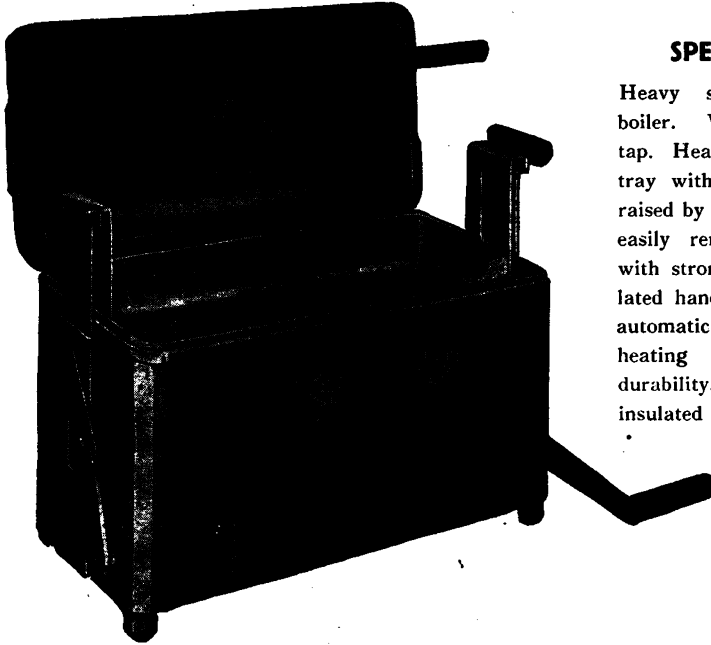
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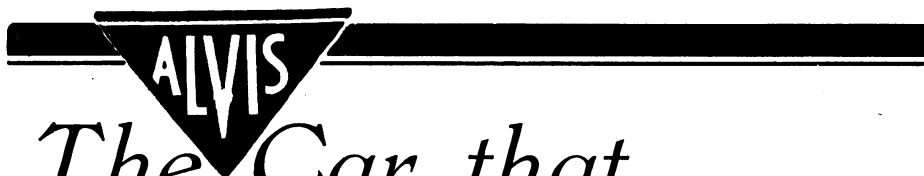
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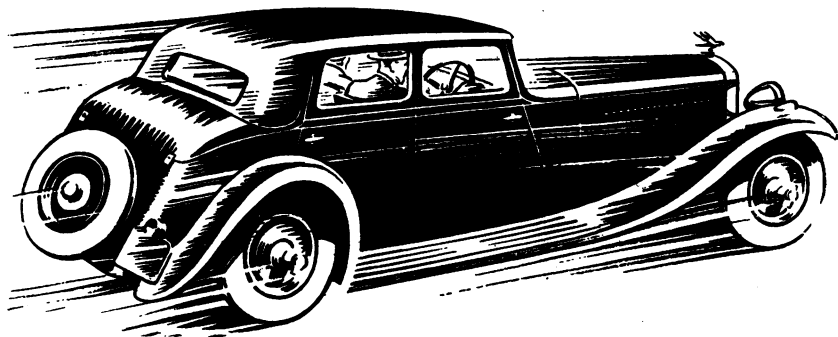
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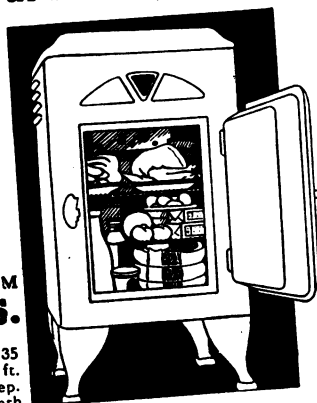
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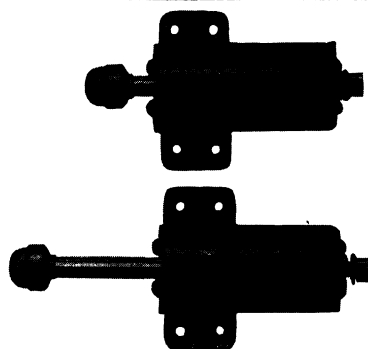
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THE ULSTER MEDICAL JOURNAL

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1st January, 1st April, 1st July, 1st October.

THE ULSTER MEDICAL SOCIETY

THE MEDICAL INSTITUTE,

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Dear Sir (or Madam),

If you are not a member of the Ulster Medical Society, we would appeal to you to give the question of joining your consideration. The Society has been in existence since 1862, and has always been active in keeping its members interested in the advances in medical science as well as in current professional affairs. The Medical Institute, situated in College Square North, belongs to the Society (through the generosity of Sir William Whitla), and is ideally adapted for meetings, committee meetings, and recreation. There is a library with current medical periodicals, and facilities for reference to medical literature are available in conjunction with the library at the Queen's University. There is also a billiards-room available to members, and lighter periodicals are also provided. An annual dinner is held each year in December, and a golf competition in June. Meetings are held at intervals of a fortnight during the winter months, and papers are contributed by members. Distinguished visitors are occasionally asked to contribute papers on subjects upon which they are specially qualified to speak. *THE ULSTER MEDICAL JOURNAL*, the official organ of the Society, is issued to all Fellows and Members free of charge.

The subscription to the Society is one guinea for Fellows and Members living in the country; two guineas for Fellows living in Belfast; and one guinea for Members living in Belfast who are not qualified more than seven years. The payment of a sum of twenty guineas entitles one to election to Life Membership.

May we, therefore, appeal to you to join the Ulster Medical Society, and so enable us to widen its influence and sphere of usefulness still further? For your convenience a proposal form is attached, which, if filled in and sent to the Honorary Secretary, will ensure your name being put forward for election to membership of the Society.

If you do not wish to become a member of the Society, will you consider entering your name as a subscriber to *THE ULSTER MEDICAL JOURNAL*? The subscription is five shillings per annum, payable in advance to the Honorary Treasurer, for which a banker's order form is attached for your convenience.

We remain,

Yours faithfully,

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PUBLISHED QUARTERLY ON BEHALF OF THE ULSTER MEDICAL SOCIETY

Vol. III

1st APRIL, 1934

No. 2

EDITORIAL

THE ninety-first annual report of the Royal Medical Benevolent Fund Society of Ireland has now been issued. It is again a tale of help to our medical brethren who have fallen on difficult times, and of assistance to their widows and orphans, so often left without even the necessities of life. The great financial depression has unfortunately been reflected in the receipts of the Society, and the grants to those in need are, unhappily, of smaller amounts than would have been possible if the profession had supported as it should this, the only Medical Benevolent Fund in Ireland.

At present, while the response from some of the small and poor counties is more than generous, some of the larger and richer ones subscribe nothing, although grants are made from the Fund to these non-subscribing areas. The Six-County area, however, contributes to its funds, and Dr. Leonard Kidd, the secretary for County Fermanagh, has established a record by securing subscriptions from every medical practitioner in his county. Can another Northern record be created this year by one of the Six Counties raising the largest amount of any county in Ireland? The Fund is in need of, and deserves, our support. Last year it suffered a grave loss to its income by a decrease of £267. 12s. 4d. in the yield from its investments. Subscriptions also from the branch secretaries show a decline of £71. 0s. 3d. This latter loss, however, is more apparent than real, as after the financial year had closed a cheque value £60 was received, and the decrease is thus a mere £11. The decrease in revenue is a serious matter, as the total sum distributed in grants amounted to only £2,267, divided amongst ninety-four recipients: 69 widows, 17 orphans, and 8 doctors.

We appeal to the medical profession of Northern Ireland to support this deserving charity. Subscriptions will be gratefully acknowledged by any of the following county treasurers for this area:—Co. Antrim: Dr. V. G. L. Fielden, Belfast; Co. Armagh: Dr. W. J. Dawson, Newtownhamilton, and Dr. Dougan, Portadown; Co. Down: Dr. Nolan, Downpatrick; Co. Tyrone: Dr. R. H. C. Lyons, Dungannon; Co. Fermanagh: Dr. Leonard Kidd, Enniskillen; Co. Londonderry: Dr. J. W. Killen, Londonderry.

SIR WILLIAM WHITLA, M.A., M.D., D.SC., LL.D.,

Pro-Chancellor and Emeritus Professor of Materia Medica, Queen's
University, Belfast; Consulting Physician, Royal Victoria Hospital;
Honorary Physician in Ireland to His Majesty the King.

BRITISH medicine has lost a remarkable and fascinating personality.

Sir William was born in Monaghan in 1851. Shortly after leaving school he came to Belfast as an apprentice to the leading firm of dispensing chemists, Wheeler & Whitaker. He soon decided on reading for the medical profession, and when his apprenticeship was completed he entered Queen's University. His college career, a brilliant one, terminated in 1877, when he obtained his M.D. with first-class honours and a gold medal.

Soon afterwards he was appointed resident medical officer and superintendent of the Royal Hospital. His exceptional ability both as a physician and an administrator soon became evident. He quickly revolutionized the working of the whole institution.

When his term of office came to an end he decided to commence practice in Belfast. He was already so favourably known that his success was instantaneous. Professor Gordon was so deeply impressed by his ability that he invited him to assist him at all his private operations. At that time, indeed, his bent was chiefly surgical. He rapidly developed an extensive general practice, and soon perceived that his future lay in medicine.

It is doubtful if any medical man ever gained and maintained the confidence, trust, and affection of his patients in a higher degree than William Whitla.

His appointment as physician to the Royal Hospital gave him an opportunity to demonstrate his ability as a clinical teacher, and he soon attracted a large clinical class.

In the midst of his hospital work and an extensive and exacting practice, he turned his attention to literature. He wrote his "Materia Medica and Therapeutics," which was at once a pronounced success. In this work the value of his early training as a chemist was very evident. It was the first textbook to reproduce woodcuts of pestle and mortar, measure-glasses, and other apparatus used by dispensers. The book met the requirements of the dispensing chemist and the qualified medical man. The section on non-official remedies proved a special attraction. The book had an enormous sale. The twelfth edition, revised and edited by Dr. Gunn of Oxford, happily reached him about two months before his death. He displayed it to his friends with natural pride.

The resignation of the Chair of Materia Medica by Professor Seaton Reid paved the way for his appointment as a professor in Queen's College. He soon breathed new life into the teaching of the dry and difficult subject. His fame and popularity rose still higher.

An indefatigable worker, he next produced that remarkable book, "The Dictionary of Treatment." His versatility and the extent of his reading may be gauged from the fact that every article in the first edition was written by himself.

The success of this work was immense. It was published simultaneously in England and America. The English edition of eight thousand copies was sold out within nine months; two thousand of the American edition were imported into this country to meet the immediate demand. The book met a definite want, and was soon to be found on the bookshelf of almost every general practitioner. A specially-bound volume of the Chinese edition was amongst his most treasured possessions.

Apart from his medical knowledge, Sir William Whitla was a widely-read and cultured man. His knowledge of English literature was amazing. He was one of the greatest living Shakespearean scholars. The annual visits of Sir Frank Benson and his company to Belfast were almost red-letter days to him. Those who were privileged to participate in the delightful suppers which he gave to the leading members of that company can never forget the charming atmosphere of that hospitable table.

Appreciation of his merits by those best qualified to judge is indicated by the number of honorary degrees conferred upon him, which included M.A., R.U.I.; LL.D., Glasgow; M.D., Dublin; D.Sc., Q.U.B., and by King Edward, who included his name for a knighthood in his list of Birthday Honours.

Sir William was an intimate friend of the late Sir Donald Currie, and was largely responsible for his magnificent gift of £20,000 to Queen's College, Belfast.

A really great man, with a strikingly picturesque mind, he was a wonderful raconteur; his stories were innumerable, and were recorded with a wealth of detail that made him a unique and fascinating companion.

On the difficult problems connected with education he was a recognized authority. He was an early advocate of the establishment of a university at Belfast.

It was a fitting tribute that he should have been elected as the first representative of the Queen's University of Belfast in the Imperial Parliament, and one of its Pro-Chancellors.

Sir William was a widely-travelled man; his descriptions of Russia and of the famous world fair at Nizhny-Novgorod, of Palestine, of Italy, Canada, and America, were an educational treat.

Members of the British Medical Association will long remember his presidency on the occasion of the annual meeting in Belfast in 1909. His bounteous hospitality, brilliant garden-party, and a presentation to each member who attended the meeting of his recently-published book, "A Dictionary of Medicine," are not easily forgotten.

A man of wonderful vision, he never did anything in a small way; he never looked for praise or reward.

The Medical Institute, the home of the Ulster Medical Society, contains a bronze plate with the following inscription: "This building was erected, equipped, and presented to the Ulster Medical Society by Sir William Whitla, M.D." The foundation stone was laid by Peter Redfern, M.D., on the 12th April, 1902. The building was declared open by His Excellency the Earl of Dudley, Lord Lieutenant of Ireland, on the 26th November following.

The cost of the building had exceeded £6,000. When one tried to thank him, he simply replied, "I owe it to the profession; they gave me the money for my book."

A man of deep religious conviction, his Bible was his constant companion. The tale of his private benefactions will never be known, but no one in real distress ever applied to him in vain.

For almost four years he has been unable to leave his room; his mental powers and his memory remained almost perfect to the last. His devoted wife, Lady Ada Whitla, predeceased him by eighteen months.

A great man, a very great man, has gone to his long rest; a rest which he earnestly desired and for which he patiently waited. William Whitla has not lived in vain.

A. B. MITCHELL.

Some Recent Advances in the Diagnosis and Treatment of Pulmonary Tuberculosis

By R. B. CLARKE, M.D.,

from the Forster Green Hospital for Chest Diseases, Fortbreeda, Belfast

THE title of my paper might be taken as a challenge, so to disarm criticism I will state at once that the advances in diagnosis are entirely of a technical nature.

Nothing has taken the place of a skilfully elicited case-history and a full and painstaking examination of the symptoms of the patient. The temperature, pulse-rate, and weight-curve remain valuable guides in assessing the activity of the disease, although more delicate tests, such as the blood-sedimentation rate or the monocyte count, are undoubtedly improved methods of measuring slight degrees of toxæmia.

