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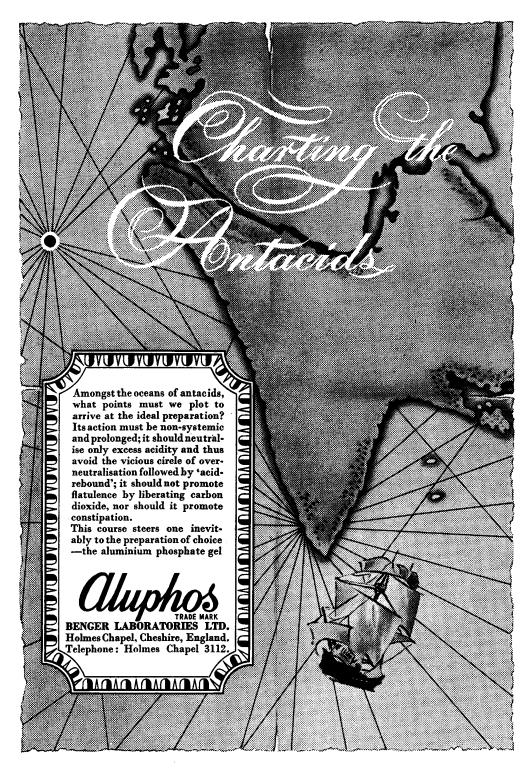
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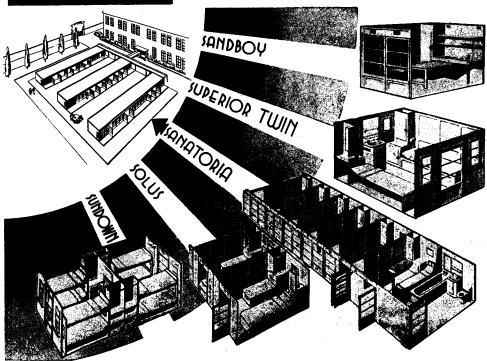
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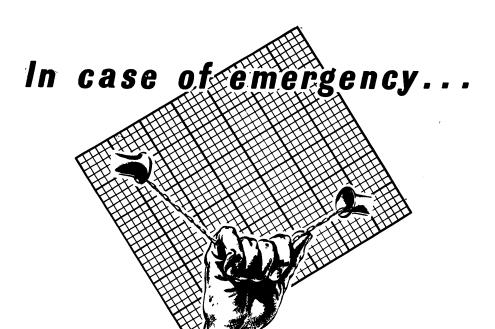
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# **CONTENTS**

| No. 1. May, 1951  | PAGE |
|---|------|
| A Medical Survey of the Irish Famine of 1846. LtGen. Sir William MacArthur, K.C.B., D.S.O., O.B.E., M.D., D.SC., F.R.C.P., F.R.C.P.1. | 1    |
| THE HAPPINESS OF THE CHILD. F. M. B. Allen, M.D., F.R.C.P.  | 16   |
| THE GENERAL PRACTITIONER IN ECLIPSE. J. B. Young, M.C., M.B   | 31   |
| THE VALUE AND LIMITATION OF ELECTROENCEPHALOGRAPHY. J. H. D. Millar, M.D., M.R.C.P.(LOND.)  | 41   |
| Hyperchloræmic Acidosis with Nephrocalcinosis. Joan B. T. Logan, M.D., M.R.C.P.   | 58   |
| EVALUATION OF THE TREATMENT OF DISSEMINATED SCLEROSIS WITH ARSENICALS. J. H. D. Millar, M.D., M.R.C.P.(LOND.) -                       | 62   |
| Post-Partum Eclampsia. William S. Campbell, B.Sc., M.B., B.CH., F.R.C.S. (ENG.), M.R.C.O.G., L.M.(ROTUNDA)                            | 66   |
| LEUCOTOMY: A FOLLOW-UP OF SIXTY CASES. C. B. Robinson, M.D., F.R.C.P.(I), D.P.M.  | 75   |
| RECENT REVERSES IN IMMUNISATION. W. J. McLeod, M.D., D.P.H.   | 78   |
| Congenital Atresia of the Esophagus. A. A. H. Gailey, M.D., D.C.H., M.R.C.P.E., and W. A. B. Campbell, M.D., D.C.H., M.R.C.P.E        | 81   |
| ROYAL MEDICAL BENEVOLENT SOCIETY OF IRELAND   | 86   |
| List of Additions to the Medical Section of the Library of the Queen's University of Belfast, October, 1950-February, 1951            | 87   |
| Reviews 30, 57, 61, 65, 9   |      |
|   | .,   |
| No. 2. November, 1951   |      |
| GILBERT AND SULLIVAN. J. G. Johnston, M.C., M.D., J.P   | 93   |
| A SHORT HISTORY OF OTOLARYNGOLOGY. Kennedy Hunter, M.B., F.R.C.S. (EDIN.), D.L.O.(ENG.)   | 106  |
| A HISTORY OF THE QUEEN'S UNIVERSITY OF BELFAST DENTAL SCHOOL. P. J. Stoy, B.D.S., F.D.S.R.C.S.  | 118  |
| THE PATTERN OF DISEASE. F. M. B. Allen, M.D., F.R.C.P.  | 131  |
| Dental Disease and Civilisation. P. J. Stoy, B.D.S., F.D.S.R.C.S.   | 144  |
| THE CONTROL OF DISEASES OF TISSUE REACTIVITY BY CORTISONE. Wm. Townsley, M.D.   | 159  |
| Doctor and Patient in the Age of Hippocrates. E. D. Phillips, M.A.  | 172  |
| RECENT TRENDS IN MATERNAL MORTALITY IN NORTHERN IRELAND. E. A. Cheeseman, B.SC. (ECON.), PH.D. (MED.)                                 | 181  |
| THE LACRIMAL CHANNELS IN CYCLOPIA. W. R. M. Morton  | 189  |
| List of Additions to the Medical Section of the Library of the Queen's University of Belfast, March-September, 1951                   | 195  |
| REVIEWS 105, 130, 143, 158, 171, 180  |      |

# **CONTENTS**

|  | PAGE   |
|--|--|
| A Medical Survey of the Irish Famine of MacArthur, K.C.B., D.S.O., O.B.E., M.D.,   |  |
| The Happiness of the Child. F. M. B. A   | llen, M.D., F.R.C.P 16   |
| The General Practitioner in Eclipse. $J$ .   | B. Young, M.C., M.B 31   |
| THE VALUE AND LIMITATION OF ELECTRO Millar, M.D., M.R.C.P.(LOND.)  | DENCEPHALOGRAPHY. J. H. D.   |
| Hyperchloræmic Acidosis with Nephroc M.D., M.R.C.P   | CALCINOSIS. Joan B. T. Logan,  |
| Evaluation of the Treatment of Dis<br>Arsenicals. J. H. D. Millar, m.d.,   |  |
| Post-Partum Eclampsia. William S. Camp<br>(Eng.), M.R.C.O.G., L.M.(ROTUNDA) -  | obell, B.SC., M.B., B.CH., F.R.C.S.  |
| LEUCOTOMY: A FOLLOW-UP OF SIXTY C. F.R.C.P.(I), D.P.M  | ASES. C. B. Robinson, M.D.,  |
| RECENT REVERSES IN IMMUNISATION. W.  | J. McLeod, м.D., D.Р.Н 78  |
| CONGENITAL ATRESIA OF THE ŒSOPHAGUS. M.R.C.P.E., and W. A. B. Campbell, M.   |  |
| ROYAL MEDICAL BENEVOLENT SOCIETY OF I  | RELAND 86  |
| List of Additions to the Medical Sect<br>Queen's University of Belfast, (  |  |
| Reviews  | - 30, 57, 61, 65, 91, 92   |
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# THE ULSTER MEDICAL JOURNAL

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#### DATES OF PUBLICATION

Under present difficult circumstances it is hoped to issue two numbers each year: on 1st May and on 1st November.

It is felt that the incidence of the condition is greater than suspected and that, since even nine cases out of 15,900 live births is believed to be an under estimate, this would suggest that at least fifteen cases should be expected yearly in Northern Ireland out of a birth rate of 30,000. The condition ranks as a surgical emergency and, since the chances of saving life are good when operation can be performed in the first twenty-four hours after birth the importance of early diagnosis is stressed.

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#### ROYAL MEDICAL BENEVOLENT SOCIETY OF IRELAND

DR. Harrington has sent us the following report for inclusion in the Journal. Fellows and Members of the Society will agree that the medical community as a whole are indebted to Dr. Harrington for the outstanding success of his campaign to increase the membership of the Society. The cases that come before the Fund's committee for consideration are not numerous, but each is pitiful, and, were the circumstances widely known, would arouse instant sympathy. In recent years the local funds have risen gradually above the point where we got more out of the Irish fund as a whole than we put into it, but the present position, for which once more, we must congratulate Dr. Harrington, is at last becoming more in keeping with the size and means of this city and medical school. With the ever decreasing value of the pound it is important that this should be maintained, and we would take this opportunity of bringing the Royal Medical Benevolent Fund Society of Ireland to the attention of those who are still not subscribers.

The Annual Meeting of the Belfast Branch of the Royal Medical Benevolent Fund Society of Ireland was held in the Whitla Medical Institute on the 6th April, 1951, with Dr. Robert Marshall in the chair, and a full attendance of members.

The Honorary Secretaries were happy to report a big increase in membership—the total being doubled within two years.

In 1949, there were 182 members; in 1950, 213; and in 1951, the total, including four new Life members, reached 369.

The gain has been consolidated by the unanimous support of old subscribers. On the financial side the funds for the year have passed the £500 mark.

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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? A proposal form is appended: your proposer and seconder must be Fellows 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.

We remain.

Yours faithfully,

SAMUEL BARRON, President.
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|   | <u>.</u> 191  |
|---|---|
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# **CONTENTS**

|   | PAGE  |
|---|---|
| A Medical Survey of the Irish Famine of 1846.<br>MacArthur, K.C.B., D.S.O., O.B.E., M.D., D.SC., F. |   |
| The Happiness of the Child. F. M. B. Allen, M.D.  | ., F.R.C.P 16   |
| THE GENERAL PRACTITIONER IN ECLIPSE. J. B. Young  | g, M.C., M.B 31   |
| THE VALUE AND LIMITATION OF ELECTROENCEPHA Millar, M.D., M.R.C.P. (LOND.)                           | LOGRAPHY. J. H. D.  |
| Hyperchloræmic Acidosis with Nephrocalcinos m.d., m.r.c.p   | Is. Joan B. T. Logan, 58  |
| EVALUATION OF THE TREATMENT OF DISSEMINAT ARSENICALS. J. H. D. Millar, M.D., M.R.C.P.(              |   |
| Post-Partum Eclampsia. William S. Campbell, B.Sc (ENG.), M.R.C.O.G., L.M.(ROTUNDA)                  | C., M.B., B.CH., F.R.C.S.   |
| LEUCOTOMY: A FOLLOW-UP OF SIXTY CASES. C. F.R.C.P.(1), D.P.M  | B. Robinson, M.D., 75   |
| RECENT REVERSES IN IMMUNISATION. W. J. McLe   | od, M.D., D.P.H 78  |
| CONGENITAL ATRESIA OF THE ŒSOPHAGUS. A. A. H. M.R.C.P.E., and W. A. B. Campbell, M.D., D.C.F.       |   |
| ROYAL MEDICAL BENEVOLENT SOCIETY OF IRELAND   | 86  |
| List of Additions to the Medical Section of Queen's University of Belfast, October,                 |   |
| Reviews   | 30, 57, 61, 65, 91, 92  |
| Editorial Board   | Hon. Treasurer  |
| Prof. W. W. D. Thomson, B.A., B.SC., M.D.,<br>F.R.C.P.(LOND.), D.P.H.                               | . C. C. CRAWFORD, M.D.  loint Editors                             |
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# THE ULSTER MEDICAL JOURNAL

PUBLISHED ON BEHALF OF THE ULSTER MEDICAL SOCIETY

Vol. XX

1st MAY, 1951

No. 1

# A Medical Survey of the Irish Famine of 1846

A Robert Campbell Memorial Oration

By LT.-GEN. SIR WILLIAM MACARTHUR, K.C.B., D.S.O., O.B.E., M.D., D.SC., F.R.C.P.I.