The blood-sedimentation rate, which is now widely used for the diagnosis and control of treatment of tuberculosis, is a modern adaptation of a fact known for many hundreds of years. The old physicians, when they bled a patient, often noted that the serum separated very rapidly from the clot in cases of fever. This was spoken of as the "buffy coat," and the phenomenon is due to an alteration of the proportions of serum fibrogen, serum globulin, and serum albumin in the blood-serum. In 1918 Fahræus rediscovered this long-forgotten phenomenon, and a simple technique has been elaborated for measuring the fall of the red cells in the citrated blood-serum of the patient. Of course the test is not in any way specific for tuberculosis, as it is positive in many inflammatory and wasting conditions. It is, however, of great value in assessing the degree of toxæmia when the temperature is normal. Although not specific, the blood-sedimentation test has also a definite value in diagnosis, as there may be a marked deviation from the normal in early phthisis. The sedimentation rate is usually normal in bronchitis, asthma, and pulmonary fibrosis, also in arrested or healed pulmonary tuberculosis.

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Personally, I have found this test of great value in studying the course of the disease and the response of the patient to treatment.

EXAMINATION OF SPUTUM.

Next to history and symptoms, the sputum test is the most valuable single aid to diagnosis. I will say something of three recent advances in the examination of sputum for bacillus tuberculosis.

1. **CONCENTRATION METHODS.**—The old antiformin concentration method has been largely replaced by concentration in hypertonic saline or petrol. The technique is simple, given a mechanical shaker, and many additional positive results are thus obtained when the bacilli are scanty.

2. **THE LARYNGEAL REFLEX.**—Many patients fail to produce sputum for examination in the early stages of phthisis. Usually they swallow the sputum unconsciously, and the bacilli may be recovered from the stomach-washings or the fæces. A simpler method which is often successful is to insert a laryngeal mirror into the patient's mouth without warning, thus exciting a spasmodic cough. The method does not conduce to the popularity of the physician, but tubercle bacilli are often recovered without difficulty from small plaques of sputum adherent to the mirror.

3. **CULTURE OF TUBERCLE BACILLI FROM SPUTUM.**—It looks as if this is to be the method of the future, and it is probable that in a few years all negative specimens will be planted out as a routine. Up to the present the practical application of this method has not been perfected.

When a positive sputum result cannot be obtained, and the test has been repeated several times, the possibility that any case, however suspicious, is not one of tuberculosis must be seriously considered. It is in such cases that radiology is specially helpful, and it may be necessary to supplement this by the injection of lipiodol. A famous New York physician wrote recently that many cases of bronchiectasis go to their graves diagnosed as tubercular, after suffering the rigours of unmerited and useless sanatorium treatment.

The X-ray photograph has its uses even when the sputum test is positive. It may be found invaluable for convincing the relatives that the patient really has pulmonary tuberculosis, although no case has ever been heard of in the family before. The X-ray film also supplies a ready method of assessing the extent and character of the disease, and often discloses an early cavity which gives rise to no stethoscopic signs.

I have not referred to physical examination, and indeed I believe that if some point must be neglected in the stress of modern life, it is better to neglect auscultation of the chest than consideration of the symptoms or examination of the sputum. It is authentic that one of the best Victorian chest specialists in London was extremely deaf, and I would remind you that he practised before the days of radiology.

The aspect of the patient, the tone of the chest muscles, and the movements of the chest wall, are points of the utmost importance. Accentuation of the pulmonary second sound is also a fairly constant sign in early phthisis. As to percussion and

auscultation, now that such authorities as Dr. Fishberg and Professor Ramsbottom have admitted that they are often unable to detect physical signs in the early case, I will freely make the same admission. The most constant and the most important early stethoscopic sign of a tuberculous infiltration is the presence of an area of weak or rough breathing. The detection of this requires considerable powers of concentration, long experience, and absolute silence. Unfortunately this very sign (weak breath-sounds) is commonly associated with healed tuberculosis or thickened pleura.

To sum up my personal views on the diagnosis of tuberculosis of the lungs, the history, symptoms, and sputum examination are the surest foundations on which to build a diagnosis. In all doubtful cases a good X-ray film is invaluable positive or negative evidence. Physical signs may make the diagnosis obvious, but these signs are often absent at the early stage, unless the lesion is situated close to the apex.

Dr. Batty Shaw, senior physician to the Brompton Hospital, wrote recently :

“The only medical practitioner who is relatively happy with regard to the diagnosis of tuberculosis of the lungs—a process requiring repeated observation, and not an episode requiring only one—is he or she who can command repeated examination of the sputum for tubercle bacilli, and repeated skiagraphic examinations of the chest. No wonder that in the past, and, alas ! also in the present, some superintendents of sanatoria, armed with a bacteriological laboratory and skiagraphic outfit, have had, and have rightly had, such a low opinion of the mere clinical powers of general practitioners, of tuberculosis officers, and of consulting physicians not so armed.”

It follows that the diagnostic efficiency of a tuberculosis scheme, in city or county, may be measured by the readiness with which general practitioners are provided with facilities for sputum tests and X-ray photographs of the lungs. Given a positive sputum result, or a definitely positive X-ray result, the general practitioner can make his own diagnosis. Without these aids, only a superman can detect with certainty an early tuberculous infiltration in the lung.

Although physical examination of the chest has severe limitations in the truly early case, it is invaluable as a method of observing the course of the disease, if the pathological processes in the lung advance.

In children between the ages of three and twelve years, phthisis is an extremely rare disease. If a sick child has a cough and abnormal physical signs in the chest, it is very probable that the cause is not tuberculosis. Non-tubercular pulmonary fibrosis is as common in children as tuberculosis is rare, and the after-history of such cases, followed for many years, shows that they do not often develop frank tuberculosis of the lungs.

The modern treatment of tuberculosis began with George Bodington, who started the first open-air sanatorium in Europe at Sutton Coalfield in 1851. Apart from fresh air, the principal points in Bodington's treatment were the administration of large draughts of good wine and regular exercise on horseback. Some good

results were claimed, but he suffered the common fate of pioneers, and in a few years his sanatorium was converted into a private asylum.

Some years later Dr. Bennett, a Scottish physician practising on the Riviera, drew attention to the value of physical rest. He discouraged his patients from horseback-riding and similar exercises, and urged them to take the air in an open carriage. Subsequently the Germans and other Continental physicians elaborated the rest treatment, which has received strong support during the present century from the work of Dr. Jacquerod of Leysin and others. I am convinced that the greatest advance in the treatment of tuberculosis in modern times is the conclusive proof by Jacquerod that a large proportion of tubercular lesions in the lungs will heal by resolution, provided the patient is kept at absolute rest for a sufficient length of time. Before Jacquerod's work, it was generally taught that tuberculous lesions progressed to softening or healed by a slow process of fibrosis and calcification. It is now a commonplace that large tuberculous infiltrations may disappear in the course of a few months with rest treatment, leaving little or no evidence of scarring. Of course, Jacquerod's patients, who belonged to the well-to-do classes, were treated by the combination of fresh air, wholesome food and rest, usually summarized in the phrase "sanatorium treatment." He insisted on a long period of rest in bed, to be counted in months and not in weeks. A normal temperature is no excuse for allowing a patient to take exercise, if an active lesion is present in the lung.

I have the utmost faith in rest treatment, but the practical difficulties are enormous, even in a hospital. Naturally the expense of nursing and attendance is increased, as compared with the old method of letting every patient out of bed when the temperature has been normal for a few days. To apply this treatment successfully, the doctors and nurses concerned must have complete faith in it, and they must be unrelenting in the encouragement and admonition of their patients. The psychological effect of a too-prolonged period of bed-rest is bad for some patients, who become insubordinate, depressed, or morbidly introspective, according to their various temperaments. The average patient endures rest treatment more cheerfully if it is combined with some specific and systematic medical treatment, which convinces him (sometimes without much reason) that the doctor can influence radically the course of the disease.

Outside sanatoria and hospitals the difficulties of carrying out rest treatment are probably greater. It has been truly said that the poor man cannot afford to have early tuberculosis, and in these days of economic competition the same difficulty weighs on most of the non-manual workers also. The practical limitation of rest treatment is that its operation may be extremely slow. Twelve, eighteen, or even twenty-four months rest may be necessary to bring about the resolution of an early tuberculous lesion. The various methods of collapse therapy, artificial pneumothorax, phrenic evulsion, adhesion cutting, apicolysis, and thoracoplasty, are most valuable aids in suitable cases to shortening the period of treatment. Before saying anything more about collapse therapy, I should like to mention sanocrysin. This drug, which is a double salt of gold and sodium, has been used for the last ten

years. We have treated several hundred cases with it at the Forster Green Hospital, and many of them have remained well for a period of years. The drug is usually given intravenously in gradually increasing doses at intervals of a week or longer. Six injections constitute a course, and a second course should follow some weeks later; a third course may be given with advantage some months later. Nowadays complications are infrequent with the graduated system of dosage, but albuminuria or diarrhoea may occur. The treatment is particularly suitable for the early subacute case, when the ultimate outlook appears to be bad, but the patient's present condition is good or fair. Its success is probably due to a stimulation of the natural resistance of the body, and the patient must be kept at rest during the injections. It may be asked whether sanocrysin is a suitable drug for use in general practice. Personally, I have known a number of cases treated successfully with sanocrysin by private doctors, and I do not regard it as a more dangerous drug than salvarsan or digitalis.

When treatment with sanocrysin, or one of the many other gold preparations such as solganol or crisalbine, is contra-indicated by albuminuria, or is not desired by the patient, a course of tuberculin (B.E.) may help to bring about an improvement. It may, indeed, appear in the end that tuberculin cautiously used with the patient at rest, is the equal of sanocrysin. With sanocrysin and tuberculin alike, reactions are to be avoided, and it should be remembered that a small dose which does the patient good is better than a large dose which provokes a harmful reaction.

VITAMINS AND PULMONARY TUBERCULOSIS.

A diet rich in vitamins is an important factor in raising man's resistance to infection, and should therefore promote the healing of disease. Recently an interesting observation was made at a Swedish military academy, where the incidence of tuberculosis among the cadets had been very high for some years. It was found that the diet of the cadets consisted largely of white bread, hard biscuit, preserved meat, salt pork, margarine, and tinned vegetables. An entirely new diet scale was introduced, including large quantities of fresh eggs, fruit, and vegetables, supplemented by cod-liver oil. Within two years of the introduction of this diet, the incidence of fresh cases of tuberculosis had fallen to one-third of the former rate. No doubt other factors may have had a part in reducing the number of cases, but the figures published are so striking that it appears the change of diet produced a definite effect.

ARTIFICIAL PNEUMOTHORAX.

Artificial pneumothorax and other collapse methods are valuable aids to the healing of tubercular lesions, especially the closing of cavities. Rest treatment alone may operate so slowly that it is a practical impossibility to carry it to a successful conclusion, and when a cavity of any considerable size has formed it is unlikely to heal with rest alone.

Artificial pneumothorax is the method of choice for collapsing the lung in nearly every case. The technique is simple, the operation almost painless, and in skilled hands the risk of serious complications is slight. The treatment first came into

prominence as a remedy for advanced and apparently hopeless cases of phthisis, and it is true that in such cases the immediate results may appear little short of miraculous. When a diseased lung is successfully collapsed by the introduction of air into the pleural space, fever, cough, and sputum may disappear in the course of two or three weeks. It has been found, however, that the usefulness of the treatment in advanced cases is often nullified by pleural adhesions, and the present tendency is to resort to this treatment at an earlier stage of the disease. I do not think it justifiable to advise artificial pneumothorax for a patient with an early lesion which is likely to heal by rest alone. The treatment is indicated in nearly every case with early cavity formation, and in a large number of cases where the effects of rest are slow. The persistence of tubercle bacilli in the sputum or a raised sedimentation rate after the rest treatment has had a trial, are other indications. The decision to start an artificial pneumothorax must be based on a comprehensive survey of the patient's disease and his reaction to it, including temperament, social position, financial resources, and, to some extent, personal inclinations.

A few years ago artificial pneumothorax was discussed on the assumption that this treatment applied only to unilateral cases. Unfortunately, many cases have definite involvement of both lungs at an early stage, but the modern view is that this does not usually contra-indicate artificial pneumothorax. Many cases are admitted to the Forster Green Hospital who are considered too ill to have an artificial pneumothorax, but this is only rarely because of the extent of the disease in the better lung. Cachexia, chronic myocardial weakness, and marked fibroid changes render artificial pneumothorax useless or dangerous, but disease in the better lung is not in itself a contra-indication.

In bilateral cases an attempt should be made first to collapse the worst lung, and if this is successful, a partial collapse of the other lung may be affected also. When collapse is prevented by adhesions, a similar effect may be produced by the operation of phrenic evulsion. Given time and patience, a great deal can be done for even the bilateral case, but it must be admitted that the cost of treatment to the individual or to the State is heavy.

The active treatment of pulmonary tuberculosis, whether by rest, sanocrysin, collapse methods, or by a combination of two or more of these methods, is useful chiefly in the early stages of the disease. At this time the lesions in the lungs are spreading, fibrosis has probably not begun, and the patient's natural powers of immunity are low. Energetic and thorough treatment will yield good results in a high proportion of cases and lack of treatment will usually end disastrously.

At a later stage, the position of the patient is quite different. He has either had a long course of treatment, or perhaps he has survived in spite of having struggled on at his work. Fibroid changes in the lungs will prevent collapse of cavities by artificial pneumothorax or phrenic evulsion, though it is true that thoracoplasty may effect a permanent cure. Before sending such a patient to hospital or a sanatorium, it is well to consider the Hippocratic maxim: "In acute cases consider the disease; in chronic cases consider the patient."

Three Examples of Chordoma

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CHORDOMATA, or tumours derived from remnants of the notochord, are not common, the first recognized example in this country having been described by Stewart as recently as 1922. The tumours are usually slowly growing and possess a comparatively low grade of malignancy, but their surgical extirpation is extremely difficult, if not impossible. Further, one of their favourite sites of origin is in the base of the skull—at or near the spheno-occipital synchondrosis—so that a timely recognition of the nature of the growth might spare the patient one or more major operations and the surgeon a hopeless task. Chordomata of the sacral region, and of other parts of the vertebral column, are more accessible to surgery; therefore the prognosis is not quite so unsatisfactory as in the cranial growths, but even here it is not good.