Many years ago as a house surgeon in the Royal Victoria Hospital, it sometimes fell to my duty to call on Mr. Robert Campbell in the middle of the night to deal with some surgical emergency in the wards. And when on these occasions I would hang on every word of his oracular pronouncements—and unlike some other oracles I have encountered, I never knew Mr. Campbell to be wrong—I did not dream then that my name would ever in any way be linked with his.

He was an extraordinary man, with long continued silences, so that he would go through all the stages of a complicated operation without uttering a word; then, at too infrequent intervals, his outbursts of eloquence, when he would pour forth a store of wisdom and knowledge to the profit and delight of all fortunate enough to hear him. Sometimes he would go on at such length as to disorganize the whole work of the Extern Department, and the Sister in desperation would rattle dishes and turn on taps to warn "Robert" that it was time he went off. I remember one such day when the Sister dispatched a nurse to find out if these demonstrations had had any effect. The scout peeped cautiously round the door, but Mr. Campbell—who never missed anything—saw her and looking up with a little twinkle in his eye, he said, "Robert's still here!" And, Ladies and Gentlemen, I think that, both in spirit and in influence, "Robert' is still here to-night.

I assure you I will always treasure this Medal; first, because of its association with a man whom, when I knew him, I revered on this side idolatry; and second, because the award has been made by fellow-members of my own Medical School.

It is assumed not uncommonly that the London Plague of 1665 was an isolated event, and so it is often referred to as "The Plague of London," whereas we

know that it was merely the last of a long series of epidemics of plague which had flared up with wearying monotony for centuries past, each of these standing out in the popular memory until in turn it was replaced by another. In the same way the Irish famine of a century ago has become "The Famine," although relative to the population it was no worse than others that had gone before. These dreadful visitations were not peculiar to Ireland, but were once the common lot of Europe, and so universal and calamitous could they be that we find an English chronicler crying out that the hand of God was lifted against the people of Christendom.

The basic cause of all these famines wherever they occurred was the low standard of economic life of the people, most of whom at the best lived only one season ahead of starvation. Any extensive failure of the crops resulted in famine, local or widespread. In England, the standard of life rose steadily over the centuries; owing to the introduction of new root crops, cattle could be fed throughout the winter instead of being killed off in large numbers as before, and although in lean years there might still be scarcity with distress and fever in one part or another, the generalized famines and generalized famine fevers in England came to an end with the fourteenth century.

In Ireland, however, there was no parallel improvement. Here wars of conquest, internal strife, confiscations and penal enactments combined to depress the economic standard of the Irish peasant. The potato had proved a cheap and plentiful source of food, and any young man who could obtain a scrap of ground and build some kind of a hovel might marry and raise a family, and so overpopulation was added to the existing evils. As there was not enough land to go round, holdings were divided and subdivided; middlemen rented land from the owners, and instead of cultivating it themselves let it out in plots at inflated rents. In the end nearly three million of the population came to be dependent on the potato for their existence. This number was made up of three main classes: (1) Peasant farmers renting an acre or two of ground; (2) Labourers who lived in cabins on the farm where they worked, cultivating one or more roods of land for themselves; (3) Men without any fixed employment, working when they could at a wage of fourpence to tenpence a day, and hiring a scrap of land to serve as a potato garden. People existing in these deplorable conditions could not lay by reserves to carry them over bad times, and when these arrived, as sooner or later they did, the whole crazy system collapsed in chaos.

Food shortages in Ireland were commonly brought about by prolonged wet and cold; one bad season caused distress, two in succession meant famine. The famine which concerns us, however, had a different origin, a fungus disease of the potato due to *Phytophthora infestans* which appears in the form of black spots on the leaves, and on the under surface a whitish mould containing the spores. These fall to the ground and destroy the tubers, and are conveyed to other plants by wind, rain and insects. The fungus is checked by dry weather, and

flourishes in profusion when the weather is warm and damp. The blight showed itself in 1845, but was limited in its distribution so that there was severe want in some localities, and abundant food in others. The summer of 1846 was damp and of "unprecedented heat;" the blight broke out once more, this time with the greatest virulence, and swept over the country. One day the fields were covered with a luxuriant growth; a few days later the leaves and stalks were black and dead, and the food of a whole countryside had vanished. The price of potatoes rose from two shillings a hundredweight to seven shillings and later to twelve shillings when obtainable at all. The people lacked the means to buy grain in substitution. Some farmers of the more comfortable class sold out their stock of animals and fowls, fearing that these would be raided by starving mobs, a short-sighted policy that recoiled on their own heads, and swelled the rising tide of misery. By the winter of 1846-47 the food position was desperate, and widespread famine reigned.

Every famine in these islands provoked an epidemic of fever with the regularity of clockwork. The famine fever was made up of two elements in varying degrees of admixture, typhus and relapsing fever. conveyed by the common louse of man, though this was not proved until sixty years after the Famine. Typhus was familiar to every doctor in Ireland-"the disease natural to our climate," they called it, for it was endemic in depressed and congested parts of the country and in the slums of towns, though varying greatly in extent and severity from year to year. Foci of relapsing fever must have existed also, for the disease always appeared in famine, but much less is heard of it in normal times, and some doctors did not recognize the disease when eventually it reached their districts, and reported that it "presented symptoms of a new and extraordinary nature." Relapsing fever was described as propagated more rapidly than typhus, and as running through whole families in a few days. I suggest that the more rapid spread was probably due to the fact that blood from a relapsing-fever patient can convey infection if it comes into contact with the skin or the eye. Profuse nose-bleeding was a common feature of an attack, and in addition, extensive hæmorrhages occurred from other parts due to associated scurvy. In the crowded cabins contamination with such blood could not well have been escaped.

Outbreaks of relapsing fever, under the name "intermittent fever," were recorded in Dublin in the early 1700's, and described as occurring alongside spotted fever, the name "typhus" not having then come into use. The disease was also clearly described by Rutty, the Quaker physician of Dublin, in the famine of 1740. In spite of this, as late as 1846, a small minority of doctors maintained that typhus and relapsing fever are no more than clinical variants of the same infection, pointing out that in two patients removed from the same house and even from the same bed, the attack might follow a relapsing fever course in the one, and a typhus course in the other; and that in the same individual an attack might begin with

symptoms of relapsing fever, and later assume those of typhus, or vice versa. We know to-day that the latter vagaries resulted from double infections.

A natural consequence of the spread and intensification of the famine was a steady deterioration in the sanitary conditions of the hunger-stricken. People striving to keep body and soul together by what they could find of dock leaves, nettles and the like, with perhaps an odd handful of raw meal, or going altogether foodless for days on end, would not concern themselves much about personal cleanliness, even had they strength enough to carry water from the well, or any fuel to heat it. Their clothing of any market value had been sold long since to passing pedlars; they piled on whatever rags they had left, and wore these night and day. huddling together for warmth in the colder weather. The neighbours crowded into any cabin where a fire was burning, or where there was food of sorts to be shared or bartered. In these ideal conditions lice multipled and spread, and even those who ordinarily were cleanly in their persons became infested. Thus there was prepared a rich soil ready and waiting for the seeds of fever which sooner or later were sure to fall.

A slow and gradual extension of fever was noticeable before the storm broke. At the beginning of December, 1844, the fever cases in the workhouse hospitals numbered 362; at the same date of 1846, the figure had increased fivefold. Over the same period the number of workhouses containing fever patients rose from thirty-two to seventy-one. A similar portentous increase in fever was reported from various dispensary districts.

As the famine strengthened its hold, crowds of the starving deserted their homes and took to the roads, in the hope of escaping certain death from hunger. To the voluntary migrants were added others evicted for rent default, "turned adrift to find a living where no living was to be found." Some of the wayfarers were incubating fever and developed a frank attack on their journey. Others, even a greater menace, had the disease in too mild a form to be prostrated by it—in the words of a doctor of the day, "I saw many pass through the fever while they were literally walking about." Convalescents carried infection with them in one way or another. The miserable lodging-houses frequented by the class of strolling beggars became centres of dissemination. In one such on the Sligo Mail-Coach Road, there were sixty deaths from fever in one period of three months; the original proprietor and his successor both died of typhus.

In spite of the pickets of able-bodied men posted on the roads (by authority of 59, Geo. 3, cap. 41), these migrations were the most active means by which disease was spread abroad, and one example of the after-results may be given. The district of Trim in Co. Meath was accounted amongst the wealthiest in Ireland. The inhabitants were comfortably circumstanced and engaged mainly in breeding animal stock. Hence the failure of the potato crop, moreover not complete in the locality, did not result in starvation. In spite of these special advantages, disease became very prevalent. A doctor of the place described how the fugitives flocking

in from the west carried the fever in their own persons, and mixing with the people attending markets and fairs, imparted the disease to them. "I often observed whole families belonging to distant counties lying in fever on the roadside."

Wherever the fugitives went they left a trail of disease behind them, so that the ominous term "road fever" passed once more into current use. In the conditions then prevailing, the introduction even of a single case of fever was like tossing a lighted match into a powder magazine. This is exemplified by the history of Ballinrobe workhouse, Co. Mayo. This institution, unlike so many others, had escaped fever until the end of February, 1847, when a strolling beggar was admitted, and a few days later died of typhus. The disease swept through the workhouse, crowded far beyond its capacity with men, women and children, huddled together in the same compartment, living and sleeping in their clothes, for they had neither bedding to lie on nor a blanket to cover them. Large numbers died. The physician, the chaplain, the master, the matron and the clerk of the Union, went down with typhus simultaneously, and only two of these survived.

The epidemic developed and spread in this irregular and sporadic fashion until nearly the whole country was engulfed. In Co. Kilkenny fever was rampant as early as the summer of 1845. In Cork, it broke out early in 1846 and reached its height in the following year. "During the first six months of that dark period," wrote Dr. Callanan of Cork city, "one-third of the daily population of our streets consisted of shadows and spectres, the impersonations of disease and famine, crowding in from the rural districts, and stalking along to the general doomthe grave—which appeared to await them at the distance of a few steps or a few short hours." In Dublin, the epidemic was described as beginning with the year 1847, or a few weeks earlier. Fever in Belfast began to spread in September, 1846. Tyrone was attacked in December, 1846, and Derry in the spring of 1847. In Co. Down, fever appeared generally in the early spring of 1847, but the Hillsborough district escaped fever until the middle of June, after which date "it prevailed greatly"; in this month a "great prevalence of disease" was reported in Donaghadee and Newtownards. It is interesting that in Wexford, fever broke out in two distressed areas in April, 1847, but in the rest of this county where there was little actual starvation, the disease did not appear generally for more than another year. The remote northern parts of Co. Leitrim escaped until the end of 1847, and the western islands of Inisbofin and Inishark until the middle of 1848, by which time the epidemic was virtually over in some parts of the country.

The only extensive area to escape famine fever altogether was that part of Co. Down centred on Rostrevor and Warrenpoint. Local medical opinion ascribed this peculiar immunity to the fact that want was seldom or never known there, because of the amount of employment provided by the numerous and wealthy resident gentry. Other parts of Ireland, however, equally favoured in this way suffered heavily from imported fever. The most likely explanation is that the district was not traversed by any of the direct routes to the larger towns which were the goal

of the hosts of starving migrants. It is significant in this connection that the same district also escaped the famine fever of 1816.

Dysentery of the bacillary variety has always been a concomitant of famine, whether this was brought about by natural causes or by war. The infection is predisposed to by the unhealthy state of the intestine and consequent diarrhœa brought about by weakness and unsuitable food. The disease is of long standing in Ireland. Under the name Ruith fola—bloody flux—it is recorded as widely epidemic in A.D. 763; a king of Connaught died of it in 767. Gerard Boate, writing during the Cromwellian wars, says that "The looseness" is so general in Ireland that the English settlers have given it the name of "The country-disease," a term used by Cromwell in his despatches from Ireland. Boate gives the warning that if those attacked by the looseness do not check it, "they do commonly after some days get the bleeding with it, . . . and at last it useth to turn to the Bloody flux."