Of the three examples recorded in this paper one (Case I) was discovered at post-mortem examination growing from the region of the spheno-occipital synchondrosis. The site of origin of the other two is doubtful, because both are biopsy specimens, one (Case II) from the neck, and the other (Case III) from the retropharynx.

Small localized proliferations of notochordal tissue were recorded by Luschka and Virchow (1857-8), growing in the form of soft, jelly-like masses from the region of the clivus and penetrating the dura mater. Virchow regarded them as *ecchondroses*, but they were peculiar in that they contained large vacuolated cells, perhaps arising from degeneration. He introduced the term "*ecchondrosis physaliphora*" to describe them. Soon afterwards Müller suggested, on the basis of comparative anatomical studies, that these formations were derived from notochordal remnants, and his opinion was ultimately confirmed by Ribbert (1894). Their notochordal origin is now generally accepted, and the term "*eccordosis physaliphora spheno-occipitalis*" was introduced by Stewart, who observed four of them in a special series of 350 consecutive autopsies. Larger masses of a fundamentally similar nature, but possessing the property of slowly progressive growth and showing a tendency to recur after removal, may arise in the clivus and in other situations in the spinal column, and it is to this type of tumour that the term "*chordoma*" is applied. Stewart (1922) presents an historical summary together with a list of cases published prior to that time. Stewart and Morin (1926) publish a review of the literature and record a new case. Cappell (1928) indicates an interesting parallel between the developing notochord in the dogfish and the evolution of the fully formed tumour.

It will be recollected that in the human subject the notochord disappears comparatively early in the life of the embryo, and persists only as the nucleus pulposus in the intervertebral discs. It has been shown, however, that in addition to the *ecchordosis physaliphora spheno-occipitalis* described above, small remnants may be found on occasion almost anywhere in the spinal column. It is conceivable that

from such elements tumour formations arise. Theoretically, then, one might anticipate the appearance of chordoma in any situation in or near the confines of the original notochord, but in actual practice the tumour is found with much greater frequency in positions corresponding to the upper and lower extremities of this structure. Stewart and Morin (1926) point out that in a series of fifty-five tumours twenty-five occur in the region of the spheno-occipital synchondrosis and twenty-seven in the sacro-coccygeal region. Examples of the tumour in other situations are not wanting, however, as evidenced by those of Cappell (1928) in the cervical and dorsal region, Hutton and Young (1929) in the dorsal region, and Davison and Weil (1928) in the lumbar region.

NAKED-EYE APPEARANCES OF CHORDOMA.—The macroscopic appearances are highly characteristic, the tumour presenting itself as an encapsuled and lobulated growth. The inter-lobular septa are composed of fibrous tissue, and the spaces are occupied by whitish semi-transparent material of a gelatinous nature. The degree of malignancy is apparently inversely proportional to the amount of this gelatinous substances present for the active varieties present a solid opaque appearance. The whole formation bears some resemblance to the “mixed parotid tumour,” or in some cases to a “mucoid” carcinoma. The tumours vary greatly in size and may on occasion attain very large dimensions. A spheno-occipital growth measuring eleven centimetres in length is described by Jelliffe and Larkin (1912), whilst that of Willis (1930) occurring in the sacro-coccygeal region was fifteen inches in length.

Chordoma usually is slow-growing and of low malignancy, but it tends to recur after removal. A particularly notable feature is its tendency to destroy adjacent bone. In the intracranial cases, penetration of the skull with extension into the nasopharynx, orbits, and sinuses is not uncommon. The tumour may also grow upwards into the interpedicular region, as in the case described by Bailey and Bagdasar (1929). In the sacro-coccygeal region, invasion of sacrum, coccyx, and os innominatum, with ulceration into the rectum, has been described. A notable feature of this group is the manner in which the skin remains intact even when stretched over a very large tumour mass. In the cervical region, extension into the nasopharynx, fauces, and laryngopharynx occurs.

Metastases would appear to be relatively uncommon, although these have been described in the cervical glands by Lewis (1921) and over the right scapula by Stewart (1922). In Willis’s sacro-coccygeal case (1930), very widespread metastases occurred in lung, spleen, liver, kidney, etc., this being attributed to direct spread by the iliac veins.

HISTOLOGICAL APPEARANCE.—Microscopically the tumour comprises a dense fibrous stroma, part of which goes to form the capsule and part of which is involved in the production of the interlobular trabeculæ. Resting on this fibrous tissue there are columns and solid alveolar masses of epithelial cells. The younger or more recent areas are composed of solid aggregates of epithelial cells with uniformly staining cytoplasm, but later these cells are liable to become vacuolated. Later still, this vaculation is very marked, producing the so-called physaliphorous cells. Occasionally individual cellular outlines are lost, resulting in the production

of masses of vacuolated syncytium. The vacuoles would appear to be due to the production within the cells of mucin, and this passes out into the inter-cellular spaces, where it tends to lie in columns. Ultimately, what may be described as a pool of mucin is formed in which only a few isolated islands of cells remain. This pool is surrounded by tumour cells, and it may contain extravasated blood.

The cells themselves vary greatly in shape and size, mitotic figures are not uncommon, and there is reason to believe that amitotic division, which is said to occur in the primitive notochord, also takes place in the chordoma. Nuclear hyperchromatism is a prominent feature. Nuclear vacuolation was emphasized by Stewart (1922) and Cappell (1928), but the nature of the contents of these vacuoles is not known. Another interesting feature is the presence of glycogen within the cells, especially the actively growing ones. This is apparently independent of the mucinous secretion, and no support can be lent to the suggestion that the nuclear vacuoles contain this substance.

CLINICAL ASPECTS.—Age does not appear to play a very important part in the incidence of the tumour, but the majority of cases occur between the ages of 30 and 50 years. The growth has been recorded, however, in patients of 16 and 68 years. Males are affected rather more often than females.

The symptoms of the sphenoccipital group are those of any other slowly-growing tumour at the base of the brain. Headache, vomiting, and papilloedema are frequent symptoms, and appear at a comparatively early stage. As the growth increases in size, involvement of various cranial nerves is manifest. Symptoms of bulbar paralysis may ensue, or, again, the signs may point to a tumour of the cerebello-pontine angle. Epileptiform seizures have been described (Stanton, 1932).

The prognosis in this group is very unfavourable, the average duration of life after the first appearance of symptoms being two years. Operative interference can only meet with partial success, owing to the fact that the tumour invades and destroys the base of the skull. In one case, described by Bailey and Bagdasar (1929), a tumour of the suprasellar region was removed by curettage and the patient was in good health fifteen months later, but this would seem to be rather exceptional.

In the sacrococcygeal region, the tumour presents itself as a slowly-growing, firm, elastic mass situated in the middle line. Symptoms depend upon whether the growth extends inwards towards the rectum, or tends to spread posteriorly to appear beneath the skin of the back. Pain in the coccyx is usual in either event. Tumours situated anteriorly may be palpated through the rectum, and pain and difficulty in defæcation, sometimes accompanied by blood-stained mucus in the stools, will be noted. Where the growth is posterior to the sacrum its presence beneath the skin should lead to a provisional diagnosis of chordoma. Treatment consists in wide surgical removal at the earliest possible opportunity. A portion of normal bone should be removed together with the tumour. It would seem that recurrence takes place in the vast majority of cases, but sometimes the secondary growths can be removed with success. Owing to the slow nature of the growth recurrence may not take place for many years, and relief from symptoms for long periods is recorded. Radium and deep X-ray therapy appear to be of little value.

CASE I.

A male patient, aged 33, whose previous health had been good, first came under observation on 21st November, 1931. He gave a history of increasing hoarseness for a period of six months prior to this date. Three months previously he had commenced to suffer from dysphagia. He noticed that phlegm tended to gather in his throat.

On examination his general condition was good. His sight, fields of vision, and fundi, were normal. No abnormality of the third, fourth, fifth, sixth, seventh, and eighth cranial nerves was detected. The palate was drawn up and deviated to the right. The tongue deviated to the left on protrusion. The left vocal chord was immobile. There was dropping of the left shoulder associated with wasting of the trapezius and sterno-mastoid muscles of that side. The Wassermann reaction was negative, and C.S.F. and X-ray examination yielded nothing of note.

This patient next came under observation on 18th October, 1932. He was then suffering from severe headache, generally frontal in situation, but occasionally occipital. He complained that his vision was dim and that he staggered when he walked. He vomited occasionally in the morning.

On examination the general condition was fair. The left optic disc was blurred in outline, the right normal. The pupils were unequal (left 4 mm., right 3 mm.). The left was inactive to light. The consensual reflex was present on both sides, but diminished on the left. The reaction to accommodation was greater on the right than on the left. Lateral nystagmus to either side was present. The fifth, seventh, and eighth cranial nerves were normal. The tongue was markedly atrophic on the left side, and on protrusion deviation to that side occurred. The palate was raised in the middle line. The voice was normal. Weakness was present in the left deltoid and trapezius muscles. The left sterno-mastoid appeared to have recovered. Dorsiflexion of the great toe occurred on the left side on attempting to elicit the plantar reflex. The response on the right side was normal. The gait was slightly reeling in character, with a tendency to deviate to the right. Romberg's sign was absent.

On 13th November, 1932, the base of the brain was exposed at operation. A tense, firm, encapsulated tumour was observed between the spinal cord and the foramen magnum. It was pressing on the left hypoglossal nerve. No attempt at removal was made, and the patient died next day.

The post-mortem examination was performed by Professor Young. A brief résumé of the salient facts is given :—

The dura mater and the cerebral hemispheres were normal in appearance. The cerebrum was removed by section of the peduncles in order to expose the posterior fossa without disturbance of the anatomical relationship. At this stage a rounded extradural tumour was observed on the surface of the basi-sphenoid to the left of the middle line. The cranial nerves eight, nine, ten, and eleven, were stretched over the surface of the growth on the left side, but they were not directly involved and could be readily detached. The spinal cord was then exposed, and was removed intact in continuity with the medulla, cerebellum, and brain stem. The tumour was now completely exposed. It was elongated from before backwards, and apparently extradural in its whole extent. It measured two inches by one inch. The base of the skull was removed with the tumour intact.

CASE II.

A male patient, aged 26, was first examined on 23rd November, 1933. He stated that he had had a slowly growing swelling in the left side of his neck for seven months. This caused pain and limitation of movement on chewing. There was no dysphagia. His throat was occasionally painful.

On examination a swelling about the size of a tangerine orange was found below the left ear. It was firm, smooth, and painless. A smaller similar swelling was observed below the right ear. The left tonsil was rough, and there was proptosis of the left eye. The pupils were unequal and slightly irregular. They reacted sluggishly to light and accommodation. X-ray of the skull yielded no definite information.

On 1st December, 1933, a portion of the tumour was removed for microscopic examination. On 14th December, 1933, deep X-ray therapy was commenced, and the patient was discharged two days later with instructions to return at stated intervals for further X-ray treatment.

CASE III.

A male patient, aged 60, was first seen on 16th January, 1934. Three years previously he had experienced difficulty in breathing through his nose. Eighteen months later he underwent an operation on his left nostril. He was unable to state the nature of this operation, and no record was available. He stated that for some time afterwards his breathing was improved, but gradually it reverted to its original state.

On examination he was only able to breathe through his mouth, and also suffered from some degree of dysphagia. The soft palate was displaced forwards by a mass about the size of an egg situated beneath the mucous membrane of the posterior pharyngeal wall. The mass was firm and painless, and the mucosa overlying it free from ulceration. X-ray confirmed the clinical diagnosis of retropharyngeal tumour.

At operation on 20th January, 1934, the soft palate was divided with the diathermy knife and the mass exposed. It was incised in the middle line vertically, and the contents removed piecemeal. The cavity was cleaned and the palate sutured. The patient was discharged from hospital five weeks later.

COMMENT.—Microscopically the three tumours present the characters of chordomata as tabulated above.

Case I represents an intracranial growth occurring in a typical situation, and producing the symptoms of increased intracranial pressure together with involvement of certain of the cranial nerves. The progress and duration of the case are such as might be expected from the previous records.

The site of origin of the growth in Case II is a matter for considerable speculation. Three possible theories might be advanced :—

- (a) That the primary focus is in the spheno-occipital region, and direct extension into the neck has occurred.
- (b) That a similar extension has taken place from a tumour originating in one of the cervical vertebræ.
- (c) That the mass in the neck represents secondary glandular involvement from a tumour in either site. No glandular structure was, however, observed in the microscopic preparations.

In Case III it is reasonable to assume that the growth has originated in the basisphenoid region, and extends directly behind the posterior pharyngeal wall.

The histological appearances in all three cases leave little room for doubt, although Ewing (1928) issues a note of warning against the possibility of confusion of this type of tumour with myxochondroma or "colloid" carcinoma. The microscopic picture in each case was, however, highly characteristic.

I am indebted to Dr. Allison, Mr. Irwin, and Mr. Purce for permission to publish these cases, together with the clinical details.

I should like also to acknowledge my indebtedness to Professor Young for his help and advice in the preparation of this paper.

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An Anencephalic Embryo of 25 mm. C.R. Length

By RICHARD H. HUNTER, M.D., M.CH., PH.D., M.R.I.A.

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ONE of the commonest malformations of the human foetus is anencephalus. Descriptions have been given of it since earliest times, but the first worker to give a clear account of it as "an arrested closure of the primitive neural groove" was von Recklinghausen¹ in 1886. Since this time his description has been accepted by all writers, and Faldino² in more recent times has confirmed his work. This description, however, makes no attempt to discuss the factors which inhibit the closure of the medullary groove, and many writers since von Recklinghausen have made tentative studies to discover the basic influence. Gaddi³ claimed that a constant feature was the absence or aplasia of the supra-renal body, a claim substantiated by other writers. Mandruzzatio,⁴ in a long description of an anencephalic human embryo of 48 mm. length "from vertex to the podalic extremity," states that there were present "noticeably hyperplastic supra-renal glands," the converse condition from that found by Gaddi. The other ductless glands were not discussed.

With this divergence of opinion on the subject of the condition of the supra-renals, and without any observations on the other glands, the appearance of a human anencephalic embryo of 25 mm. C.R. length in my laboratory was hailed as an opportunity to study, in this malformation, the condition of the supra-renal and other ductless glands, and to learn if support could be found for either of the views expressed by the many writers on the subject. It must be remembered that the development of this embryo is greater than its total length would at first suggest, owing to the absence of head development. If the head had been of normal size, the total C.R. length of the embryo would have been increased by about 10 mm. The stage of development of the structures of the embryo, apart from the abnormal regions, also suggest that it should be compared to an embryo of 35 mm., and as a consequence I have adapted this standard for purposes of comparison.

The embryo was first photographed (figs. 1 and 2), and then cut into transverse sections of ten microns thickness. The sections were then stained in alternate slides with hæmatoxylin and eosin, and with Mallory's triple connective tissue stain. These sections showed the supra-renal glands to be normal in development and differentiation for an embryo of 35 mm. C.R. length.* They also showed the

* In a full-term human anencephalic foetus which I was enabled to study, marked hypoplasia of both supra-renal glands was observed. This condition agrees with the description given of all full-term anencephalics in the literature. It would seem, therefore, that taken in conjunction with

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The embryo was first photographed (figs. 1 and 2), and then cut into transverse sections of ten microns thickness. The sections were then stained in alternate slides with hæmatoxylin and eosin, and with Mallory's triple connective tissue stain. These sections showed the supra-renal glands to be normal in development and differentiation for an embryo of 35 mm. C.R. length.* They also showed the

* In a full-term human anencephalic foetus which I was enabled to study, marked hypoplasia of both supra-renal glands was observed. This condition agrees with the description given of all full-term anencephalics in the literature. It would seem, therefore, that taken in conjunction with

thymus and the thyroid glands to be normal in size, shape, and position, as well as in their histological differentiation. The condition of anencephalus therefore, could not be the result of any lack of, or abnormal constitution of, the secretion of any of these glands. The pituitary, however, did show changes from the normal. There was complete lack of differentiation into the well-known sub-divisions of pars anterior, pars intermediate, and pars posterior. It was present in the form of a sparsely-filled sac, which most probably represented that part of the pituitary developed from Rathke's pouch, and it lay in the relationship to the body of the sphenoid bone which it normally occupies. Histologically the tissue which composed it showed a marked predominance of eosinophils over the basophils. This, with the lack of development of the parts which normally arise from the infundibular downgrowth from the floor of the mid-brain, were the only differences from the normal which were found in this or any of the other ductless glands. This anomaly of the pituitary, however, does not seem to have been the causative factor in producing the condition, as Frazer⁵ in a description of an anencephalic embryo states, "The pituitary body was smaller than that in a 28 mm. specimen, but almost as well developed."

The open cranial fossa of the embryo was filled with a mass of fibrous tissue rich in blood-vessels. But it would appear that at one period in development, normal medullary tissue must have existed, as both optic bulbs were present. These showed the retinal tissue of each bulb to be thrown into folds of tissue consisting of differentiating ganglion cells, similar to those seen in a normally developing embryo of similar size to the one here under discussion, and as described by Mann⁶ in an encephalic embryo of 28 mm. C.R. length.

Nerve fibres, however, were not present leading from the retina backwards to form nerve trunks. The retina, instead, extended postero-medially as a drawn-out, cone-shaped prolongation lying within a space lined by cubical-shaped cells, which were in continuity with the pigmented layer of the optic bulb, but which themselves showed no evidence of pigmentation. Around this cone-shaped zone of retinal tissue there was a mass of undifferentiated mesoderm continuous with that of the developing sclera. The optic muscles were present, and well developed. The inner ear was also at a stage normal for an embryo of 35 mm. length, as were the circulatory, respiratory, urogenital, and alimentary systems.

In the specimen which is here described, the skin of the neural tube area was closed in the thoracic, lumbar, and sacral regions, although the cartilaginous neural arches were ununited behind. But throughout these regions the spinal cord was represented merely by strands of fibrous tissue rich in blood-vessels, and closely similar in appearance to the tissue which lay in the open base of the skull. The latter condition would appear to correspond to the open form of spina bifida commonly found in the lumbar region of the body, as described originally by Kock,⁷ and in order to regularise terminology, it would seem better if it were termed encephalo-bifida instead of the more usual term of anencephalus. The vertebral

the condition of the supra-renals of the embryo here described, and the embryo anencephalic embryo of 48 mm. C.R. length described by Mandruzzatio, the aplasia of the supra-renals is the result of the anencephalus, rather than the anencephalus the result of the failure of the supra-renal development.

regions of this embryo, still following the terminology of Kock, corresponded to the sub-cutaneous or cystic type of spina bifida.

DISCUSSION.

Although agreement has been reached in considering the open form of spina bifida as being due to a failure of the lips of the neural groove to meet, fuse, and differentiate from the ectoderm, the causative agent at work is unknown; neither has any agreement been found to explain the closed or subcutaneous form of spina bifida. Several explanations have been advanced, but none of them, unfortunately, has been substantiated by direct evidence. One suggestion, made by Wheeler,⁸ is that the developing neural tube after closure remains attached to the ectoderm, and that under such conditions the neural tube tissue is inhibited from differentiating. The two conditions, open and closed spina bifida, by accepting this explanation, would thus be the result of the same pathological agent, but acting at different stages of development, i.e., open spina bifida beginning before closure of the neural tube has occurred, and closed spina bifida after closure has occurred but before the neural tube has separated from the overlying skin.

Such an explanation, however, does not touch on the real problem, which is the discovery of the pathological agent which inhibits or prevents these changes. The problem of the causes underlying defective development in general has been discussed by Mall,⁹ who, as a result of a study of 163 pathological ova, suggested that malformations might be due to "injurious influences of atypical environmental factors," due either to faulty nutrition of the embryo, or to imperfect elimination of the waste products of embryonic metabolism. Mall then makes the specific suggestion that the imperfect nutrition may be due to imperfect implantation of the ovum. Although such an explanation might be satisfactory for certain forms of malformations, it is not a satisfactory explanation for certain others, and the results obtained by experimental embryologists suggest that other agents are concerned with the production of spina bifida. These results point to a much earlier genesis: that organogenesis, normal and pathological, begins in the ovum itself, as the majority of defects produced experimentally are referable to changes which can be induced physically on certain specific portions of the egg. The embryonal parts would appear to be pre-localized in the cytoplasm of the ovum, and that they make their appearance, in the words of Lankester, "as a sequel of differentiation already established and not visible." Working on this assumption, Baldwin¹⁰ destroyed, by means of ultra-violet rays, specific areas on the equator of the white hemisphere of frogs' eggs, and these eggs resulted in the formation of spina bifida embryos. These experiments of Baldwin, being specific in localization, are a refinement of, and give support to, the earlier work of Morgan¹¹ and Hertwig,¹² who found that by experimentally damaging frogs' eggs, by means of chemicals, the closure of the blastopore was inhibited, and the process of closure of the medullary lips was as a consequence prevented. Many other experimental embryologists, working on the same lines, have obtained closely similar results. Stockard¹³ pointed to the chemical changes which are known to occur in the blood-stream of the mother in certain diseases, and he came to the conclusion that such abnormal chemical

substances might possibly be the causative factors which produced the physical changes in the ovum, and which resulted in spina bifida. This view, however, was purely a hypothetical one, and was unsupported by any experimental evidence. Werber,¹⁴ acting on the suggestions contained in Stockard's paper, tried to prove its truth by direct experiment. He treated eggs of fundulus with chemical substances which occur in the blood of diseased persons, and he found that butyric acid and acetone produced "monsters analogous or homologous respectively to those found in human and mammalian foetuses." These two substances are found in the blood of persons suffering from diabetes, but before they can be accepted as the causative agents in the production of malformations, it would be necessary to show that such monsters appear in confinements of those who have had acetone and butyric acid present in the blood. Malpas¹⁵ touched on this point in a paper based on the study of a series of 294 foetal malformations which had occurred in a series of 13,964 deliveries. The statistics given by Malpas do not include the question of diabetes (acetone and butyric acid), although the observations made point to abnormal biochemical changes in the blood. His conclusions are summarized as follows: "Malformations are mostly attributable to the interplay of environmental factors, that among mothers there was very definite evidence of poor physique, and in many there was a low standard of living."

SUMMARY.

It would appear the results obtained by experimental embryologists, taken in conjunction with the lack of evidence of any constant malformation of the ductless glands, or to constant malformations in cases of imperfect implantation of the ovum, that the condition of spina bifida must be due to some inherent defect in the egg itself, or to an injury produced physically or chemically to it before the primary divisions of cleavage have occurred. The possibility of biochemical changes in the blood-stream of the mother causing injury to the unsegmented egg cannot be excluded as being the pathological agent involved.

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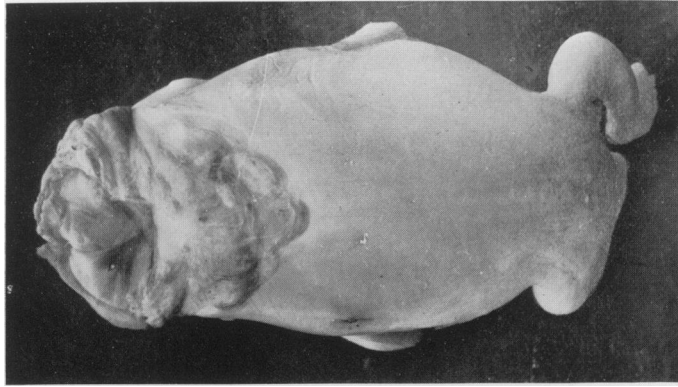


FIG. 1.
Dorsal view of anencephalic embryo of
25 mm. C.R. length.

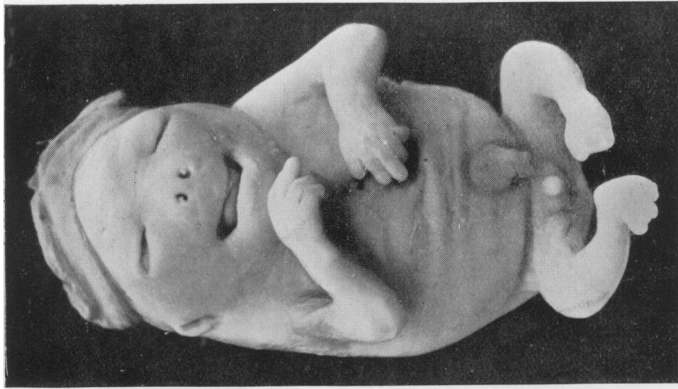


FIG. 2.
Ventral view of the same anencephalic
embryo.

Developmental and Generalized Bone Diseases

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A STUDY of the literature reveals considerable confusion as regards the classification of these developmental and generalized bone diseases, a confusion increased by the very elaborate and redundant nomenclature.

Conditions thought at one time to be individual and distinct entities, have been shown in the light of further knowledge to have common ground either in causation or in the pathology of the bony changes they produce, and it may be possible to classify them etiologically in the same group.

On the other hand, conditions totally distinct have been confused by a similarity of nomenclature, while individual writers have also been generous in bestowing new and individual titles on the same condition.

To a radiologist, dealing primarily with the shadows of the pathological changes in the bone, additional difficulty arises from the similar appearances shown in the radiograms of conditions of bone of quite different pathology, making the diagnosis from his standpoint alone difficult or perhaps impossible.

From time to time further information on these conditions is being elicited, leading us to alter our original ideas and original classification.

For these reasons, I have thought it worth while making an attempt to review the present position with regard to these bone conditions, and to try to classify them in the light of recent research and recent theories.

It would be impossible to cover the whole field of developmental and generalized bone diseases in a paper such as this, but I have taken several of the most typical conditions.

It is likewise impossible to get examples of all these rarer conditions in one's own work, and I am greatly indebted to Dr. Brailsford of Birmingham and Dr. Duncan White of Edinburgh for the loan of illustrative slides, and also to the former for the privilege of seeing the proof-sheets dealing with this subject of his recent book "The Radiology of Bones and Joints" while still in the press.

The developmental diseases are those in which the growing bone (growing either in cartilage or membrane) is affected. To understand their production we must know something of the method of bone growth.

Histologically, as far as I can gather, the growth of cartilage itself is still somewhat of a mystery.

Harris has attempted to simplify the analysis of the disorders of bone, and of bone growth, in terms of the three main processes :—(a) Proliferation in cartilage, (b) calcification in cartilage, and (c) actual bone formation.

Mitotic division of the cartilage cell had been shown in vegetable tissues and in invertebrate tissues. Harris demonstrated it in sections from the human embryo, prepared by a special method that had been used in the study of the chromosomes.

So much for the cartilage cell proliferation, but little is known of the fibrillar matrix or the chemical composition of the interstitial substance.

We do know, clinically, that the regenerative powers of cartilage are of a low order. If the cartilage of a joint is destroyed by trauma or infection, it does not reform as cartilage. Its place is filled by connective tissue, the underlying bone becomes eburnated, and ankylosis may ensue.

Harris, having succeeded in demonstrating mitosis; plotted out on one diagram the position of the mitotic figures in many slides, and found that the vast majority of the cells showing mitosis are arranged in a ring which he calls the "mitotic annulus." No mitosis occurs centrally in the head of the bone, where ossification of the epiphysis would commence, and none at the joint surface.

The actively growing cartilage cells lie in this ring. As we approach the epiphysis or the diaphysis from this zone, we get older cartilage cells, which become more crowded together, get less nutriment, pass into a stage of senescence, and calcify. Blood-vessels grow into the calcified matrix, bone is formed, and the pattern of the calcified matrix is followed by the bony trabeculae, reticulate in the case of the epiphysis, and in columns with longitudinal and transverse bars in the case of the diaphysis.