In those parts of the country hardest hit by famine, dysentery broke out long before the fever had begun to spread. Elsewhere, dysentery might precede the fever, accompany, or follow it. Belfast escaped lightly compared with other places, and only some 1,800 cases of dysentery were admitted to the hospitals there. The type was severe, however, with a mortality of 32 per cent.

Two non-infectious disorders became prevalent during the famine, scurvy and famine dropsy. It is an eloquent testimony to the anti-scorbutic properties of the potato that although millions of peasants had lived for years exclusively on potatoes, scurvy was unknown amongst them until the famine, and so unfamiliar were many doctors with the disease, that its symptoms were often regarded as a complication or sequel of relapsing fever. One instructive point was recorded from Waterford city. Here the comfortable artisan class adopted a famine diet of bread, tea, and porridge made of Indian meal. They and their families suffered severely from scurvy, whereas the destitute of the place escaped the disease entirely. In the words of the recorder, "I can really attribute this to nothing else but the soup given them by the Quakers, which was well seasoned with vegetables."

Wherever scurvy and famine dropsy were both in evidence, the dropsy was a later development as representing a more severe grade of deprivation. The swelling showed itself first in the feet and ankles, spreading upwards until in the end the sufferer was water-logged. When the people of the parish of Schull were dying at the rate of fifty a day, the Rev. Dr. Traill\* wrote that in the frightful and fearful havoc around him, the aged and the young "are almost without exception swollen and ripening for the grave." This devoted rector who laboured unsparingly for these unhappy creatures, himself died of typhus.

In this famine, just as in that of 1816, it was noticed that the incidence of fever and its mortality were greatly influenced by the social standing of the sufferers. Many of the gentry and of the upper class generally, contracted typhus, but

<sup>\*</sup>In contemporary reports, this rector's name appears both as "Traill" and "Traill Hall."

relapsing fever was almost unheard of amongst them, even when this was the form of disease to which they had been most exposed. Also the proportion of fatalities from fever was much in excess of that usual amongst the peasantry, differing by as much as 16 to 1.

A third difference was that when one of a better-class family contracted typhus, the disease did not spread to other members of the household even when no form of isolation was adopted. These discrepancies were reported independently by doctors all over the country, and their occurrence cannot be gainsaid. It would be interesting to discuss the reasons for them, and I suggest the following explanations. In the louse the organisms of typhus—Rickettsia—invade the cells lining the intestine of the insect. The invaded cells, packed Rickettsia, swell out like balloons, and eventually burst. The organisms thus set free pass out with the intestinal contents. This fæcal matter dries to a light dust, which is easily diffused and blown about and in this the contained Rickettsia can remain alive for long periods. Consequently, it is possible for persons to become infected by this fæcal dust-through the skin, the eye, or by inhalation—without having lice on the body. This method of infection does not occur in relapsing fever, for here the spirochætes causing the disease remain within the body of the insect, and are set free only when its delicate structure is sufficiently damaged—as in scratching—to allow its body fluid to exude on to the skin. Although the role of the louse as a vehicle of infection was unknown, the great increase of vermin among the sick and the starving was common knowledge, and the efforts made by the better class to avoid picking up these parasites would at the same time save them from louse-borne relapsing fever, but not from typhus conveyed by fæcal dust. Persons contracting typhus in this way, being themselves free from lice, could not pass on the disease to others.

The higher proportion of fatalities from fever among the better classes was partly due to their liability to contract the more deadly typhus rather than relapsing fever. This, however, is not the full explanation, for their case-mortality rate from typhus was also higher than that from typhus amongst the poor-60-70 per cent. being given as the former, and 20-25 per cent. as the latter. Here age would have a great influence. Typhus is much more deadly to the middle-aged and elderly than to the young, because of the great strain which the disease throws on the heart. Most of the victims among the better classes—clergy, doctors, magistrates and others set in authority—would fall into the group unfavourably placed as regards age, and so were handicapped from the start; whereas amongst the peasantry where the whole family was liable to attack, the milder infections usual in children and adolescents would keep the average mortality at a lower figure. In addition to this, however, it has long been observed that in localities where typhus is endemic, those native to the place on the whole suffer less severely from the disease than incomers who have not had the same close personal association with it in the past. The probability is that many of the former have

acquired some degree of immunity either from mild and unrecognized attacks in childhood, or by a process of vaccination with small and sub-infective doses of virus over a long period, such immunity being insufficient to protect against a later heavy infection, but enough to lessen the virulence of the attack.

With a view to meeting the apprehended danger of fever in Ireland, the Temporary Fever Act, 9 Vic., cap. 6, was passed by Parliament on 24th March, 1846. This empowered the Lord Lieutenant to appoint unpaid Commissioners of Health, up to five in number, to constitute a Central Board of Health; and to appoint additional medical officers for famine duties, these to be paid by the Treasury. Any two Commissioners, in writing, could require a Board of Guardians to set up, equip and staff a temporary fever hospital, and also make arrangements for a dispensary. In accordance with the Act, the Commissioners appointed were: Sir Randolph Routh (in charge of the commissariat operations in Ireland), Sir Robert Kane (originally a medical professor of chemistry, and later President, Queen's College, Cork), Edward Twistleton, Esq., (Chief Commissioner of the Poor Laws), Sir Philip Crampton (President Royal College of Surgeons), and Dominick John Corrigan, Esq., M.D. (best remembered to-day by "Corrigan's pulse").

The summer of 1846 passed without producing, in the opinion of the Board, much cause for alarm. Reports from the country—soon to be falsified—foretold luxuriant crops. Only seventeen requests for the Board's assistance had been received throughout. The Act, therefore, was allowed to expire on 31st August, 1846, a tragic error of judgement. The Board ended its sittings, and the additional medical officers were discharged. It was ruled that where it was necessary to keep on a temporary fever hospital, this could be done under the Poor Law Amendent Act, 6 and 7 Vic., cap. 92.

In a report dated 5th December, 1846, the Board gave an appreciation of the situation, based mainly on an analysis of the incidence of fever in Dublin in twelve foregoing years. The increase in 1846 they thought to be no more than one of the seasonal fluctuations which often occurred, and the then rarity of severe cases of typhus was taken as a good omen. Their summing up was that the existing scarcity of food must excite an apprehension that fever might spread extensively, but that appearances rather suggested that this development might not take place.

If the Commissioners had looked further afield than Dublin, and taken into consideration only one report which they had received, they would have had some warning of the magnitude of the catastrophe that lay ahead. This was from the clerk of Skibbereen Union, who pointed out the great increase in the number of inmates in the workhouse on 21st November, 1846, as compared with the number on the same day a year before. The workhouse had accommodation for 500 inmates. The total number in the workhouse had risen from 240 to 889; in the infirmary, from 40 to 729; in the fever hospital, equipped for 40 beds, from none to 140. That is to say, of nearly 900 inmates occupying space intended for 500, there were

only 20 who were not sick in hospital. Deaths in the month had risen from 1 to 67.

As the season advanced, reports controverted all belief that famine and disease might not spread beyond a few unhappy parishes, and in view of the alarming developments the Lord Lieutenant reappointed the Board of Health in February, 1847, and thereafter it remained in being until the end of the parliamentary session of 1850.

The hospital arrangements in Ireland were ill adapted to meet a great epidemic. In addition to the voluntary hospitals in some of the larger towns, the provisions for the sick poor were: (1) Workhouse infirmaries; (2) Workhouse fever hospitals; (3) County infirmaries; (4) District fever hospitals; (5) Dispensaries. Of these, the County infirmaries had no accommodation for fever patients, and the treatment of these in workhouse infirmaries was disapproved of by the Poor Law Commissioners as dangerous to other patients.

The Irish workhouse system was established in 1838 by the Act 1 and 2 Vic., cap. 56. The task was long and laborious. The country was first divided into 130 Unions, and these sub-divided into 2,049 electoral areas; extensive surveys and valuations had to be carried out, and a workhouse built in each of the Unions. By the end of 1844, 113 workhouses were open; a year later, 122 and most of the remaining eight opened in 1846. Each workhouse included an infirmary, with a fever hospital in a separate building, unless provision for fever patients already existed in the neighbourhood.

When the Temporary Fever Act was before Parliament in February, 1846, the Chief Secretary for Ireland declared that as regards the danger of approaching fever in Ireland, the Poor Law Commissioners had made the most ample arrangements. In fact, the hospital accommodation was totally insufficient, and was unequally distributed. The District fever hospitals were small and poorly equipped, and their funds inadequate for expansion. Large areas of the country were unprovided for. The dispensaries could not cope with thousands of fever patients in wretched cabins scattered over miles of country. The hospitals soon became packed with sick, often more than one patient to a bed, and others lying on the floor in between. Armagh fever hospital for instance, with accommodation for 100 patients, held 255. The workhouses were crowded far beyond their capacity with miserable wretches admitted by the authorities against their better judgement, for the only alternative was to leave them to starve to death outside. In such places no effective isolation was possible—hospital wards, convalescent wards, living space, schoolrooms, and even stables were overflowing with sick. Admission to a workhouse in these conditions too often meant for the destitute only the exchange of death from hunger for death from disease. Thus, in Lurgan workhouse ninety-five inmates died in one week, and of these, fifty-two had been free from any disease when they were admitted. The ravages of fever amongst the staff of hospitals, medical and lay, added to the existing horrors. During the worst period of the famine, forty-eight

medical men died in Munster (nearly all from typhus); in Galway, eleven; and in Cavan, seven. Of the 473 medical officers appointed by the Board of Health to special fever duty, one in every thirteen died at his post.

While the slow moving Government measures were taking effect, unofficial relief committees and private persons brought help to the sick. Religious bodies were very active in these works of mercy. The Quakers, with their customary generosity, shipped cargoes of supplies to doctors in remote parts. In one place, within a month the Roman Catholic clergy nursed 268 sick for whom there was no room in hospital. In another, the Society of St. Vincent de Paul relieved about fifty families every week, each with an average of six or seven members down with fever. The needs of these simple folk were not great, and a good bed of clean straw laid on their mud floors they thought the height of luxury.

The Temporary Fever Act proved in practice to have many defects which had not been foreseen. In particular, there was the difficulty in coercing recalcitrant Boards of Guardians, especially when the Law Courts were not in session, and the Board had no funds to make good the defection of local authorities. So in the following year another Fever Act (10 Vic., cap. 7), was passed. This made provision for a Finance Committee and under this a Relief Committee for each of the 2,000 -odd electoral areas, or for two or more in conjunction if thought advisable. The committees were provided with funds for relief of the sick. They could also enforce and pay for the cleansing of houses, persons and clothing—measures which at the time were described as having saved "innumerable" lives. They could also deal with one crying scandal, for they were authorized to arrange and pay for what the Act called "the proper and decent interment" of the dead.

Acting chiefly on reports from its medical inspectors, the Board of Health directed existing hospitals to increase their accommodation by taking over houses, stores and other buildings including even military quarters, and by erecting hospital sheds to prescribed official plans. Such a shed, for example, to hold one hundred patients cost £250. Tents were freely used too, especially at the height of the emergency, and the military hospital tents each providing fourteen beds proved particularly satisfactory. Accommodation for the temporary fever hospitals was provided in the same way. In all, the Board of Health granted requisitions for 373 of these institutions; some were large, containing upwards of 900 beds. Regular returns from the temporary fever hospitals were furnished only from July, 1847—by which time in some parts the epidemic was declining—and from this date until August, 1850, when the Fever Acts expired, the recorded admissions numbered 579,721. It must be remembered, however, that large numbers of the sick—in many districts the great majority—did not enter hospital, and their numbers, therefore, are unknown.

One of the most complete of the reports was that furnished by Dr. Seaton Reid for Belfast. He describes the epidemic as having lasted for precisely two years, September, 1846, to September, 1848, and it broke out, he says, while food in the

town was still abundant and unusually cheap, and work plentiful, conditions which lasted until the early part of 1847. Thus it was the old story once more, fever conveyed by fugitives from elsewhere. These incomers to Belfast numbered about 10,000, a figure to be added to the then 100,000 inhabitants of the Belfast Union district. At the height of the epidemic, three hospitals were in operation with, in addition, tents sufficient for 700 convalescents.

The largest number of hospital patients on any one day (17th July, 1847) was 2,118, and soon after this date the Board was informed that the three Belfast graveyards were "choaked up." The admissions for fever numbered 13,469, to which should be added several thousand more to allow for those treated in their own homes. Included in the fever total are many cases of typhoid, a disease which also became epidemic in Belfast. In Dr. Reid's own hospital over 900 cases were given this diagnosis, about one-fifth of the "fever" group admitted there. His story makes curious reading. He describes the tongue as dry and brown, and the brain as "a good deal involved;" the mortality was 10 per cent. This would suggest typhoid of an ordinarily severe grade, but it is puzzling to learn that the duration of attack averaged only fifteen to twenty days. This short duration would be in keeping with paratyphoid fever, but the other characters he mentions are strongly against this possibility. Dr. Reid shows himself to have been a stern precisian, and the diagnostic criteria that he lays down for typhus and relapsing fever are so severe and exclusive that many genuine cases of both diseases must have been shut out from their rightful place; the addition of some proportion of these shorter fevers-e.g., typhus with little or no rash, and relapsing fever without a relapseto the group labelled "typhoid" would reduce the number of days which the attack averaged.

Dr. Reid contrasts the symptoms of typhus and relapsing fever. He stresses the sudden onset of typhus, and says that almost invariably a patient would declare himself in a state of perfect health one day, and on the next be seized with rigor, headache and pain in the back. The onset of relapsing fever is even more sudden, and the rigor very severe. There is headache, but not the dizziness and ringing in the ears, nor the suffused and injected eyes of typhus. The pulse is quicker than in typhus. The typhus rash comes out about the fifth day, being seen first on the front of the shoulder joints, and on the epigastrium; at first the spots are capable of being temporarily removed by pressure, but become less so as the disease advances. The rash generally remains out for the course of the attack, but may last only for thirty-six or forty-eight hours; none the less, it is just as characteristic as that of scarlatina or of measles. The duration of attack is commonly fourteen days from the rigor, and the crisis is accompanied either by increased secretion of urine, or by gentle perspiration; if the latter is severe "it is in general a fatal symptom." In relapsing fever from the beginning there is great irritability of the stomach and everything is vomited. Muscle pains are frequent and severe enough to simulate acute rheumatism. These violent symptoms continue usually until the fifth day when, often with a preliminary rigor, the patient falls into a profuse

sweat, saturating the bedclothes. Immediately after this crisis he declares himself to be well, is clamorous for food and for permission to leave his bed. In none of Dr. Reid's cases did the crisis appear before the third day or later than the ninth The extremes of the duration of the succeeding relapse were one day, and nine days. He is emphatic that he never saw more than one relapse in any case, contrary to the experience elsewhere; nor did he see a second attack of typhus nor a second attack of relapsing fever in the same person. He cites the case of a patient who had at intervals typhus, relapsing fever, and typhoid.

Jaundice as a complication of relapsing fever occurred frequently enough to give rise to the name "yellow fever," which is still sometimes to be heard in fireside stories of the famine time. "Yellow fever" was not a popular name only, but was employed by many medical writers including Stokes and Graves. The title which the latter used for the section on relapsing fever in his textbook is, "Yellow Fever of the British Islands."

A complication of typhus recorded from many parts was erysipelas. Dr. Reid describes how a patient during convalescence would be seized with a rigor and a return of fever, followed in a few days by redness, pain and swelling about one of his ears, thence extending over the scalp and face, through the nostrils into the throat, and then into the larynx "producing there one of the most fatal complications that can occur."

A strange state of affairs unusual in hospital administration developed in Hillsborough. The town was served by a temporary fever hospital established at Culcavy, but for some reason which does not appear, a section of the population incited by the Archdeacon of Down, the Rev. Robert Moorhead, objected to its use. They set up an opposition hospital in several houses in Ballynahinch Street, "nominating themselves Governors," and persons "labouring in Fever" were conveyed there in carts. The local Relief Committee took a stern view of these proceedings and ordered the removal of the patients, by force if necessary, to such fit and proper place as their medical officer, Dr. Croker, should point out. The Archdeacon's party then attempted to bring fever patients into the town by force, and for a time a serious riot seemed likely to break out, but the presence of two magistrates—Colonel Hawkshaw and Hill Wilson Rowan, Esq.—supported by a body of police overawed the crowd, and after denouncing the authorities, the Archdeacon advised his followers to withdraw peaceably, which they did, taking their fever patients with them.

As is well known, the great weight of the visitation fell on the counties of the west and the south-west; but in this darkest category there must be included one of Leinster and one of Ulster, respectively Queen's County and County Cavan. Donegal was hard hit too, and here it was that the first outbreak of epidemic scurvy was reported. Antrim was one of the counties in which the largest proportion of the recorded deaths was ascribed to dysentery. Those parts of Ulster where the farms were larger and a smaller number of the population wholly dependent on the potato, never sank to the same depths of misery and degradation

which were commonly experienced in Munster and Connaught. The counties of Ulster which suffered least severely were Down, Tyrone and Fermanagh, but in parts even of these, death had been very busy.

The figures of mortality given in the census of 1851 are quite unacceptable and understate the number of deaths, I believe, by more than one-half. They were compiled from two sources, information obtained from householders, and from hospital returns. As regards the former, the details were collected after the famine when whole areas were depopulated and desolate. Many of those who had suffered most and would have had most to tell, were gone, no one knew where. Tens of thousands died in their cabins without having been seen by any doctor. Others were found dead in deserted houses, in fields and in ditches, and were buried where they had perished. At the height of the famine one road inspector in Mayo reported that he had secured the burial of 140 corpses found lying by the wayside. The value of statistics supposed to cover these poor wretches who died uncared for, often far from their homes, can be imagined. Hospital records, too were subject to gross inaccuracies. Many patients were admitted in a dying state, suffering from starvation, from fever and from dysentery, often from scurvy or famine dropsy as well, and the diagnostic label chosen to cover them would depend mainly on individual fancy. Through the chaos resulting from the illness and deaths of so many members of the staffs, the books of some hospitals were unintelligible. In others there were long gaps owing to the same cause. All the same, in many hospitals the records were kept with exemplary care in spite of all difficulties and handicaps, but between this high standard of performance and failure to keep any adequate records at all, there must have been every grade of clerical defect and omission to vitiate still further the value of the official statistics. The following are the mortality figures, under the more important headings given for the famine years: Deaths from fever, 192,937; from dysentery and diarrhœa, 125,148; from starvation, 20,402—to which should be added most of the 22,384 deaths attributed to "dropsy." For 99,015 of the deaths, no cause is given. It is my belief that the total mortality attributable to the famine cannot have fallen far short of one million, equal to about one-eight of the population.

At an early stage of the war against famine and disease, the Government and its agents lost the initiative by two fundamental miscalculations. Basing their forecast on previous happenings, they assumed that after the partial destruction of the potato crop in 1845 the next year would bring, if not a rich crop, at least a sufficient one, for never before had the blight in its second year of duration swept across the country with all-consuming fury. For the second error, the Board of Health was responsible: the misreading of portents so that the diminution in fever observed in the summer of 1846 was taken as an indication that the local outbreaks had already begun to decline. It is impossible to-day to dissociate one-self from after-the-event wisdom, and assess the culpability of the members of the Board for their failure to discern the signs of the times. They could not foretell that the scheme of Relief Works, which looked admirable on paper, would miscarry

in practice—as the best laid schemes have a way of doing. Had it succeeded as was hoped and believed, there would have been no starving crowds to throng the roads and carry disease all over the country. If the Board had been wiser in its deductions, or if its members had included a prophet or a prophet's son, whatever preventive measures it might have proposed in order to meet the approaching epidemic, there was only one then available which could have had any immediate and far-reaching effect, namely, to provide additional hospitals to isolate all the sick as the cases arose, and so limit infection. In view of the obstruction offered later to the Board by local authorities when the epidemic was actually upon them, what would have been their response, and that of the Treasury, to proposals to provide hospital beds to accommodate tens of thousands of fever cases which did not then exist, and which never might exist? It was unfortunate that the Board of Health never enjoyed the confidence and full support of the medical profession. Two of its three medical members-Crampton and Kane-would never have been appointed if the advice of any representative body had been sought. Crampton chanced to be President of the College of Surgeons, but his long experience as surgeon to the Meath Hospital was no preparation for the burden he had to share. Kane had long ceased the practice of medicine, and, as Graves said, he was absolved of all blame for the shortcomings of the Board through his uniform non-attendance at its meetings. Corrigan, the third medical member, did not neglect his duties; on the contrary he was attacked for doing too much, and for being in himself the whole Board of Health. He was not popular with his fellows, who thought him vain and self-assertive; his head had been turned, it was said, by his being called on to take the Viceregal pulse. But to many of his critics any stick was good enough to belabour Corrigan and the Board. He was held responsible for the outrageous daily fee of five shillings which the Treasury paid for attendance at the fever hospitals, and for all the other instances of Government parsimony in medical affairs. His opponents fancied that by some magic which Corrigan did not possess, they in his place could have softened the stony heart of the Treasury in London: clearly they had never studied the peculiar quality that distinguishes the nether millstone. Curran, an able young professor of medicine, denounced the official fee as an insult, and refusing to accept it, continued to attend fever patients gratuitously. His lamented death from typhus, by some process of reasoning, was laid to the charge of the Board of Health. Some of the critics were biased by past controversies with Corrigan. Knowing that an outbreak of fever followed every famine with mechanical regularity, he had contended that hunger originated the fever. He was right to this extent, that it was the whole circumstances of famine that allowed the fever to blaze up and spread. One opposing party maintained that fever was engendered by overcrowding and uncleanliness. They were equally wrong and equally right, for these conditions cannot generate fever, but only enable a pre-existing focus of infection to extend its range. In blaming the relief measures for spreading disease by causing crowds of the destitute to congregate, they forgot, or did not choose to remember, that famine

fever had ravaged Ireland long before anyone dreamt of Poor Laws, Fever Acts, or Government aid. In what way nearly three million persons could receive food gratuitously from the hands of relief officials in one single day without causing the recipients to gather in crowds in the process, they did not explain. In fact, where the evils of overcrowding were at their most potent was in the homes of the poor, and in order to remedy this, millions of people must have been re-housed. It does not come within the scope of this address to discuss the possible results if the food shortage had been handled from its beginning with generosity, vigour and foresight. But once famine had the country in its grip, fever was inevitable, and no Board of Health at that date, even if given dictatorial powers and unlimited funds, could have brought the epidemic to a speedy and dramatic end. A century before the famine, Lynd had evolved the theory that typhus is carried by lice, but abandoned it through misinterpreting some of his observations on the spread of the disease, and medical opinion returned to the belief that typhus is transmitted by noxious emanations given off from the body of the sufferer. It was known that typhus even in its most malignant form would not spread if the subjects were thoroughly cleansed, and given fresh clothing which was kept clean. But of all the methods of disinfection used at the famine period with the object of counteracting the supposed bodily emanations retained in contaminated clothing and bedding, and rendering them infective, only two would have served to eradicate the lice which infested these, namely, stoying in heated ovens, and boiling. Neither of them would have been chosen for use as a general measure, because of the impossibility of setting up an organization and providing equipment on a scale sufficient to meet the needs of millions of people; and the substitutes adopted—so far as the destruction of lice in clothing and bedding was concerned were no more than a mere beating of the air. To-day, only by wholesale treatment of a population with the new insecticides, and this compulsorily enforced by some authority with the power of military law, could epidemic typhus and relapsing fever of a like degree be stamped out effectively and with dispatch.

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# The Happiness of the Child

By F. M. B. Allen, M.D., F.R.C.P.

Nuffield Professor of Child Health in the Queen's University of Belfast

# Inaugural Lecture delivered in the Great Hall of the University, February, 1951

It is customary on the occasion of an inaugural lecture to refer to one's predecessors in the chair to which one has been appointed. My position is different, however, in that I am privileged to be the first holder of the Nuffield Chair of Child Health in Queen's University. I am very conscious of the honour and responsibility of my office and the duties entrusted to me. I do not recollect, however, anything in my terms of appointment which stated that I had to deliver an inaugural address!