As we approach the articular surface of the joint, the cells become separated instead of congested, and flattened, and take on characteristics similar to those of the stratum corneum of the skin.

Following these lines, we see the unity of the growth of cartilage, giving rise from the one source to diaphyseal bone, epiphyseal bone, and hyaline cartilage.

To apply this principle to the study of perversions of bone-growth, we must see how the orderly progression of cartilage via calcified cartilage to bone is interfered with.

Anything interfering with mitosis in the fifth week of foetal life may lead to total failure of cartilage development and complete or partial absence of one of the long bones. Slighter interference may cause deformity of the bone-end, bone-shaft, or articular cartilage.

This interference with mitosis may be caused by nutritional errors, hereditary influences, or special susceptibility to toxic substances. Different times of onset of this cause of interference will produce different abnormalities, depending on the process of development active at the time. During the fourth and fifth weeks of foetal life the cartilage skeleton is actively developing. During the seventh to ninth weeks calcification of the cartilage of the long bones is the prominent feature. In the last two months of foetal life there is rapid growth of the long bones, while after birth there is calcification and ossification at the knee, hip, shoulder, and ankle.

ACHONDROPLASIA, or chondro-dystrophia foetalis is the first developmental abnormality to be considered in the light of this theory.

It is a type of dwarfism, and shows very definite irregular development, thus differing from true dwarfism, where the whole skeleton is small, but in perfect

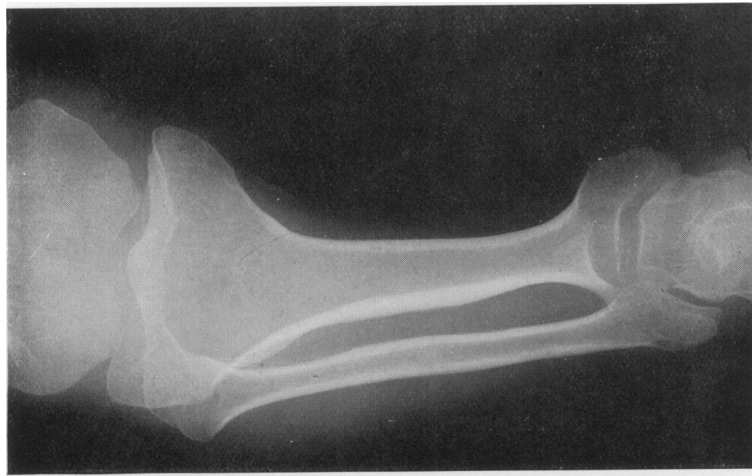


FIG. 1.

Achondroplasia in an adult male, showing typical short and thick bones.



FIG. 2.

Achondroplasia in an adult female, showing divergence of the metacarpals.

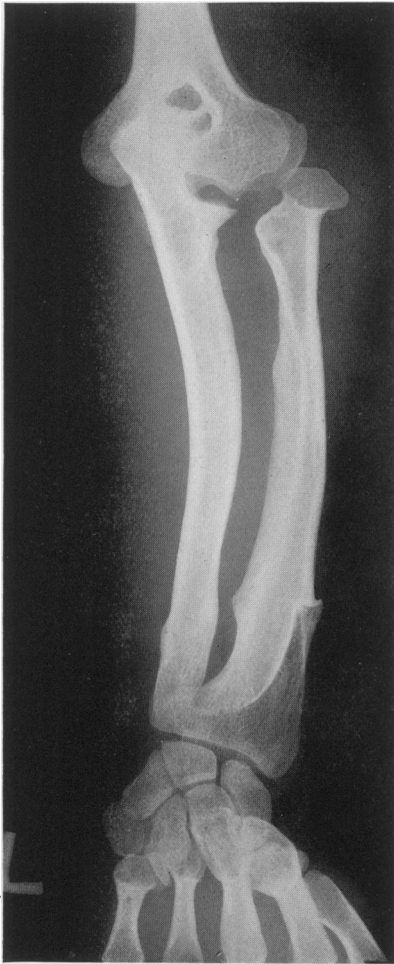


FIG. 3.

Hereditary Deforming Chondrodysplasia showing irregular growth of the forearm bones with dislocation of the upper end of the radius, and exostoses.



FIG. 4.

Osteogenesis Imperfecta in a girl of 11 with multiple fractures, showing expansion of the extremities of the long bones, and thin shafts of compact bone.



FIG. 5.
Osteitis Fibrosa Cystica, showing multiple cystic areas in the upper third of the humerus.

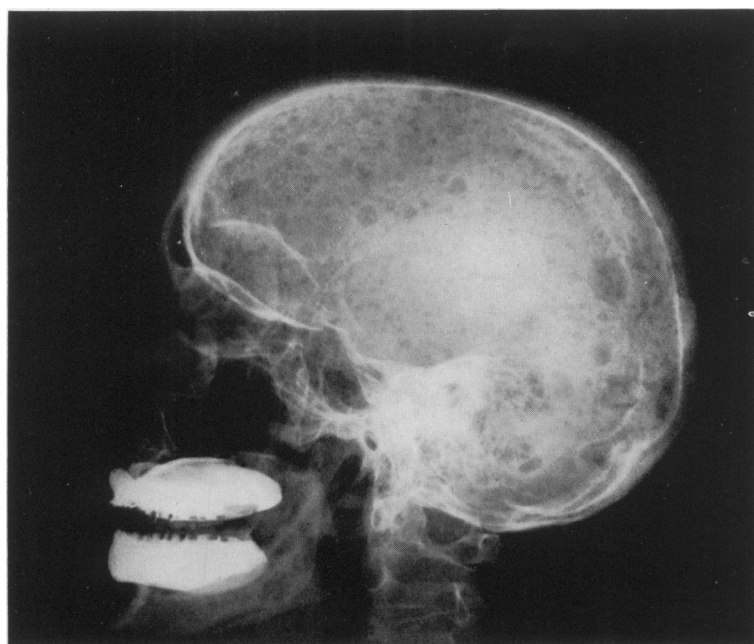


FIG. 6.
Multiple Myelomatosis, showing multiple "punched-out" areas in the cranial bones.

proportion, trunk and extremities showing equal development, the head large, mental capacity normal.

In achondroplasia we find stunting of the extremities; all the bones are present, but are thickened and shortened. In distinction to other conditions they do not bend, nor do they show any tendency to fracture.

Examples of the outward appearances of an achondroplasiac are seen in the representations of some of the old Egyptian gods, and in later times in pictures of "court fools," the nimbleness of wit and rather impish proclivities at times associated with the condition affording qualifications for that profession.

The name was given to the condition by Perrot in 1876. Kauffman in 1892 suggested the title chondro-dystrophia fœtalis, on the grounds that the condition was not due to absence of the cartilage, but rather to a nutritional disturbance. Lawford Knaggs holds that the condition is "a defective evolution of endochondral ossification at an early period of foetal life." Brailsford states that "the radiographic appearances suggest that the factor, whatever it is, producing the growth disturbance, must have ceased to act before ossification began," on the grounds that the bones are stunted only, and show no changes in the shapes of the diaphyses.

The skull is large. The bones of the cranium and face show no stunting. At the base, the basilar process is small and poorly developed, thus the skull is diminished in length and increased in breadth. The membrane developed bones are not affected.

The pelvis shows delayed development, and preserves the "funnel-shaped" type seen in the child.

The skin over the thighs and legs is loose, as if it had grown more rapidly than the bone it covers, and in the radiogram gives an appearance as if the patient were wearing plus fours.

The long bones are short and thick. The epiphyseal line is sharply defined, and closes earlier than normal. The bony trabeculæ in the shaft are abnormally arranged, there being fewer longitudinal and more transverse trabeculæ. The appearances are those of a bone telescoping on itself. According to Harris, these transverse striations represent a series of lines of arrested growth, with senescence of the cartilage cells, and calcification. The epiphyses ossify irregularly and slower than normally, more bone is laid down at the diaphyses, and this tends to surround the epiphyses like a funnel. The hands show what Pierre Marie described as the *main en trident* deformity.

In the foetal hand there is marked divergence of the metacarpals, which diminishes rapidly with development, until in the normal adult the fingers converge on the middle finger, which is in a straight line with its metacarpal. In achondroplasiacs this divergence remains, showing first a *main en quident*, which later becomes a *main en trident*, or occasionally *bident*.

Jensen in 1912 elaborated a theory for the causation of this and other developmental abnormalities, by infolding of the amnion and increased amniotic pressure,

prior to the eighth week of foetal life in this case, and at a later date in cases of cleido-cranial dysostosis.

Harris, however, claims to have shown that the essential feature present is mucoid degeneration of the cartilage. His sections appear to show widespread mucoid degeneration, especially in the ring where mitosis should be most active. Thus in these cases, instead of normal calcification followed by ossification, there is swelling of the cartilage cells, and conversion of the matrix to a viscid fluid. The matrix breaks up and the cavity collapses, giving an alteration in the shape of the epiphysis, and on the diaphyseal side areas of calcification separated by areas of mucoid degeneration. No reason has been suggested as yet for the presence of this mucoid degeneration. It may be inflammatory or nutritional.

HEREDITARY DEFORMING CHONDRODYSPLASIA.—This theory also applies to an associated condition or group of conditions described by Ehrenfried in the Journal of the American Medical Association in 1917, and labelled by him "hereditary deforming chondrodysplasia." Much confusion has arisen with regard to this condition, owing to the variations in its manifestations, and the great variety of names applied to it by different writers.

There are two main divisions of this condition, one with multiple exostoses, called by Keith "diaphyseal aclasis," and the other with multiple chondromata.

By the kindness of Mr. Ian Fraser, I have been able to get an example of the former from the Children's Hospital, Belfast, and of the latter from a doctor in Galway. I have also a case of the latter from Mr. Irwin's wards at the Royal Victoria Hospital, Belfast. While classifying these two conditions separately, they are very closely associated, and are both included by Ehrenfried in his "chondrodysplasia." One might consider them as branches arising from a central stem.

In the first division the salient features are multiple exostoses near the extremities of the diaphyses of the long bones, with other skeletal deformities such as deficiency of growth in length of certain bones with widening and irregularity of their extremities, and a sort of dislocation consequent on the shortening. The hereditary factor is very prominent, but though the disease exists in foetal life, it often does not come under observation till some of the bony deformities, which increase with skeletal growth, cause the patient to seek advice.

Keith states that the bones formed entirely within cartilage are unaffected. No exostoses are therefore to be found growing from the tarsal or carpal bones, the epiphyses of the long bones, the vertebral bodies or sternum.

The exostoses are commonly found at the upper and lower ends of the femora tibiae and fibulae, at the upper ends of the humeri and lower ends of the radii and ulnae, and in the long bones of the hands and feet.

The arms and legs are crooked and the stature often short. Pressure from the growths on neighbouring structures may produce pain or dysfunction.

Vacuolated areas, suggesting cysts, may be seen in the ulna, radius, or fibula. The ulna and fibula are short in proportion to the radius and tibia, with the result

that they do not take part in the wrist and ankle articulations, with a consequent ulnar deviation at the wrist and a valgus deformity at the ankle. The fingers and toes are often irregular in length.

In the second type, or multiple chondromata, similar deformities appear with a similar distribution, but the radiographic appearances are different. Cartilaginous overgrowths occur towards the extremities of the long bones, and may distort the growth of the epiphyses. This type is associated with the name of Ollier, and is sometimes called "Ollier's Disease."

Radiographically, defects in ossification at the ends of the long bones are found. As a result, one limb may be shorter than its fellow. One bone of the forearm may be short, as in multiple exostoses. Multiple chondromata are found in the phalanges, lower thirds of the radius and ulna, upper third of the humerus, pelvis, and both ends of the femur and tibia. Within the clear chondromatous areas small spicules of irregular ossification are often seen.

Cases of both these conditions have been described, showing secondary sarcomatous changes in the affected areas.

As to the causative factor in these conditions, many theories have been advanced. Virchow explained the condition as the action of some general disease, rickets, syphilis, or tubercle on the epiphyseal line, but this view has been abandoned.

It has been explained as a failure of the metaphysis to develop into a normal shaft because of some hereditary defect; that this defect may cause arrest of development and persistence of the perichondrium (the forerunner of the periosteum) at certain parts, producing the exostoses or chondromata and the deformities.

Harris would seem, however, to include this condition with achondroplasia as due to a mucoid degeneration of cartilage, probably at a different period of growth.

CHONDRO-OSTEO-DYSTROPHY.—Another allied dystrophic condition is that described by Brailsford in the *American Journal of Surgery* in 1929, and called by him "chondro-osteo-dystrophy."

Somewhat similar cases have been recorded by other observers. The radiograms show large gaps between the extremities of the bones at the joints, with subsequent pressure-deformities of the epiphyses and diaphyses. The active stage seems to end about the time of union of the epiphyses, but the deformities remain. There are irregular osseous nuclei in all the epiphyses, and marked deformity of the vertebral bodies with dislocation in the lumbar region, producing gross spinal deformity.

This dystrophy may affect the whole skeleton, and the resulting deformity depends on the severity of the lesions and the amount of pressure on them.

A similar case was examined histologically by Harris and Russell, and reported at the Section of Orthopædics, Royal Society of Medicine, as showing mucoid degeneration of cartilage, bringing this type into line with the previously described conditions.

Hypothyroidism produces definite bony changes. The main feature is delayed ossification. The epiphyses appear late and join late.

Radiograms of the pelvis of a definite case of this condition show no ossification of the femoral heads at the age of 3, and fragmented epiphyses resembling Perthe's disease at the age of 10.

We pass now to another group of bone conditions characterized by deficient or abnormal bone development, which for want of a better term we may group under the title "Malacic Diseases of Bone," a generic term used by the American Röntgen Ray Society at a discussion of these conditions a year ago, where an attempt was made to get away from looking on the different types as individual entities, and to classify them under their etiological factors.

The abnormal calcification present leads to weakness of the bone, bending, and a tendency to pathological fracture.

Some of them, such as osteitis fibrosa cystica, have been definitely related to endocrine gland dysfunction, and further investigation seems to be progressing in the line of such relationship in others of the group.