I should be lacking in courtesy and generosity should I fail to recall to this audience the names of some of those who devoted their time and interest to the welfare of infants and children; those who fostered the impulses of an awakening social conscience and directed the enthusiasm of the charitable public into those monuments of voluntary effort which are the pride of our modern civilisation; those who followed the precepts of Christian charity and set an example which was supplemented by official action and eventually absorbed by the State. John McCaw, Robert Leathem and Brice Smyth were my teachers and personal friends, and to their help and encouragement I owe much; Robert Campbell and Andrew Fullerton were the successors of Sir John Fagan, one of those who inspired the establishment of a children's hospital in Belfast in 1873. The surgical procedures which they initiated and in which they were pioneers are now regarded as commonplace. S. T. Irwin pursued an interest in developing orthopædics in Northern Ireland, and R. J. McConnell was—and still is—the ultimate professional opinion in the difficult abdominal emergencies of childhood.

To each and all of them I pay tribute, not only for the inspiration and encouragement they gave me, but also for their value as teachers in this medical school. Further, they earned the gratitude of those countless children who throughout the years have benefited by the skill of their professional services. Many parents are indeed deeply indebted to those pioneer physicians and surgeons for the successful outcome of their work in this chosen field of their endeavour.

The occasion of this lecture also affords me the opportunity of paying further tribute to the generosity of Viscount Nuffield, who, acting through his trustees, established the Chair of Child Health in this University with a very handsome endowment. The magnificent gift gave great pleasure to the Senate and Council of the University and was in itself a testimony to the personal interest and affection of this munificent benefactor for Ulster and its University. Had it not

been for this action it is very doubtful if the available resources of the University would have permitted the establishment of this Chair for many years to come.

### THE CREATION OF HAPPINESS.

In selecting a title for this address I deliberately chose one which would allow me considerable latitude. The happiness of the child is of a unique nature. It has a fundamental quality based upon trust and sincerity. Its happiness depends upon basic principles of good health, affection, security, companionship and activity of mind and body.

It would be quite impossible for me on this occasion to consider it from all these aspects and to co-relate the many contributory factors and influences. I have chosen, therefore (as you must have foreseen), to select mainly the role of physical health in relation to the creation of happiness.

I fear many of you may be disappointed that I do not dwell at length upon the particular topic which lies near your heart, but I hope I may touch upon some aspects of my subject to interest some of you.

It is not my intention to guide you as to the appropriate colour for the baby's nursery so that no psychological harm may result from some offensive colour scheme; nor shall I attempt to give any recipe for tantrums in childhood; nor the measures to be adopted when appetite fails in a healthy (but temperamental) subject.

Health is an essential feature of a child's happiness. Aristotle says:

"Let us grant that happiness consists in well-being with virtue, or in self-sufficiency of life, or in the life that is most pleasant and secure or in abundance of possessions with the power of defence and exercise," and goes on:

"If then happiness is of that general character it implies also the physical qualities such as health, beauty and strength."

A sick or ailing infant or child cannot be happy. How true this is, is realised when one sees the child in sickness. One notes day after day in the course of an illness how the sick child fails to smile—and then one day the expression has altered and a change in the countenance occurs.

Nurses as well as many doctors are aware of Guthrie's observation that when a sick child begins to smile again it is on the way to recovery.

Nurses are sometimes better observers of patients than doctors. Admittedly, they are in more continuous attendance upon their patients; but we as doctors can learn much from them—how to observe the change in expression, the alteration in the texture of the tissues, the cleaning of the tongue, the mottling of the skin, the brightness of the eye and the significance to be attached to these observations which are often more reliable than reports from the laboratory or the special departments of the hospital in assessing the recovery of the patient.

The definition of health is too formidable for me to undertake. It has often

been attempted. I suggest that we accept Aristotle that "health is that virtue of the body by which we exercise our use of our bodies free from disease." The achievement of happiness for children can only result from the understanding of home influences, the exercise of proper care and more knowledge of the cause of disease and its cure.

The Chinese have a proverb that:

"If you wish to be happy for an hour, drink wine;
If you wish to be happy for three days, get married;
If you wish to be happy for eight days, kill your pig and eat it;
But if you wish to be happy for ever—become a gardener."

Can this be the explanation of the injunction of so many mothers, "Go into the garden and play!"; of the instinct of the child to plant and tend his own patch of garden and to dig in the sand; and of so many seniors to retire to the country where they can exercise their talents and devote their energies to their garden!

Happiness, like freedom, is elusive. I am assured that happiness is unattainable in the absence of good health. Try as we may, some people can never reach this desirable state. For the child it is possibly more easy to attain because of its place in the cosmos. It is an infinitely small unit in the world's population and in the 3,000 million years of the world's history and in relation to the mountains, the rivers, the stars, the sun, the moon and the planets. What does the child know of current world or political problems? Fortunately, nothing. What does it know of the economic distresses of individuals, of nations or of the world? Of the dangers of war? Of the history of mankind? Surely this ignorance is of inestimable value in its possession of happiness.

Copernicus revolutionised man's simple ideas of the canopy of heaven; Newton discovered the law of gravity; Darwin gave us a new view of the origin of man; Einstein has not solved the riddles of time and space in spite of his theory of relativity; Rutherford smashed the atom and now a new age of nuclear physics is upon us.

And yet to mothers the infant is the most important thing in the world. Children carry the hope of mankind. In our democratic culture they give ample evidence of the potentialities of the human spirit and its engaging qualities.

We as adults should imitate them to the extent of endeavouring to capture something of their transparent honesty and sincerity. It has taken man a long time to gain much knowledge of himself and to be aware of some aspects of the universe in which he has his being.

#### THE CHILD IN THE PAST.

It is good for us to look back upon the past to see how far we have travelled and to learn the course upon which we are set.

The doctrine that parents existed for the sake of children was not accepted a couple of centuries ago. The loving care and devoted attention bestowed upon

children of to-day would have appeared ridiculous to sensible people of the 18th century.

Even the sentimentalist Rousseau abandoned all his children, one after the other, to be brought up as unknown foundlings. His conduct, while regarded as odd, was not thought to be vile!

Those of you who are interested in the social history of our country will recall that child labour was an accepted practice 150 years ago. The depression after the Napoleonic Wars with the onset of a revolution in industry demanded the cheapest form of labour without regard to social consequences. Children were engaged in many occupations and worked for long hours daily.

There were factory schools where children were admitted at the age of three years and taught the elements of reading. At 4 years they began technical instruction and at the age of 5 or 6 they were able to earn 2d. or 3d. per day.

The children of the poor were neglected by a nation absorbed in industrial survival. They were the only ready source of cheap labour and Lord Shaftesbury maintained that parents pledged their children's labour for money. They were employed in the cloth and silk mills of England for as long as fourteen hours a day, and others were lodged in households in various parts of the country ostensibly as apprentices to a trade or craft. One master chimney-sweep testified to boys of 4 years being employed to sweep chimneys; it was quite common practice for children of 7 years to be engaged in this occupation. Hanway suggested that master chimney-sweeps should "breed their own children to the business, then perhaps they will wash, clothe and feed them. As it is, they do neither."

The employment of children of 7 or 8 years in coal mines was almost universal. Some of the children were engaged in pushing or pulling trucks along the pit tunnels. "They were harnessed like dogs in a go-cart" and crawled on all fours down passages in some places only eighteen inches high. Others worked at the pumps standing ankle deep in water for twelve hours. One child of 6 years is stated to have dragged a load of half a hundredweight every day up a distance equivalent to the height of St. Paul's Cathedral.

They belonged to agricultural gangs, they worked in brickfields, they were hired out by Boards of Guardians when over 5 years to work in factories, ostensibly as apprentices, where their masters saw to it that they worked as many hours daily as adults!

Was it lack of social conscience or an indifference to sentiment that permitted such conditions in our country? It is to be remembered that Wellington was winning battles in the Peninsula with a few thousand soldiers and a very unreliable supply system. Napoleon had invaded Russia and suffered the destruction of an army in the retreat from Moscow. England had subsidised the nations of Europe with gold to maintain freedom. The Battle of Waterloo, which eventually brought peace, was recorded under a subsidiary headline reporting the "battle in the

Netherlands" and pride of place was given to sentence of imprisonment for a jarvey for refusing a fare! But there remained the post-war depression and the need for economic recovery.

At that time anxiety was expressed about the size and powers of the Civil Service. The Foreign Office had a staff of twenty-eight (including two undersecretaries and a Turkish interpreter) and the Home Office consisted of twenty clerks!

The works of Malthus had created in serious minds a haunting fear that the increase in population would outgrow the earth's productive capacity and culminate in famine. The beliefs propounded by Adam Smith that the wealth of men and nations depended upon the unimpeded operation of economic law were held in reverence. These theories were responsible for an overshadowing policy that "the more the population increased and the greater the consequent suffering of the poor, the more incumbent it became on those who governed to refrain from any interference with economic processes." The most one could hope for, in the view of the professors of this dismal science, was that hardships suffered by them in the course of obtaining food were in reality blessings, "since without them they and all mankind would starve."

The most eminent economist of our generation believed that "the great puzzle of effective demand with which Malthus had wrestled had vanished from economic literature." Let us hope so. Let us encourage economists who will endeavour to solve our difficulties and at the same time consider the deeper problems with which our society is confronted. Let humanity and justice prevail in the study of our present-day situation.

## THE EMANCIPATION OF CHILDREN.

Emancipation of children took place gradually after the African slaves had been freed. To men like Addison, Steele, Shaftesbury and many others this demanded years of argument, publicity and campaigning. Cobbett said in the House of Commons that the bulwark of England apparently lay, not in her Navy, but in the labour of thirty thousand little factory girls. He was regarded as pronouncing "perverse nonsense."

But, as a result of much effort by prominent individuals, by the establishment of institutions for the care of the sick and ailing, by the founding of homes for the unwanted and the abandoned child, laws were enacted which gradually over a period of years prohibited child labour in factories and mines, introduced schemes for infant and child welfare, encouraged the building of hospitals and the setting up of dispensaries. Until, ultimately, we have arrived at the welfare state as we know it to-day. The first Children's Hospital was opened in London in 1852 by Charles West and now there are forty-seven in the United Kingdom. It is true that a nursery for sick children was established in 1686, but it did not survive for long. Otherwise children received attention at one of the hospitals (St. Bart's

or St. Thomas's), although some hospitals refused admission to children under 7 years and others if under 3 years. At that time the death rates among the newborn, in infancy and as a result of infectious diseases such as whooping-cough, measles, etc., was very high; gastro-enteritis was responsible for a heavy mortality.

#### RETROSPECT.

We are entitled to look back and gain a true perspective of the situation as it existed when the Tsar of Russia visited London after Waterloo,

"The visitors saw something of the great charities—the offspring of private benevolence—with which the islanders had endowed their capital. They visited the Charterhouse, the Foundling Hospital, the palaces built for naval and military pensioners at Greenwich and Chelsea, and dined in the Halls of the Goldsmiths and Merchant Taylors—representatives of Corporations which spent between them as much on relieving and educating the poor as a Continental sovereign on maintaining his Court. The British capital had twenty voluntarily supported hospitals, 120 almshouses, 50 free dispensaries, 45 endowed free schools, 250 parochial schools educating, clothing and feeding nearly 20,000 children. St. James's Palace was the smallest and least imposing in Europe. London could claim that her real palaces were hospitals—Wren's Greenwich and Chelsea, Gibb's St. Bartholomew's with its Hogarth staircase, St. Thomas's with its four great quadrangles. 'In no other country was there so much voluntary corporate goodness towards the hungry, diseased and weak'.''

The welfare services were essentially in the hands of well-meaning individuals who were responsible for either founding or forcing state or municipal bodies to establish centres for the care and welfare of the sick, the abandoned and the destitute.

The care of the sick child was originally part of the practice of obstetrics. Then he was regarded as coming under the care of the physician being an immature adult and not requiring special study. Following this a generation has arisen who regarded "pædiatrics" as medicine applied to a particular age group and demanding special study in relation to development and growth. Now the tendency is to devote special interest to the care of the premature and to study the influence of antenatal conditions on the infant—unborn and newborn. In 1927, under the inspiration of Professor C. G. Lowry, a pædiatrician was appointed to the Belfast Maternity Hospital to become a member of the team of those responsible for the welfare of the newborn infant. This type of appointment is now commonplace. Thus has the circle of the relation between the sick infant and the doctor been completed, and the association with the obstetrician once more established.

The pædiatrician is a collaborator in the obstetric team and takes on the care of the infant after birth. The evolution of infant welfare centres is a great credit to our generation. By developing it, using it as a medium for education, for the

detection of minor illnesses in their early stage before they become serious, there would appear to be a great opportunity for achieving results.

There has been considerable progress in recent years in securing a reduction in the death rate in infants and children. The use of "sulpha" drugs and antibiotics such as penicillin, streptomycin, aureomycin and chloromycetin has been of great assistance as will be seen in the significant fall in the last ten years.

Deaths (in England and Wales) of children of 1-5 years expressed as a rate per million have fallen dramatically in the last thirty-five years.

|                               |    |       | (1911-20 | to | 1948) | 1.1 |
|-------------------------------|----|-------|----------|----|-------|-----|
| Measles from                  |    |       | <br>2279 | to | 55    |     |
| Scarlet fever from .          |    |       | <br>253  | to | 1     |     |
| Whooping-cough from .         |    | • • • | <br>1091 | to | 88    |     |
| Diphtheria from               |    |       | <br>811  | to | 21    |     |
| Tuberculosis from             | •• |       | <br>1505 | to | 275   |     |
| Bronchitis and pneumonia from | om |       | <br>4441 | to | 321   |     |
| Enteritis and diarrhœa from   |    |       | <br>1212 | to | 58    |     |

It is true, therefore, that much has been achieved in the last 100 years, and a great deal of the progress in the past 25 years. It can be claimed that much that was unknown about nutrition is now commonplace; vitamins have been discovered and their relationship to deficiency diseases ascertained; the study of growth and development in relation to illness and disease is providing valuable information; the better care of the infant in the antenatal and neonatal periods is procuring a lower death rate and, it is to be hoped, a healthier generation of children; the treatment of infectious diseases such as pneumonia, whooping-cough, and diphtheria with modern drugs and adequate preventive measures has saved many lives; better education of mothers in infant feeding has reduced the appalling mortality of infective diarrhœa; rickets is now a disappearing disease and should be unknown; diphtheria is essentially a preventable disease and should be as rare as smallpox is, if parents would insist upon preventive inoculation. And so one could catalogue a list of achievements which reflect the greatest credit upon the mass of scientific, professional and voluntary workers who have laboured so diligently and so fruitfully.

# CONGENITAL MALFORMATIONS.

A substantial proportion of deaths in infants is due to congenital malformations which are incompatible with life.

It is not always realised how much credit is due to observation and anticipation. In 1939 a severe outbreak of German measles, with an unusually high incidence in adults, swept Australia. Soon after it was noticed that there was an increase in congenital cataract in children, and it was established that there was a relationship

between the occurrence of this disability in infants and the fact that the mothers had had this relatively innocuous form of measles in the early weeks of pregnancy. It is now generally recognised that this incident in the mother can influence the development of the unborn infant. The acceptance of this view promises to revolutionise medical thought, because other factors may be found which have an equal influence.

If research substantiates the new idea that the health and nutrition of the mother (especially in the early weeks of pregnancy) has a definite relationship with the structural perfection of the infant, then indeed we may be within sight of the time when we can regard congenital malformation as being in the realm of preventable disease.

### THE PROBLEMS OF THE FUTURE.

The Premature Infant.—One of the greatest causes of loss of life in the newborn period is that due to premature birth. The infant born before its normal term is an immature individual who requires very special care and treatment. The obstetrician tries to avoid premature birth, but he is usually compelled to facilitate the event by circumstances outside his control.

The measures which must be adopted are being learned by observation, by knowledge of the factors which cause death, by intricate laboratory studies. Doctors and nurses as well as mothers must co-operate in conserving the strength of this immature individual by the utmost care to avoid infection, to secure warmth and to provide appropriate nourishment.

Already the loss of life from this cause has been substantially reduced by more general application of these relatively simple principles.

Tuberculosis.—In England and Wales during 1947 all forms of tuberculosis caused 23,075 deaths, of whom 1,955 (8.5 per cent.) were children. In Northern Ireland during 1946 tuberculosis caused 1,111 deaths, 157 (14 per cent.) being children. Deaths from tuberculosis have the highest numerical significance of the infectious diseases in childhood. In a study in Birmingham the numerical order of fatal termination of some infectious diseases was scarlet fever 2, diphtheria 15, measles 48, whooping-cough 114, and tuberculosis 203.

The disease was described as "scrofula," first mentioned in English literature in 1664 and known as the "King's Evil." For centuries it was believed that the touch of a Royal hand had the power of healing. There is no doubt that the condition originally described as scrofula and reiterated by subsequent writers was swollen glands in the neck, usually due to tuberculosis. It is believed that the last "Royal Touch" performed was by Prince Charles Edward in 1745.

The cause of tuberculosis has been known since the discovery of the tubercle bacillus by Koch more than sixty years ago. We know that humans become infected from other humans suffering from tuberculosis and from the ingestion of infected milk.

If all cows' milk were obtained from tubercle-free cows or were effectively pasteurised before consumption, bovine tuberculosis in man would almost disappear. The sick, the maimed and the crippled would no longer be a reflection upon us, and the orthopædic problems of the tuberculous sufferers would cease to be an anxiety.

Infected humans, by coughing and by the careless disposal of their sputum account for some 80 per cent. of the fresh cases. Surely we should be able to secure segregation or better hygienic habits of these people who are such a menace to the infant and the adolescent. It should be realised that the result of a first tuberculous infection in infancy carries a very heavy mortality rate, and at the end of childhood the results are also very unfavourable.

There is an opportunity for an intensive educational campaign to impress upon all grades of the community the dangers of allowing infants and adolescents to associate with sufferers from "open" disease and the consumption of infected milk.

In recent years considerable attention has been directed to the value of vaccination against tuberculosis with B.C.G. vaccine. The experience over a period of twenty-five years in Scandinavia has convinced many of us that herein lies a method of reducing the mortality from this dread disease. Tuberculous meningitis is unknown in a successfully vaccinated child. It is heartening to record that facilities for B.C.G. vaccination have been established in Northern Ireland and are being developed. Already many newborn infants have been immunised, as well as university students and nurses; it is hoped to extend this service to children leaving school, to those entering industry. Thereby provision will be made which will be a credit to our generation by reducing the incidence of the disease and its mortality.

Streptomycin and P.A.S. have been useful in the treatment of certain forms of established disease. In association with sanatorium treatment, fresh air, good diet and adequate rest much can be done to facilitate recovery from tuberculosis of the lungs. The modern practice of surgery has advanced so far that certain specialised operations secure a cure in patients who would otherwise die.

This all entails a prolonged period of invalidism, a great deal of nursing care and much surgical skill—all of which would be eliminated by prevention and reduced by early diagnosis.

There is an opportunity for an educational campaign to increase preventive vaccination in the uninfected, to avoid dissemination of the disease so that its dread spectre may disappear from our civilisation.

Rheumatism.—Of rheumatic fever in children—known better as juvenile rheumatism—I have nothing very encouraging to say, except that possibly its incidence is decreasing. The clinical picture of the initial illness has altered without any change in the risk of ultimate rheumatic heart disease occurring which takes such a heavy toll amongst adults in the most fruitful years of life. Our senior colleagues recall for us the days when the patient with rheumatic fever suffered

excruciating pain—so much so that walking in a room or hospital ward evoked an urgent protest from the sufferer.

Nowadays it is an insidious disease of childhood with little or nothing but vague ill-health, some loss in weight and a slight temperature to indicate the presence of the infection and the serious consequences which may ensue from an apparently trivial amount of ill-health.

Numerous children have their first attack between the ages of five and ten years. The heart may escape, or the damage may be slight and of no consequence. Repeated attacks almost inevitably lead to valuular disease of the heart with considerable handicapping of the function of this organ. A breakdown may become evident in early adult life and death occur before the half-century is reached.

We are as yet not fully aware of the cause of this infection. We know that prolonged rest is necessary in the early phase to reduce the permanent damage of the heart; also to avoid the risk of re-infection. The provision of a long-stay hospital is much needed. This would provide facilities for nursing care, medical observation and suitable conditions of nutrition and housing.

Intensive research ought to determine the cause of this relatively common disease. If the origin eludes us, perhaps a line of successful treatment may be discovered. There is some reason to believe that one of the new hormones "Cortisone" or A.C.T.H. may indicate the way to a cure and may lead us to discover the cause.

Before passing on to some other aspects, I wish to interpose a plea for the provision of long-stay hospitals and the necessary facilities for children who require prolonged medical supervsion, such as the tuberculous or rheumatic; for children suffering from orthopædic deformities who need the technical help which can only be given by the patience and skill of the surgeon and the physiotherapist; for the handicapped and homeless child who must receive special consideration.

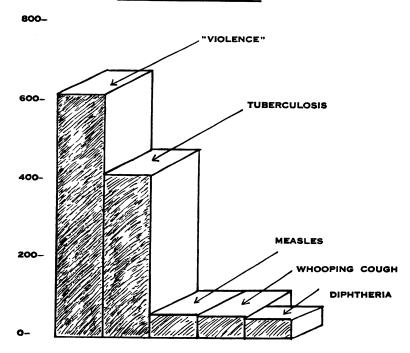
For all of these, educational facilities in the long-stay hospital must be secured, because, while the child is being made physically fit for his place in life, he must also be educationally equipped to maintain his position in the struggle for existence. Here indeed is an opportunity for the fusion of interests into a comprehensive scheme for the increase of happiness.

Deaths by "Violence."—The loss of life and maiming of children as a result of "accidents" is on the increase. The children are the chief victims of nearly 7,000 fatal accidents in the homes of England and Wales every year. Road accidents, associated with the increase in vehicular traffic and the greater speed of travel, are to some extent understandable, but education and propaganda should reduce the toll.

A review of the mortality figures in the 2-5 year age group shows that "violence" was responsible in 1948 for 616 deaths per million, compared with 430 from tuberculosis, 46 from diphtheria, 73 from measles, and 74 from whooping cough.

Accidents in the home through burns account for one fifth of these. Almost every day a child is admitted to our hospital suffering from burns acquired in the home by spilling a kettle or teapot over themselves or by their clothing catching

## DEATH RATE PER MILLION



fire, or by falling into the fire. The most dangerous age period for one of these accidents is that up to four years. A number of these children die after a period of intense suffering; those who survive require all the skill of the physician and later the craftsmanship of the plastic surgeon and his trained technical and nursing staff. It may take months of specialised treatment and care to save a life and to restore the function of a damaged body.

#### SOCIAL ASPECTS OF CHILD CARE.

Social Medicine.—Social medicine is in the van of popularity at the moment. The study of environmental conditions in relation to health and illness must be informative. Possibly some of the social studies now being carried out will help to clarify the complex issue of certain diseases. The investigation of factors of heredity in relation to disease will possibly help to solve our difficulties, particularly in diseases such as rheumatism, disturbances of growth, and diseases of muscles and the nervous system.

There is a great opportunity for integrating the social services in child health. The whole pattern is like a jigsaw, some parts of which are already in place, others

are misplaced and some are absent. So much has grown in a haphazard manner as the result of the enthusiasm of separate benefactors.

History bears testimony to the stimulus which the care of the child has always derived from private philanthropy and individual effort. The picture tends to become confused from time to time by the variety of problems involved. It is our duty to avoid wasteful duplication of effort and unnecessary competition by a proper fusion of interests.

I am convinced that the divorce of the family doctor from his proper role is to the disadvantage of the health service. His services should be widely and generally used in all health service activities. He is just as much a specialist in medicine as the physician or surgeon, only his field is that of family health. My view is that by providing facilities for post-graduate refreshment his interest in infant and child health will be re-awakened and his place as the family counsellor will be more useful than that of many of the more remote members of our profession.

The municipal and state welfare services—infant clinics, school health services, etc.—should not be carried out in seclusion away from the family doctor and the hospital. Already the association of some of these medical officers with the hospital has given a new lease to a valuable service, and I personally would like to see a very close liaison between these services and the hospitals.

The intimate day to day study of the sick reveals the importance of the wider study of the patient in relation to his environment, particularly at home. A better knowledge of home conditions, of factors of heredity and of social problems will often facilitate restoration of health.