Others again, while not strictly belonging, in the light of present knowledge, to the group, are introduced because of their similar radiographic appearances causing difficulty in differential diagnosis.

OSTEOGENESIS IMPERFECTA.—Osteogenesis imperfecta, the first of the group, is one of the conditions suffering from an excessive subdivision into groups with consequent redundant nomenclature. It may appear at various periods of life, with varying severity and various clinical symptoms. Thus different writers describing cases have attached different titles to the disease, according to the appearances present in their case and the period of growth in which it appeared. Such titles are—*fragilitas ossium*, *idiopathic osteopsathyrosis*, *osteogenesis imperfecta*, *periosteal aplasia*, *maladie de Lobstein*, etc.

Knaggs, in a valuable paper in the *British Journal of Surgery*, describes four types—the foetal, the infantile, the adolescent varieties, and a type found in middle life.

Fairbank classifies from a pathological aspect into the thick-bone type, the slender fragile-bone type, and links up the marble-bone type, which will be described later separately, though presumably of the same etiology.

Most authorities seem to be agreed that the primary defect is mesoblastic, and that there is a deficiency of formation of osteoblastic cells to lay down the groundwork for bone. Chemically there is no fault. Calcium metabolism is normal, and in the vast majority of cases the blood-calcium and blood-phosphate estimations give normal readings.

Three cases have been reported which showed enlargement of the parathyroids, but the connection of this condition with endocrine dysfunction is very doubtful and far from proved.

Heredity in some cases seems an important factor, and the frequent presence of blue scleræ in association with the disease agrees with this. In other cases no

history of familial involvement can be found. Some writers on this account subdivided the disease into hereditary and non-hereditary forms.

I propose to consider all the sub-groups as branches of the one entity, as it seems most probable that the foetal, infantile, and adult groups are all manifestations of a similar process at different age periods.

Clinically the disease is associated with poor physical development and very marked brittleness of the bones, so that fractures occur on the least strain or pressure. Bending of the bones does not occur, except as the after-result of a fracture united in bad position. The fractures, though occurring readily, often show good union with strong callus. The child may be born with multiple fractures. Such cases are usually very severe, and the child is either stillborn or dies a few days after birth. On the other hand, some cases reach adult life or even old age, and the tendency to fracture seems to definitely lessen with advancing age.

In the cases in early life, radiograms show limb-bones broader and shorter than normal. They show no internal cancellous structure, and the cortex appears as a thin line. There are usually marked defects in the ossification of the skull, and the bony tables are very thin. The thorax is often compressed, and the ribs bent by multiple fractures. Multiple limb fractures, with or without callus repair, are present. Later a marked degree of osteoporosis is seen, and there is an expansion of the extremities of the long bones, the middle of the shafts being of normal width. As age advances, the osteoporosis persists, the extremities of the long bones still expand, but the shafts seem to shrink, so that about puberty one gets a very thin shaft, with compact cortex, little or no medulla, with extremities which, though about normal in size for the patient's age, seem, by contrast with the shaft, to be greatly expanded. If the patient survives to adult life, the shafts of the long bones seem to be formed of slender rods of compact bone with no medulla, and the extremities consist of coarse, rarefied, cancellous bone. Bromer shows a case which in most respects is typical of osteogenesis imperfecta, but which also shows wide, dense bands near the extremities of the long bones, resembling marble bones or Albers-Schönberg's disease. This seems to justify the conclusion that there is a definite etiological link between the two conditions, as Fairbank maintains.

ALBERS-SCHÖNBERG DISEASE.—This very rare condition was first described by Albers-Schönberg in 1904. For ten years after that no other case was described.

Only about forty cases have been reported altogether. Radiography is necessary for a diagnosis.

Clinically the same proneness to fracture as in osteogenesis imperfecta is found, but the radiographic appearances are very different. The cancellous structure of the bones is replaced by a dense homogeneous shadow obliterating the medullary cavity.

Owing to the radiographic density, the title "marble bones" was used, but with the marked brittleness and the chalky consistency of the bone on section, Pirie of Montreal suggests that "chalky bones" would be a better name.

Cases have been reported from the ages of 3 to 48. It is commonest between 12 and 20.

The condition shows a progressive anæmia due to loss of the medulla. Blood-calcium is usually normal, but cases with raised blood-calcium have been reported. The skull bones are dense, the pituitary fossa contracted, and the cranial foramina may be narrowed, causing nerve pressure.

Familial history is frequent, and a history of consanguinity was found in several cases.

The knees show an expansion of the ends of the tibia and femur of dense bone. A similar condition is seen at the upper end of the humerus and the lower end of the radius.

These expansions show linear strands of dense bone parallel to the epiphyseal line. In the phalanges, linear cracks vertical to the epiphyses are seen, an appearance, I think, first described by Brailsford.

In every case the whole expansion shows even density. As we approach the middle of the shaft the bony appearances approach normal.

The ribs show even and uniform density, but are normal in shape.

The pelvis shows marked density about the acetabula and sacro-iliac joints.

The vertebræ show an upper and lower plate of dense bone with a median portion of cancellous bone.

The tarsal bones show a dense periphery with clearer centre—most dense at the points of greatest strain.

No definite theory of causation has yet been propounded, but it seems probable that Fairbank is correct, and that the condition is closely associated with osteogenesis imperfecta.

OSTEOPOCKILIE.—Osteopockilie is another example of abnormal ossification also described by Albers-Schönberg.

In this condition we find bones of normal size and development, but the cancellous bone contains multiple little islands of compact bone. This condition is not uncommon. It is symptomless. Its etiology is unknown, but it is probably an hereditary abnormality with disturbance in the ossification of cartilage during development.

OSTEITIS DEFORMANS.—Another generalized disease of bone, rather more common in occurrence, is osteitis deformans, or Paget's disease.

This occurs in later life, usually after 45, and is slowly progressive.

The causation is still unsettled, many theories having been elaborated, and recently Mayer-Borstel and also Hirsch have claimed that it is akin to osteitis fibrosa cystica (which we know is due in most cases to hyperparathyroidism), and that the two conditions are different manifestations of the same disease.

Radiographically we can distinguish two types. In both the fibrous element of scaffolding in the normal bone has been ossified, but in one the matrix has been absorbed, and in the other the cancellous space thus left has been filled in with calcium deposit. We can call these the fibrous type and the sclerotic type.

The earliest signs are usually found in the pelvis. The femora and tibiæ are soon affected, and typical changes are seen in the skull. Bending of the long bones occurs in the fibrous type, and is also found in the sclerotic type, having occurred before calcification set in. The bones are brittle, and pathological fractures are common. The affected bones show thickening of the cortex, and the bony trabeculæ are arranged in longitudinal strands.

Cystic areas are often found in the new cortical bone.

The cranial bones are greatly thickened, and the radiograph shows a blurred outline with fuzzy outer and inner tables.

The histological condition seems to be an absorption of bone, which is replaced by ossification of a lower order, a formation of new coarse membranous bone. Thus we get enlargement, elongation, and weakening of the bone, with consequent bending or fracture.

The appearances of individual bones may simulate those found in metastatic malignant deposits.

OSTEITIS FIBROSA CYSTICA.— The disease held by some to be of similar etiology is osteitis fibrosa cystica, or von Recklinghausen's disease. Here more is known of the causation, as in many cases, at least, it is associated with a parathyroid adenoma, and is definitely consequent on this, as removal of the parathyroid improves the bony condition. The blood-calcium is raised and the blood-phosphorus lowered. It is a disease of later life and, as Harris says, "it shows that calcium metabolism is of great importance in adult life, as well as in the period of active growth."

The condition of the bones was first described by von Recklinghausen in 1891. In 1901 Askanazy called attention to its connection with parathyroid pathology, a connection established by Mendyl in 1926, who obtained definite improvement in a case after removal of a parathyroid tumour.

The disease may occur at any age, but is commonest in women between 30 and 60.

The first complaint is usually of "rheumatic pains" or possibly of a spontaneous fracture. Radiograms show marked general osteoporosis of bone, with cyst-like formations.

The skull usually shows a characteristic picture, giving a mottled appearance due to small opacities of bone condensation, with intervening areas of rarefaction. The outline of the outer table is poorly defined. Those who claim a relationship between this and Paget's disease hold that the skull appearances are similar, but in the latter the sclerosed patches are usually much larger.

The vertebral bodies show marked calcification. The periphery is dense, but the internal structure scarcely visible.

The pelvis shows a coarse cancellous trabeculation, and is usually distorted by the weakened bones yielding to pressure.

In the long bones the cortex is thin, the internal structure is ill-defined, and cysts may occur in any of the long bones and in the bones of the hands and feet.

The extremities of the long bones show coarse strands in the long axis of the limb, with absorption of the intervening trabeculæ.

Pathological fractures are common, and renal calculi are found in a large number of cases.

Examination of the cystic areas shows that the growths are polycystic, and that giant cells predominate in the surrounding tissue.

MULTIPLE MYELOMATOSIS.—A condition which we must take into account in this connection is multiple myelomatosis; partly because the radiographic appearances are in many cases very similar to those found in *osteitis fibrosa cystica*, causing diagnostic difficulty, and partly because some writers, recently, hold that multiple myelomatosis is a malignant end-stage of *osteitis fibrosa cystica*.

Morse of Detroit says: "My own study of our cases of multiple myeloma has convinced me that multiple myeloma is not a disease of the bone-marrow system, that it does not come from the blood-forming cells, nor is related to them in any way; but that it is a primary osteoblastic tumour, arising from the primitive osteoblasts, and at present should be considered as a malignant manifestation or a malignant end-stage of *osteitis fibrosa cystica*."

The condition was first described in 1845 by William McIntyre, who was associated with Dr. Bence Jones, who in this case first described what are now known as the Bence Jones bodies excreted. It is a disease of later life, more frequent in males. Nothing is really known of its causation. Various theories have been elaborated to fit individual cases, but none of them seems capable of general application.

The onset is usually marked by intermittent rheumatic pain in the back and down the legs, accentuated by movement and with acute exacerbations. Pathological fractures are common. Tender tumours appear on the bones, especially on the ribs, sternum, clavicles, and spine. The skull and extremities are later involved. The patient walks with deliberation and with a spastic gait.

Paraplegia may be the result of vertebral tumours or intercostal neuralgia from rib tumours.

The radiograms show round, well-defined, punched-out areas in the bone. There is no bending other than the result of malunited fractures. The skull shows multiple punched-out areas, but no increase in the width of the tables.

The pelvic bones show rarefaction and cystic areas, but no deformity.

In our case multiple renal calculi are shown, which tends to favour Morse's contention, as these are common in *osteitis fibrosa cystica*.

As a rule, blood-calcium and phosphorus are normal, though cases of hypercalcæmia have been reported. A blood-count reveals nothing typical, except anæmia.

Bence Jones bodies are found in about seventy per cent. of the reported cases.

The tumour is soft and cuts easily. It is composed of gelatinous dark red tissue, and the cells are of the plasma type, but do not usually take the Pappenheim stain. Most authors seem uncertain as to whether to classify these cells in the plasma or myelocytic series.

There are many other groups that might be included under this series of malacic diseases of bone. I have not touched on the general malignant metastatic conditions,

such as carcinomatosis of bone, nor on renal rickets, where the bones are so soft that they can be cut with a knife and practically no calcium remains in the skeleton. In this condition the primary fault is probably phosphorus retention with secondary hyperparathyroidism to combat this, with the result that the blood-calcium is increased at the expense of the calcium in the skeleton. Then there is the large group where there is a primary defect of calcium absorption and deposition, rickets, and, at a later stage of life, osteomalacia, the one the expression of this factor in the growing bone, the other in the bone of the adult.

Rickets is a common condition, and its clinical and radiological appearances are well-known, so I will pass it over and just say a word about osteomalacia.

Osteomalacia is rarely seen here, but it is common in India and China. It usually appears about puberty, and is intensified in pregnancy by reason of the extra call made at that time on the body calcium. By reason of the pelvic deformities produced, it is a serious factor in midwifery. Pathologically, the condition is a decalcification of the skeleton and replacement of bone by fibrous tissue. The association with rickets is close, and Harris has shown in monkeys a gradual transition from rickets to osteomalacia.

There is general osteoporosis and softening of the bone, with bending following the application of pressure. The blood-calcium is decreased.

The pelvis shows the most typical deformity. The sacrum is pushed downwards and forwards, taking up a horizontal position. The bodies of the ilia are depressed and flattened, while the femoral heads push in the lateral walls, giving the typical triradiate deformity.

The vertebral bodies become bi-concave by pressure of the expanded inter-vertebral discs. Pseudo-fractures may occur, and multiple bone-cysts.

Clinically, there is pelvic pain and disturbance of gait. The old theory of osteomalacic areas is now forsaken. The disease occurs whenever conditions of light and vitamin D deficiency with increased calcium demand are unfavourable.

CASE REPORT

CASE OF INTESTINAL OBSTRUCTION due to strangulation of a loop of small intestine in an opening of the left broad ligament.

By J. J. MORIARTY, M.B., B.Ch.,

Assistant Surgeon, Mater Infirmorum Hospital, Belfast.

THIS type of obstruction would appear to be particularly rare, and in view of the paucity of physical signs the case described below is of somewhat unusual interest.

The patient was a married woman, aged sixty-two years, mother of five grown-up children. Her previous history as regards illness and operation was negative. Her appetite and digestive functions were normal, but she was habitually slightly constipated. On Christmas Day, before dinner, she began to notice slight, crampy

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The patient was a married woman, aged sixty-two years, mother of five grown-up children. Her previous history as regards illness and operation was negative. Her appetite and digestive functions were normal, but she was habitually slightly constipated. On Christmas Day, before dinner, she began to notice slight, crampy

abdominal pains in the hypogastric region. She had taken a dose of Epsom salts the previous morning, and her bowels had acted shortly afterwards, and they also acted, though to a lesser extent, on Christmas morning. The pains continued all evening, and she vomited once or twice. Never having had an experience of this kind before, she realized that it was advisable to seek medical advice. She was seen by a doctor the same evening, who found her condition as follows: Pulse 100; temperature normal; tongue clean and moist; no abdominal tenderness, distension, or rigidity; P.V. and P.R.—nothing abnormal detected; hernial orifices normal.