The role of health visitors in assisting towards recovery is being more and more realised by those of us interested in the social aspects of medicine. The provision of home helps to assist the mother in her arduous duties as housewife when faced with illness in the home is essential and deserves to be developed.

It is not enough that we as doctors should diagnose and treat disease in hospital; we must also be aware of home conditions and personal anxieties which retard convalescence and fail to sustain health when it has been secured.

## THE HANDICAPPED CHILD.

The provision of help for the handicapped child is much further advanced in Great Britain than here. Private enterprise and individual charitable impulses have led to the development of a multitude of services devised to assist the child who is the unfortunate victim of a physical handicap. This may be due to the loss of a limb or its function through poliomyelitis, to deafness, to loss of sight or to mental inferiority.

Much can be done to restore, partially at least, much of the function of a limb affected by poliomyelitis or disabled through a street accident or burns. The physiotherapist who is provided with the facilities of a modern department including baths, gymnasium, electrical apparatus and equipment for remedial exercises can do valuable work.

The child who is deaf must be investigated by special apparatus to ascertain precisely the type of deafness from which he suffers. Upon this depends the line of treatment to be pursued.

Loss of sight or defective vision can interfere seriously with education and the earning of a livelihood. Here again modern equipment and specialised training can convert an economic liability into a national asset. There is no field in which there is a greater opportunity for co-ordination and co-operation. The numerous interests concerned in the care of eyesight and the remedying of defects should be unified and simplified into a service.

The ascertainment of the degree of mental inferiority or backwardness is a painstaking investigation which is none the less imperative if the child is to be saved from unnecessary suffering.

The presence of a physical handicap is a challenge to our utmost endeavour. We must accept it with the purpose of securing an outlet for the child's physical and mental activity which will give the maximum of happiness.

Care of the Teeth.—An atmosphere of complacency cannot be associated with the dental services of Northern Ireland—particularly as regards children. That another generation of children is being allowed to grow up without a proper dental service is a short-sighted policy. The harvest of illness and ill-health which is attributable to dental disease is surely sufficiently well-known to demand urgent consideration. The failure to adopt a policy of conservation and proper dental care of children will be looked upon by posterity with disfavour and to our shame.

Nursery Schools.—Private enterprise still shows the way in several aspects of child health. The maintenance of Nursery Schools is one of the amenties provided by voluntary effort which has won the appreciation of parents and doctors. The gentle discipline, the inculcation of regular habits and hygienic routine set the young child further on the road to good health and happiness.

Mental Health.—Of the psychology of childhood and the necessary psychiatric services I do not wish to say much because I could easily be led into an extensive field of controversy and a prolonged discourse. This I will say, however, that the more understanding there is of the child's mind the better we shall appreciate problems of behaviour.

Experts assure us that many of the psychological breakdowns of adolescents, leading to delinquency and mental misery, can be traced to events in early life. The establishment of a happy home environment with a proper relationship between parent and child is the best guarantee of mental happiness in the future.

It should always be remembered that the child's home is his world and what he experiences there will be reflected in his conduct outside the home. If there is an atmosphere of security and stability his conduct will correspond; whereas if there is constant bickering or quarrelling at home, displays of temper, or a lowering of the standard of home life, his activities with his colleagues will tend to be similar.

Child Guidance is a valuable service provided it is directed by professional people and does not become the playground of the enthusiastic amateur who knows nothing of physical medicine or diseases of the nervous system. The tragedy will be the dissociation of child psychology from the hospital.

In this connection I must record my firm conviction that the sick infant or child should obtain the care and affection of its mother, preferably at home. If the facilities are not available at home then the patient must be treated in hospital and if necessary the mother should assist in attending to his needs by being provided with accommodation also.

### THE MODERN HOSPITAL.

To maintain good health demands the integration of the social and health services provided by State and Municipality using the facilities of hospital and maintaining the desirable relationship between family doctor and patient.

To achieve the restoration of health is the duty of the hospital service. It is not generally realised how complex is a modern hospital. The visitor only sees the patient in the ward. He is unaware of the laundry, of the kitchens, the domestic and nursing staff who also have to be fed and housed. A dietitian working in the diet kitchen prepares special diets. Radiographers take hundreds of X-ray pictures which are interpreted by the radiologist. The laboratory carries out special examinations and makes special tests upon which the ultimate diagnosis depends. The physiotherapists, the speech therapists, and the almoners each have their duties to perform. Clerks type reports, telephonists answer numerous telephone calls. The nurses, in addition to performing their essentially nursing duties, have to undergo special instruction and attend their "school" to fit them for their profession.

Doctors and students are also learning the nature of disease, its recognition and its treatment.

#### THE NEED FOR RESEARCH.

All the time investigation and research must be active. This demands hard work, much thought and study. Modern surgery of the heart is one of the most specialised of all operations. The diagnosis must be precise and before the surgeon submits the patient to the operating theatre weeks of unique examinations may have been undertaken each in itself demanding special apparatus, exceptional skill and experience.

A health service must be progressive. It can never remain static. Otherwise it will die. What of the future of medicine? I am reminded of Osler's reply to the question as to where the leadership in medicine would rest—"It is hard to say; but of one thing we may be certain. Athene places her shrine only where there is freedom, and where there is most freedom there will be made the future development of scientific medicine."

I am not a disciple of doom. It is in the mind of some that the world is running down into an abyss, with some appalling catastrophe not far ahead. I am with

those who are convinced that while the situation is perilous, it is rich in challenge for those with ideals; that there are golden opportunities for those who are prepared to face times which are arduous and even hazardous.

#### ACKNOWLEDGEMENTS.

I am indebted to Arthur Bryant's "English Saga" and "The Age of Elegance" tor much of the historical information contained in this lecture; and to W. S. Craig's "Child and Adolescent Life in Health and Disease"; also to Dr. E. A. Cheeseman for the mortality figures for the years 1911-20 and 1948.

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# **REVIEW**

HANDBOOK OF DIAGNOSIS AND TREATMENT OF VENEREAL DISEASES. By A. E. W. McLachlan, M.B., Ch.B. (Ed.), D.P.H., F.R.S. (Ed.). Fourth Edition. Pp. 368. Illustrated. Edinburgh: E. & S. Livingstone. 1951. 17s. 6d.

Dr. McLachlan's Handbook on Diagnosis and Treatment of Venereal Diseases first appeared in 1944. Its well-deserved popularity, together with the rapid advances in the treatment of Venereal Disease since the introduction of the antibiotics, have been responsible for a fourth edition since that date.

The present edition shows changes chiefly in the sections dealing with the treatment of the Venereal Diseases, though other modifications have also been made. A sane balanced outlook on modern therapeutic methods is set out and the importance of combining the old methods with the new in the various stages of the treatment of syphilis is upheld. The danger of the Jarisch-Herxheimer reaction in relation to penicillin therapy in both congenital and acquired syphilis is rightly emphasised: a point which is sometimes overlooked by the modern clinician. The inadequacy of and the disappointing end results obtained in the early days of penicillin treatment of syphilis is mentioned and the importance of prolonged clinical and serological follow-up after treatment of syphilis is stressed. Caution is expressed with regard to the end results of penicillin therapy in C.N.S. syphilis where much depends on the individual clinician treating the patient.

Modern advances in the treatment of Gonorrhoea and other Venereal Diseases is discussed. The necessity of careful diagnosis before the exhibition of the antibiotics, and the importance of thorough follow-up after treatment is emphasised.

The standard of production has been maintained with clear print, good paper, and excellent illustrations. The temptation for successive editions to increase in size has been avoided by the pruning of all irrelevant material and the present work contains only four more pages than the first edition.

This excellent little book has maintained the promise of its earlier editions, and can continue to be recommended with confidence to both the medical student and busy practitioner alike.

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J. S. McC

# "The General Practitioner in Eclipse"

By J. B. Young, M.C., M.B.

# Chairman's Address, Belfast Division, British Medical Association

As you will see from the notice convening this meeting, I have selected as a title for my remarks: "The General Practitioner in Eclipse."

Eclipse has been defined "as an obscuration of one of the heavenly bodies by the interposition of another." This is not the sense in which I desire to use the word eclipse, but rather in the sense of "being placed in ignominious obscurity." Much prominence has been given of late to the invidious position that the general practitioner finds himself to-day, in the new scheme of things, in contradistinction to the place of honour and dignity occupied by him in the past and the respect in which he was held by the community at large prior to the inception of the New Health Act.

The causes which have led to this position are numerous, some important, some not so important, and you will readily appreciate that only a few can be selected for review in the short time at my disposal to-night, and that those selected by me are not to be considered as comprehensive or the all important ones, but rather are they intended to focus your attention on an already established fact and to stimulate your interest in the devaluation of the general practitioner, its causes, and its effect on Medicine as a profession, and on the community as a whole.

I am bearing in mind that a Chairman's subject matter on this occasion should not be too dogmatic or controversial since there will be no discussion on the remarks that I am about to make.

Almost every medical man has the knowledge that a Medical Revolution is taking place.

The "Hungry Forties" of the past and present century have gone. Like the building of poor-houses in the last century to house the desolate and starving, many of us see an analogous conception in the present century to house those of us who, through no fault of our own, are compelled by economic and other reasons to be incarcerated body and soul, in the bureaucratic edifice that surrounds our profession to-day.

Gone are the days when we practised for conscience sake, and I regret to say many of us have succumbed to the temptation to practice "for dear sake." Gone are the days when the level of one's fee was directly proportional to the height of the patient's manure heap. Gone are the days when the decency and respectability of our patients may have been somewhat erroneously measured by their promptness to pay their bills.

The general practitioner of pre-Health Service days was conscious of the fact that his function had altered in comparsion with that of his counterpart of thirty or forty years ago. In those days it was within the compass of the general practitioner to have a good working knowledge of many of the specialities as developed to that time and to bear single-handed the responsibility for diagnosis and treatment of most of the ills of his patient. The highly specialised branch of Consultant and Specialist, as we know it to-day, was then in its infancy.

The general practitioner in more modern times, realizing the time-consuming techniques and study necessary to carry out new tests and aids to diagnosis, contented himself rather with the interpretation of results and in the light of these in controlling the treatment and management of his cases. In accepting these responsibilities, he felt that he was securing for his patients all that Science with its many advances had discovered for mankind.

He alone possessed the essential background with his knowledge of the factors of heredity and environment.

As a class we aspired, each of us, to merit the appellation of Sir William Osler, "That flower of our profession: the cultivated general practitioner."

An historical retrospect reveals that towards the end of the second World War it became apparent that new values on life and a new out-look would most likely emerge at its end. And it was at this time that all political parties saw the appeal of social welfare to the masses of the electorate and its usefulness as a political pawn. What the other parties would have done remains a conjecture.

No doubt however is left in our minds as to how the present Government in framing their social welfare platform, placed the medical plank well to the forefront.

By inaccurate statements, misleading slogans, and propaganda, they gilded the pill for the public and metaphorically caused the general practitioner to walk the aforesaid plank.

One can remember well J. B. Priestley's heartening prophecies of the "brave new world" which he envisaged with such apparent sincerity, when he gave his Sunday evening broadcasts during the late war. If any of you should have any interest in this author's peregrinations I would refer you to his book entitled "Delight," where the inference is that the outcome of all the planning leaves this particular planner speechless.

The medical profession for almost thirty years has been anxious to broaden the medical service for the nation, committing the profession to some form of state help and incidentally increasing control.

The old voluntary system was becoming obsolete because of legislation and the tendency of state control to enter into almost every branch of industry and the professions, coupled with the rising costs of overhead expenses.

There was also the necessity for launching a programme for building medical institutions to house the medical services to a degree unparalleled in the history of medicine. The B.M.A., recognising all these things, had proposals to fulfill the requirements of the changing scene.

In view of these facts, one may ask, How has it come about that the general

practitioner has been placed in Eclipse? What are the causes? and are there any remedies for his ills?

But the conception and delivery of the present Government's scheme was, in my opinion, ill-conceived, most untimely, and contrary to the laws of medical evolution.

Looking in retrospect, two facts which affected our profession adversely, stand out clearly. Firstly, the Ministry of Health obtained as its head a Minister of Health unsympathetic to the profession as a whole, and whose plan was to split the profession in twain. I shall deal with this gentleman later. "Divide and rule" was the order of the day and with what success is known to many of us; and secondly, our emergence from a war that left us in no fit state to have laporotomies performed at the hands of inexperienced political surgeons.