The patient volunteered the information that she had passed no flatus by the bowel all day.

A high enema was ordered, to be repeated in a few hours if there was no result.

At nine o'clock the following morning, as the enemata had produced no evacuation of her bowels, and as the griping pains continued, the patient was sent to a nursing-home for immediate operation as a case of acute intestinal obstruction.

The abdomen was opened by the usual right paramedian incision. The cæcum was not distended, so localizing the seat of the obstruction to the small intestine. On commencing to trace the ileum up from the ileo-cæcal junction, it was found that at about eight inches from that point, the ileum was held down in the pelvis; the cause of the obstruction was due to a loop of ileum about ten inches long having passed through an opening in the left broad ligament. The loop was withdrawn without difficulty, and it showed a definite though slight constriction-ring at the point where it was grasped by the opening in the broad ligament. There was a small purple patch about the size of a sixpence at the mid-point of the loop, otherwise the bowel showed all the appearances of viability.

The opening in the broad ligament was carefully inspected as to the possibility of its being a result of adhesions, but it had none of the appearances of such a condition, nor were there any adhesions in the pelvis, nor any evidence of disease of any of the pelvic organs. The opening was then obliterated and the loop of bowel again examined, when the purple patch was found to have changed to a lighter red colour. A marked ileal band was dealt with and the abdomen closed. The patient made an uneventful recovery, and returned to her home feeling fit and well on the thirteenth day after her operation.

Two similar cases were reported by Robert Janes, Department of Surgery, University of Toronto, in the *British Journal of Surgery*, October, 1929. He comments on the rarity of the condition, and states that he has only been able to trace one other case (reported by H. A. Barr in the *Medical Record and Annual*, 20th June, 1928). He suggests that the openings in the ligament may have been in some way related to pregnancy, as there was no evidence of any antecedent inflammatory condition.

In both of his cases the physical signs were not definite, i.e., in one case tenderness in the fornix and in the other a tender cystic mass behind the uterus. In the case now reported, the patient probably owes her life to the fact that she observed that she was passing no flatus by the bowel despite griping pains, and thereupon consulted her doctor.

A Case of Spontaneous Rupture of the Uterus

By C. H. G. MACAFEE, M.B., F.R.C.S.I., F.R.C.S.ENG., F.C.O.G.

CASES of uterine rupture, either spontaneous or traumatic, are relatively rare, and the following case shows some interesting features.

Mrs. H., aged 39, was admitted to the Maternity Hospital, Townsend Street, on 7th February, 1932, about 5 p.m. She was pregnant for the tenth time, and had eight living children and one miscarriage.

All her previous confinements had been normal; the last child was two years old.

During the pregnancy with which we are dealing, she seems to have been in her usual health, and the date of her expected confinement was 21st February, 1932, i.e., she was within fourteen days of full time.

At 7 p.m. on 5th February, 1932, while she was standing in her kitchen, she took "labour pains," which lasted only for one or two hours. The membranes ruptured at the first pain. After the pains passed off she complained of "soreness in her stomach," and on admission this was still her main complaint.

She was not seen by a doctor until midnight on 5th February, 1932, as her husband had not been able to go for him. At that time she was given an injection "to bring the pains on stronger" (her own words), but her condition was unchanged. Throughout the day of the 6th February, she was still complaining of "soreness in her stomach," and at midnight was given a hypodermic of morphia.

On 7th February, 1932, her general condition was much worse, and she was admitted to hospital that afternoon, travelling over twenty miles in a motor.

On admission the patient looked very ill—P. 140, T. 100°F., and she groaned on the slightest movement. The abdomen was extremely tender all over, and the patient resisted any attempt to examine her.

The foetus was lying transversely, the head being in the right iliac fossa. The foetal parts were very distinctly felt through the abdominal wall, and there was an indefinite mass felt in the left iliac fossa. There was a bloodstained vaginal loss, but no presenting part could be felt, and the cervix barely admitted one finger.

At operation the abdominal cavity was full of blood and blood-clot, the child (a large full-time male) was free in the cavity, and the placenta was in the left iliac fossa. There was an extensive rupture of the uterus on the left side involving the lower uterine segment and spreading upwards into the upper segment, and down to the vault of the vagina, just missing the left uterine artery. The foetus had evidently passed into the broad ligament, and then burst into the peritoneal cavity, stripping the peritoneum away from the lateral pelvic wall, and exposing the iliac vessels throughout nearly their whole course.

The foetus and blood-clots removed from the peritoneal cavity had a very offensive odour.

A subtotal hysterectomy was performed, a gauze drain being passed into the vagina to drain the parametria. There was difficulty in peritonizing the pelvic floor

on account of the amount of destruction and stripping of the peritoneum, but this was done as well as possible.

The abdomen was closed in layers, a rubber drainage-tube being inserted down to the bottom of the pelvic cavity.

The patient was given 50 c.c. of a fifty per cent. solution of glucose intravenously during the operation, and left the table in a much better condition than when the operation started. She had an uninterrupted convalescence. The temperature was above 100°F. on two occasions only, and she was discharged on the nineteenth day.

This case, and three others mentioned later, raise some interesting questions from the etiological and therapeutic standpoints.

In the case described, an unhealthy multiparous uterus was the most likely cause. The rupture almost certainly occurred five hours before pituitrin was administered.

Hyaline, granular, and fatty degeneration and fibrosis of the uterus have been given a prominent place by various writers, but Mahfouz Bey¹ has been unable to confirm any of their findings.

Munro Kerr² feels convinced that degeneration of the uterine wall is frequently present, and predisposes to the accident, for a slight fall or cough or violent movement by the child have been the only apparent exciting causes in some cases.

The most important predisposing factors to either spontaneous or traumatic rupture are multiparity, scars in the wall of the uterus, degenerations, maldevelopment and malposition of the uterus, and the effect of certain drugs.

The greater liability of the multiparous uterus to rupture is due to the prevalence of pendulous abdomen and malpresentations in multiparous women.

In this case we have no evidence of any determining factor, e.g., transverse presentation, but the fact that the previous eight full-time babies were born spontaneously and with normal presentations is no guarantee that the ninth child was lying in a normal position.

The probability is that the child was in a transverse position at the commencement of labour, and after ten pregnancies it is almost certain that the patient had a pendulous abdomen. This case should be a warning to every obstetrician to watch the multipara, especially the elderly multipara, as carefully as the primigravida.

The next interesting point about the case is the length of time between the occurrence of the rupture and the time of operation, viz., forty-eight hours.

The prognosis before operation looked almost hopeless, and yet the patient recovered. For this one has to thank the patient's powers of resistance rather than anything else.

This raises the question of whether it may not be better to delay operation for some time after rupture, rather than operate immediately after the catastrophe has occurred. I do not suggest leaving a case for forty-eight hours.

I have operated upon four cases of spontaneous rupture with no maternal deaths, and have seen three cases of traumatic rupture who have died. One case ruptured just before admission to hospital (she had had three previous Cæsarean sections by

another surgeon, and had ruptured on two occasions before); the rupture was in the upper end of the scar in the uterus and the child was alive.

One case was operated upon seven hours after rupture (another case of ruptured Cæsarean section scar); one case operated on twelve hours after rupture, this case being almost a replica of the case described above; and the case described, forty-eight hours after rupture.

Mahfouz Bey¹ states that the prognosis in cases of rupture of the uterus does not depend on the method of treatment. It is influenced to a greater degree by the amount of hæmorrhage which has occurred before treatment was begun, and by the traumatism caused by previous attempts at delivery, and, above all, by the degree of sepsis and the severity of the shock.

The four cases mentioned had no traumatism apart from that due to the actual rupture, and three, although shocked, had recovered from the severe initial shock. The absence of severe handling per vaginam was, I am sure, a great factor in the recovery of all these cases. Another point in their favour was that in the three seriously-ill patients, the uterus had completely emptied itself, permitting good contraction and retraction, and limiting the hæmorrhage.

All the cases of traumatic rupture which I have seen, three in number, have died, but they were all very shocked, had had severe hæmorrhage, and were almost certainly infected as a result of improper vaginal manipulations.

In the presence of severe shock and sepsis, which are the rule in most cases of traumatic rupture, laparotomy is attended with grave results.

Conservative treatment, i.e., plugging the tear, is not advised where the child has escaped from the uterus, but even in these cases, where there is no further internal hæmorrhage, a delay of a few hours to allow the patient to recover from the immediate shock would seem advisable.

I realize that the number of cases is too small to allow of any hard and fast conclusion being drawn, and on looking over the literature one does not find any expression of opinion on this point.

Davis³ says that hysterectomy is the operation of choice, and the sooner this operation is performed after rupture has occurred the better are the chances of recovery. He also states that while rupture through a Cæsarean section scar in a subsequent pregnancy is a serious accident, the prognosis for the mother is far better than in the other forms of rupture.

Hillis⁴ reports four cases of spontaneous rupture of Cæsarean section scars, operated on six hours, twelve hours, and forty-eight hours after rupture (one case time not stated), with recovery in all cases.

Sherrill⁵ reports one case seen four hours after rupture (the rupture not recognized), where operation was performed thirty days later (the child having been delivered per *vias naturales*. He says "that prompt recognition and surgical intervention will show a marked decrease in the mortality."

Wertenbaker⁶ reports two cases of rupture after administration of pituitary extract; one operated upon three and a half hours after rupture who died, and one five hours after rupture who lived.

Cornell⁷ reports one case of spontaneous rupture of a Cæsarean section scar operated on five or six hours after rupture with recovery.

There is a great fallacy in forming opinions from reported cases, because one is more likely to hear about successes rather than failures, but from my personal experience, which is admittedly small (four cases), and from the above-mentioned literature, I feel that if a case is on the point of rupture, operation should be performed at once, but if the case is seen after rupture has taken place, it is probably safer for the patient to delay operation for a few hours to allow of recovery from the immediate shock. This line of treatment is safe in those cases where the child has been completely expelled from the uterus, because further hæmorrhage is not liable to occur; and in those cases where a large vessel has been torn, or where the child is partially expelled, it is not likely that any operation will be performed in time to save the patient, and I think that the patient would be dead, or dying, before a surgeon was able to perform the operation.

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REVIEWS

THE STUDENTS' POCKET PRESCRIBER. By David M. Macdonald, M.D., D.P.H., F.R.C.P.S. Tenth Edition. 1934. Edinburgh: E. & S. Livingstone. pp. 263. Price 3s. net.

EVERY final-year medical student, and indeed many qualified practitioners, will find a gold-mine of useful prescriptions in this little book. They will also find in it a number of valuable hints on prescribing, on changes in the B.P. official titles, on synonyms for drugs, incompatibilities, incubation periods of infectious diseases, and diet. The formulæ given have all stood the test of time, and proved their worth not only by the author of this book, but the many generations of young practitioners who have used them since the first edition was published in 1882. The present is the tenth edition, and it can be as strongly recommended for study as any of its earlier editions.

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HANDBOOK OF THERAPEUTICS. By David Campbell, M.C., M.A., B.Sc., M.D., F.R.F.P.S. Second Edition. 1934. Edinburgh: E. & S. Livingstone. pp. 444. Price 12s. 6d. net.

THIS book makes no claim to give a complete account of therapeutics; it aims only at furnishing the senior student and young practitioner with a reasoned criticism of therapeutic measures, and with a simple account of what is, in the author's opinion, the best treatment of individual diseases, and right well has its author succeeded in his aims. It begins with a short account of the general hygiene of the patient, passes to the methods of writing prescriptions, and then discusses the general and local effect of medicines. The treatment of individual diseases is then considered, but Dr. Campbell does not confine himself to mere lists of prescriptions: where pain is a symptom he first indicates the methods employed to relieve it, which experience has proved of value; he discusses the question of diet for the patient, and the treatment of the complications which may arise, the general medicines employed, and finally any special treatment such as sera, vaccines, and the so-called specific drugs. There is also a useful chapter on physical methods of treatment, and a short but useful chapter on dietetics. This extraordinarily useful book will be of inestimable value to young practitioners and senior students, not only because it will provide them with a fund of information necessary for general practice, but because of the reliability of its information.

—R. H. H.

COMBINED TEXTBOOK OF OBSTETRICS AND GYNÆCOLOGY. By J. M. Munro Kerr, M.D., F.C.O.G., and co-workers. 1933. Edinburgh: E. & S. Livingstone. pp. 1,100. Illustrations 497. Price 35s. net.

THE "Combined Textbook of Obstetrics and Gynæcology" by four Scottish authors reappears in its second edition after ten years, having been reprinted once in the interval. The book has been largely rewritten, and three new chapters, which increase its value, have been added. These chapters are on maternal mortality, the infant in its first month, and radiology in relation to obstetrics and gynæcology. The teaching is sound and orthodox, and only well-tried procedures are recommended. It is rather surprising, however, to find *accouchement forcé* described in a modern textbook. In the treatment of asphyxia neonatorum, the administration of carbon dioxide and oxygen is dismissed in a brief footnote, cardiac and respiratory stimulants such as coramine and lobeline are not mentioned, while artificial respiration is described and illustrated. In the treatment of post-partum hæmorrhage and obstetric shock the solution recommended for intravenous injection is normal saline, which is much inferior, in the reviewer's opinion, to either gum acacia or fifty per cent. glucose solution. The authors recommend cutting down on a vein for this procedure, but this should rarely be necessary. Apart from these few minor blemishes, the book is well written and well illustrated, and the authors are to be congratulated on having produced such an outstanding work.

—H. C. L.

PRACTICAL POINTS IN EYE SURGERY AND DRESSING. By H. E. Jones, M.R.C.S., L.R.C.P. 1933. London: John Bale, Sons & Danielsson. pp. 28. Price 2s. 6d.

THIS little book is neither an abridged textbook nor an examination cram-book. It is rather an attempt to supply the young medical man with answers to some of the innumerable questions which arise to puzzle him in his early days as a house-surgeon. It begins with a short account of the essential points to note in examining an eye-case, gives a description of the instruments used in examination, and a discussion of the simpler forms of treatment of such cases. There are three useful tables on the examination and treatment of the commoner diseases and accidents encountered in eye practice, tables which might be memorized with profit by house-surgeons in the out-patient departments.

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HANDBOOK OF THERAPEUTICS. By David Campbell, M.C., M.A., B.Sc., M.D., F.R.F.P.S. Second Edition. 1934. Edinburgh: E. & S. Livingstone. pp. 444. Price 12s. 6d. net.

THIS book makes no claim to give a complete account of therapeutics; it aims only at furnishing the senior student and young practitioner with a reasoned criticism of therapeutic measures, and with a simple account of what is, in the author's opinion, the best treatment of individual diseases, and right well has its author succeeded in his aims. It begins with a short account of the general hygiene of the patient, passes to the methods of writing prescriptions, and then discusses the general and local effect of medicines. The treatment of individual diseases is then considered, but Dr. Campbell does not confine himself to mere lists of prescriptions: where pain is a symptom he first indicates the methods employed to relieve it, which experience has proved of value; he discusses the question of diet for the patient, and the treatment of the complications which may arise, the general medicines employed, and finally any special treatment such as sera, vaccines, and the so-called specific drugs. There is also a useful chapter on physical methods of treatment, and a short but useful chapter on dietetics. This extraordinarily useful book will be of inestimable value to young practitioners and senior students, not only because it will provide them with a fund of information necessary for general practice, but because of the reliability of its information.

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—H. C. L.

PRACTICAL POINTS IN EYE SURGERY AND DRESSING. By H. E. Jones, M.R.C.S., L.R.C.P. 1933. London: John Bale, Sons & Danielsson. pp. 28. Price 2s. 6d.

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MATERNAL MORTALITY AND MORBIDITY. By J. M. Munro Kerr, M.D.,
F.R.F.P.S., F.C.O.G., Regius Professor of Midwifery, University of Glasgow.
1933. Edinburgh : E. & S. Livingstone. pp. 382. Price 25s.

A book by such a distinguished obstetric surgeon and teacher as Professor Munro Kerr will attract widespread interest and attention. The author has treated the subject of maternal mortality and morbidity in a most thorough manner, and he deals with the problem from every conceivable angle; all the conditions affecting maternal mortality are reviewed, and the death-rate in these islands is compared, as far as possible, with other countries. The chapter on ante-natal care is particularly good, and the author has stressed a point mentioned by other writers, that ante-natal care as at present carried out is often quite inadequate. In this latter chapter several plates are included of abnormalities detected by X-ray photography, such as anencephalus, breech presentations with extended legs, twins, etc.

Obstetricians who are accustomed to the use of sedatives during labour will disagree with Professor Kerr's view that the less sedatives are used in a normal case the better it is for the patient. Sedative drugs in midwifery can be abused, and often are, but if they were used more frequently there would be fewer obstetrical disasters such as "failed forceps," which he and all teachers of obstetrics deplore. As might be expected in a first edition, there are a number of printer's errors, which will, presumably, be corrected in subsequent editions. The book will be invaluable to all interested in the subject, especially practising obstetricians and public health authorities.

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BRITISH MEDICAL ASSOCIATION—BELFAST DIVISION

THE Belfast Division began a very successful session on 26th October, when Dr. S. R. Hunter introduced his successor to the chair, Dr. George Lyttle. Dr. Lyttle spoke on "Some Experiments in Functional Sterility." He outlined his treatment, by large doses of ovarian extract, of such cases as had no causal lesion calling for operative interference. He had been successful in the majority of his cases, in all but one of which dilatation and curettage had been tried without success.

On 9th November, the Division welcomed a very distinguished Queensman in the person of Colonel W. P. McArthur, Professor of Medicine at the Royal Army Medical College. Colonel McArthur gave a brilliant paper, based on his work on cysticercus infection in man. Cases were rare, and were usually soldiers who had served in India. Subcutaneous nodules appeared, which on excision proved to be cysts, and these, developing in the brain, produced fits, usually of an epileptiform type, and later mental deterioration and insanity. When the larvæ died, calcification occurred, and numerous radiographic slides were shown illustrating this condition.

The following month Dr. Hennessy, our Irish secretary, paid us a welcome visit, addressing us on "The Position of the British Medical Association in Ireland," and on 8th February, Dr. S. B. Boyd Campbell gave a clear and exceedingly practical account of "Modern Treatment of the Anæmias," which was much appreciated by the members present. The same meeting approved the modified ethical rules proposed by the Central Ethical Committee.

J. C. C. CRAWFORD, *Hon. Secretary.*

360 Lisburn Road, Belfast.

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360 Lisburn Road, Belfast.

BRITISH MEDICAL ASSOCIATION—TYRONE DIVISION

A SPECIAL meeting of the Division was held in the County Hospital, Omagh, on Thursday, 25th January, 1934, at 4.30 p.m. It was open to non-members of the Association, for the consideration of a reply from the Ministry of Home Affairs, Northern Ireland, re the appointment of a bacteriologist for County Tyrone. Dr. McAllister was in the chair, and there were present—Drs. Leary, Eaton, Lyle, Clarke, Johnston, Chambers, Spence, Logan, Gillespie, McVicker, Elliott, Devlin, Murnaghan, Brown, Wilson, and Martin. After a lengthy discussion, in which all the members took part, the following resolution was proposed by Dr. Murnaghan, seconded by Dr. Eaton, and passed unanimously: "That we, the Tyrone Division of the B.M.A., strongly recommend to the Tyrone County Council the appointment of two bacteriologists, one for each end of the county. We recommend, too, as time is most important in the examination of specimens, that this necessity would be met by appointing one in Derry and one in Belfast."

Letters relating to the above were read, one from the Branch, one from the Ministry of Home Affairs, and one from the Clogher Rural District Council.

Dr. Ivan McCaw delivered a very interesting lecture on "The Commoner Skin Ailments." Dr. Murnaghan proposed a vote of thanks to Dr. McCaw, and this was seconded by Dr. Chambers, and passed. Dr. McCaw suitably replied.

A special meeting of the Division was held in the Tyrone County Hospital on Thursday, 15th February, 1934, at 4.30 p.m., to consider the new rules as to the Ethics of Medical Consultation and rules for medical inspectors. Dr. McAllister was in the chair, and after careful consideration of the rules as submitted by the secretary of the British Medical Association, the following amendments were made:

"We strongly disapprove of Rule 20, and we are in thorough agreement with the findings of Dr. Dane and Sir Chrisp. English."

The application of Rule 19 to a general practitioner is approved, and in the case of a practitioner in purely consultant practice, he should not see a case without the knowledge and consent of the attending practitioner, and if circumstances make this impracticable he is at liberty to do so, but he should notify the family doctor immediately.

J. R. MARTIN, *Hon. Secretary.*

Holmedene, Clogher, Co. Tyrone.

BRITISH MEDICAL ASSOCIATION—N.E. ULSTER DIVISION

THE Division met in Coleraine Cottage Hospital on Friday, 15th December, 1933. The chairman, Dr. Evans, presided over a fair attendance. The meeting was open for general discussion. Attention was drawn to the long delay in payment of sick benefit by some societies, which often caused hardship to insured patients. Members were asked to report any case of this kind, with a view to getting a list of definite cases and submitting the whole question to the Ministry of Labour.

At a meeting of the Division on Friday, 12th January, 1934, the main business was a paper by Dr. John Watson, Londonderry Mental Hospital, on "Mental Disease in Relation to General Practice." It is hoped to publish an account of this paper in our next issue.

At a meeting on Friday, 23rd February, 1934, a Kodak medical film on "Treatment of Chronic Leg Ulcers" was shown. The film showed the technique of treatment by elastic adhesive bandages, and in bad cases by skin grafting.

The Association's suggested rules regarding the ethics of medical consultation were discussed, and several alterations were suggested.

J. M. HUNTER, *Hon. Secretary.*

36 Eglinton Terrace, Portrush.

THE ULSTER MEDICAL SOCIETY

THE fifth meeting of the session was held in the Whitla Medical Institute, on 4th January, 1934. Professor W. J. Wilson, the president, occupied the chair. A resolution of sympathy with the relatives of the late Sir William Whitla was proposed and passed, the members standing. Dr. R. M. Beath then read a paper, "Developmental and Generalized Bone Diseases." This paper is published elsewhere in this number of the Journal.

The sixth meeting of the session was held on 18th January, 1934. Professor W. J. Wilson, the president, occupied the chair. The speaker was Dr. J. P. Martin, a graduate of this medical school, and now a member of the honorary staff of the National Hospital for Nerve Diseases, Queen Square, London. His subject was "Traumatic Intra-Cranial Hæmorrhage." It is hoped to publish this paper in a future number of this Journal.

The seventh meeting of the session was held on 5th February, 1934, with Professor W. J. Wilson, the president, in the chair. The lecturer for the evening was Dr. Jacques Forestier, Aix-le-Bain, France, who spoke on "Rheumatoid Arthritis and Its Treatment." This subject is one of unusual interest, and it is hoped to publish Dr. Forestier's paper in a special number of this Journal, devoted to Rheumatism, in October.

The eighth meeting of the session was held on 15th February, 1934. Professor W. J. Wilson occupied the chair. Dr. Scott-Pinchin and Dr. Morlock, both of London, spoke on "The Diagnosis and Treatment of Pulmonary Disease." They showed, by means of a beautiful collection of X-ray photographs, the results which they had obtained by the use of the bronchoscope.

The annual laboratory meeting was held on 1st March, 1934, in the Pathological Institute of Queen's University, Belfast. Numerous specimens were shown by surgeons and physicians, and there was a fair attendance of members. The president, Professor W. J. Wilson, presided.

J. A. SMYTH, *Hon. Secretary.*

23 University Square, Belfast.

LISBURN AND DISTRICT MEDICAL GUILD

THE annual meeting of the Guild was held in January at Dr. S. R. Hunter's residence, Dunmurry. In the absence of the president, Dr. Colquhoun, Dr. Hunter occupied the chair. It was proposed by the chairman, and seconded by Dr. Peatt, that Dr. White of Dunmurry act as president for the incoming year. This was passed unanimously. Dr. Peatt was re-elected secretary. After formal business, Dr. Ritchie of Belfast then gave his address on "Public Medical Service." Dr. Ritchie said that in London the P.M.S. attempts to take in both ante-natal and child welfare, and that the organization had been at work since 1926. Patients attend either their own doctor at his surgery or at their home, and received ordinary medicines. Payment is by a small subscription of threepence or sixpence per week. In some cases an entrance fee of two and sixpence is charged. In others the benefit is not allowed until after four weeks' payment have been made. These payments are made to a collector who calls at the house. The family income must be less than £5 per week. The London P.M.S. gives out leaflets of instruction in ante-natal and child welfare. In many centres in England an effort is being made to compel patients to attend for ante-natal examination. Dr. Ritchie considered that the P.M.S. is unlikely in Ireland, but he thought that a State Medical Service would probably develop.

The thanks of the meeting were voiced by Dr. J. G. Johnstone, seconded by Dr. W. S. Hunter, to Dr. Ritchie for his talk. This concluded the business.

J. W. PEATT, *Hon. Secretary.*

Railway Street, Lisburn.

LONDONDERRY MEDICAL SOCIETY

THE Londonderry Medical Society held the third meeting of the session 1933-4 on 21st December, at 8.30 p.m. The lecturer for the evening was Dr. B. R. Clarke of the Forster Green Hospital, Belfast, who addressed the meeting on "Recent Advances in the Diagnosis and Treatment of Pulmonary Phthisis." Perhaps the chief point was the great necessity for early diagnosis, to which end every resource should be available for the physician, because the earlier the diagnosis the greater the chance of recovery. Dr. Clarke was very careful to point out that the great principle underlying the treatment of every case was the enjoinder of the greatest possible rest, adequate nourishment, and the strictest attention to general hygienic principles, and in this way give every chance to allow the patient to overcome the disease naturally. All other treatments such as phrenic evulsion, artificial pneumothorax, thoracoplasty, etc., are to be correctly rated as subsidiary to the above, and only advisable to increase or augment the rest to the damaged lung obtainable by conservative measures. Dr. Clarke also gave a very beautiful demonstration of the 'sedimentation rate of erythrocytes test,' and spoke of it as the only reliable method of assessing progress in any given case.

The fourth meeting was supplementary and complementary to Dr. Clarke's lecture, and the address was given by Mr. G. R. B. Purce, who chose as his subject "Some Aspects of Chest Surgery." The most important part of his lecture dealt with surgical methods in relation to the treatment of pulmonary phthisis. Here again Mr. Purce was very careful to point out that these methods were in no way to be held as being in competition with either ordinary medical treatment, or with the aids and auxiliaries to that in the shape of artificial pneumothorax, etc. Mr. Purce in this connection described the operations of phrenic evulsion, thoracoplasty, and the methods used in severing adhesions which were preventing perhaps a full collapse of the affected lung in artificial pneumothorax cases.

In addition to the above, a résumé was given of the surgical work now being done in cases of bronchiectasis and in malignant diseases of the lungs. The lecture throughout was most profusely illustrated with lantern slides.

The fifth meeting was held in the City and County Infirmary at 8.30 p.m. on 22nd February. Dr. F. M. B. Allen was invited to address the Society, and lectured on "The Whys and Wherefores of Glucose Treatment in Children's Ailments." He condemned the misguided, and yet so universal and fashionable, use of glucose in every and any sick child, irrespective of whether such treatment was suitable or not. He showed good reason why such criticism was constructive, and just because of the good results obtained in conditions such as cyclical vomiting, and certain allergic states, that it would be a great pity that such a valuable remedy should suffer through failure to appreciate its uselessness and, in fact, its dangers in the catarrhal states. As an epilogue, Dr. Allen talked about the diagnosis and the treatment of pyelitis in infants. He showed very convincingly the traps into which the unwary practitioner may be led, and pointed out how easy it was to treat the condition if only a diagnosis is made.

J. A. L. JOHNSTON, *Hon. Secretary.*

19 Clarendon Street, Londonderry.