Our rank and file were war-worn, tired men and women, with no reserve left to withstand these onslaughts, and there was coupled the fact that service medical personnel would, most probably, flood the medical labour market, for a time at least.

To attack one's enemy at his weakest point is an age-worn maxim, and it soon became apparent that the general practitioner was the weakest link in the medical chain.

A vested interest had been created for the first time by the working of the N.H.I. Acts. Many members of the profession had committed themselves financially, by the purchase of practices, and houses to practice in. A yard stick already existed as a means to measure our remuneration in the form of per capita payments. All this was in striking contrast to the circumstances surrounding the consultant and specialist, who of necessity had to be absorbed into the new scheme, and whose remuneration, was, except for the Spens recommendation, an unknown quantity, subject to negotation and agreement between them and the Government. This circumstance was, to my mind, the bifurcation that enabled the Government to separate us, a little perhaps at first, but by this division the unity of the profession was broken. Each of us was allowed to travel along his respective pathway to the detriment of the weaker branch.

There are some who quite honestly believe that this rent or cleavage was caused by our own actions here in the Northern Ireland Branch, in "Changing horses in mid-stream," or, as I prefer to put it, in "Changing riders in mid-stream," quite a different thing. I do not subscribe to this thesis for one moment. The same problems remained with a different set of individuals in the saddle.

### WHAT ARE THE CAUSES.

Two have been mentioned already:—

- 1. The Person who became Minister of Health after the General Election of 1945.
- 2. The timing of the introduction of the Act, one found to be most propitious to the planners.

To these I would add:-

3. The attitude and conduct of the General Practitioners themselves, before the appointed day.

- 4. The Division that occurred in our ranks, in separating the consultant and general practitioner, into two major groups, whose functions and ideals should have been complementary and co-terminous.
- 5. The changed relationship between Patient and Doctor that sprouted up almost overnight.
- 6. Lastly, the emergence of the administrative Machine.

#### LET US CONSIDER.

(3) The conduct of the general practitioner himself. One poses the question, To what extent has he been responsible for his present plight? Can any of the deflections or defects be ascribed to his own action, or want of it? The answer, of course is in the affirmative. As a class the general practitioner is no less immune to the frailties of human nature than any other. Granted, apathy and lethargy have been responsible for much of his ills. Guilty to a degree perhaps of being more parochial in his outlook than others.

I know of no other professional body or organisation whose outlook was so lacking in foresight as ours was, some three years ago. It would now appear to have been ill-fitted for the mighty task of steering our branch of the profession through all the shoals and reefs that can be so easily negotiated by the planners, politicians and others.

What other body would have accepted service and conditions of service such as ours, without previous reference to our remuneration and the studied details of those conditions of service?

I say none but the general practitioner branch of medicine. No attempt has been made to ameliorate our unenviable lot in a service which some with more foresight than others predicted, and whose voice was like that of "one crying in the wilderness," unheeded or unheard. I notice here and there a pricking of the ears at the mere mention of the word remuneration. I venture to say that this matter is in large measure at the root of our troubles, and I am realist enough to think and to say that an adequately paid general practitioner service would place us once more in our proper perspective in medicine and society.

(4) The division between the Consultant and General Practitioner.

I have already indicated how this came about and how an accident of circumstance may easily change the issue without either group being party to it. In discussing the general practitioner in eclipse, at first I was diffident to mention the position of the consultants because many of them have been and are amongst my closest and dearest friends, who have rendered to me and mine in the past services both medical and otherwise, which have placed me in their debt for ever and a day. But I realize that it is incumbent on me to comment on the cleavage—one which has widened with the years.

It will be admitted that the consultant's dependence on the general practitioner is not as great as in the past—the economic urge is neither apparent nor prevalent, and many members of our branch of the profession express openly and in confidence their dismay and concern at the changed attitude towards them by their

former colleagues, and long for a return of the days of yore. We no doubt envy them their capacity to negotiate terms with the Ministry, but we are still loth to forget their withdrawal behind the iron curtain that separates us to-day, and of their virtual disappearance from and apparent loss of interest in Association affairs. Even they in an equalitarian system, have been made recipients of distinction awards, in contra-distinction to the general practitioners who, be he ever so senior, must continue to rank in the eyes of his employers as on the same rung of the ladder as the latest recruit. Experience, tradition, personality, and, dare I suggest, a bed-side manner: do they count for nought in these equalitarian days?

What a difference it would have made in the settlement of our affairs, if a united front had been presented by the profession, no expression of satisfaction that, Spens had been implemented on the one hand, and not on the other, could have been possible. Whilst the general practitioner is still left wallowing in promises made to review his remuneration within a year of the Appointed Day. What a long year it is? and it would appear to me to be no nearer its end. The Old Order changeth, and the time for a changing of "the vestures of our faith" are upon us. New ideas, and a new conception of medicine and general practice, are being evolved. The general practitioner of the past, as we knew him, is fast disappearing, but still these two great branches of the profession remain complementary one to the other, and must be joined together working in harmony once more, for the good of our patients, and the profession as a whole. Then, and not till then, will the community receive that benefit and aid which an enlightened profession can bring to suffering humanity.

## (5) The Changed Relationship between the Patient and the Doctor.

The Entrance of the administrative machine into the every-day life of the individual, during the war and post-war period, with its control of commodities and services, made the average citizen doubly conscious of the material benefits to be obtained under the new health Scheme, and of his eagerness to participate in and to exploit to the full all that could be obtained under this heading, before the source dried up and because it was free (so-called free). The stock-piling of the medicine chests of this country began in earnest without any regard to cost or necessity, and has continued to do so without loss of tempo.

As a result of the unsatisfactory and unsettled state of his remuneration, and its implications, the public soon realized the ease with which the general practitioner could be held to ransom by his patients. The fact that no penalties or regulations of any kind were in existence to discipline the patient or to curb his appetite, soon became apparent, and placed the general practitioner in the stocks. The tide of goodwill and respect was rapidly on its ebb, and soon a situation arose which placed him and his branch of the profession in no enviable position, and there it remains to-day.

The rise in the number of certificates, record keeping, and other clerical duties,

and of their necessity to keep the administrative machine moving, presented a problem as irksome as it was unexpected.

I now come to the last cause:

### THE EMERGENCE OF THE ADMINISTRATIVE MACHINE.

I have already mentioned how the entrance of the administrative machine into the lives of the individual has played its part in placing the general practitioner in eclipse. We have been described by many and sundry under different appellations. Much lip service has been paid to us in the past, but none with more skill than the heads of the different Ministries both here and in England. A wave of distrust invariably crosses my mind when I hear or read of their platitudes. We have been referred to as (1) The keystone of the edifice; (2) The linch-pin of the profession; (3) The backbone of the profession; (4) The scavenger of the profession; (5) The cinderella of the profession: The latter by the late unlamented Minister of Health in England, Mr. Aneuran Bevan, whose obstinacy, subtlety, and hatred of us as a class will ever remain a monument of inverse class distinction at its worst, the effect of which may jeopardise for a lifetime the more harmonious association between the Ministry of Health and our profession, and may even send many of us with sorrow to the grave. Those seeds of distrust, so liberally scattered in the early days of our negotations, have an uncommon habit of sprouting up in the most unexpected places and times since the appointed day. It is not to be wondered at the attitude of the permanent official toward us, and who may be a genial and kindly person off duty, he may be even polite to a turn, but well schooled in the art of negotation and one tempered and moulded by a chief such as that described above. The official and his entry into our work and day-to-day practice of medicine is, I believe, permanent. It has come as a shock to most of us, and some perhaps are reeling from the interference and attitude of this individual towards them. From his point of view it is "The Act and nothing but the Act" that matters, and woe betide the one who should stray from the path of rectitude according to the Act and regulations. For us penal clauses and regulations are in operation for our guidance and annoyance. Truly a new adventure for us, but one which helps to distract from the practice of medicine, and marks the entry of fear into our daily lives, giving us a different slant to every patient who enters our consulting rooms.

Much remains to be done in this field and it is the duty of our representatives, both professional and political, to work towards this end by every means in their power to bring about a better understanding between these two bodies and to educate and enlighten the law makers and administrators of the difficulties that bestrew the general practitioner's path. No hard and fast rules can ever replace the general practitioners common sense to act in the interest of his patient, whose welfare must ever remain his foremost consideration and concern.

A book entitled, "General Practice and the Training of the General Practitioner," was published by the Association in 1950, and it is evident from a perusal that the entry of the administrative machine into the field of medicine is exercising the

minds of those best able to gauge its implications and how it affects the general practitioner. A quotation from it warrants repetition and further study:

"The Outcome is that the range and scope of modern General Practice and the place of General Practice in the Perspective of Medicine as a whole have become dimmed. At the present time when far reaching changes are being made in the Structure of Medical Practice and when vital decisions are being taken affecting the livelihood and professional standing of the general practitioners it is of the utmost importance to Medicine, to the Public, and to general practitioners that this obscurity should be removed."

The words of Kipling's "Tommy" come to mind, when he declares, "We aren't no thin red 'eroes and we aren't no blackguards too."

The summation of the parts played in the past by the factors now enumerated, has helped to form the morass in which the general practitioner finds himself to-day and to place him in almost total eclipse.

Finally you may ask:

- (1) Has a case been made out that the general practitioner is in eclipse?
- (2) What evidence exists in the profession as a whole and outside of it, that all is not well in this particular branch?
- (3) Are there any remedies for our ills?
- (1) As to the first: It is a question for you to answer; and if it is in the affirmative: What steps do you intend to take as an individual, to remedy this state of affairs? I leave this matter to your conscience and judgment.
- (2) Regarding the second: There is much evidence that general practice and the general practitioner has been exercising the minds of the thinking members of our profession and many others in authority outside it.

A committee sat to study "The Training of a Doctor," and was later followed by another committee under the chairmanship of Sir Henry Cohen, President of the Association, with terms "To continue the study of medical education by considering the post-graduate education of the general practitioner". Its findings were published last year. A study of these are of value to any of my colleagues who are interested in the general practitioner, and his relationship to the consultant and the specialist.

The Nuffield Trust organisation is at present carrying out along similar lines a pilot survey in Northern Ireland.

The latest committee was set up by Mr. Bevan before vacating the post of Minister of Health, and it is one which promises to outclass anything that has taken place before in its comprehensiveness.

And lastly, we have the fact that Branch Council of the N.I. Branch of B.M.A. has been in touch with the University authorities regarding the possibility of forming a lectureship in the General Practice of Medicine.

The fact that these different bodies have been formed renders it obvious that the value of the general practitioner in the National Health Service has been underestimated, a gross miscalculation has occurred. A new value must be placed on the general practitioner with full statutory backing. His authority and

self-respect must be restored to him, if he is to play his full part in the medical branch of the social services of the State.

I make bold to express my personal view that such successes as have attended the National Health Service to date despite the assertions of the planners and politicians, are in large measure due to the great traditions of the past, which have supplied a momentum, whose effect carried this Service through a mælstrom, with rocks and reefs, on which it could easily have foundered. The answer to this question rests on the facts.

### ARE THERE ANY REMEDIES FOR OUR ILLS?

It is not possible to go into more detail in an address of this kind. Any of the headings under which I have subdivided this paper could be used as the subject matter for a lecture or debate in itself. But some general remarks will not be out of place.

Of the remedial measures that I consider necessary and urgent, that of the settlement of our remuneration must be placed in the forefront. I cannot think of any more delectable transfusion for the general practitioner at present. He, more than any other class, must have freedom from want and worry to enable him to give of his best and to cope efficiently with the work that he is asked to perform.

A revision of his terms of service as they exist to-day, for they require drastic pruning and re-moulding, and a more liberal interpretation of them by officials. The penalties must be standardized and commensurate with those in other walks of life. Surely here is a case for the appointment of a committee of consultants and general practitioners, acceptable to the Ministry and Profession, to re-draft our terms of service and so remove many of the anomalies that exist. Who, bar a general practitioner is asked and compelled by law, to give a twenty-four hour service, seven days a week, 365 days a year, to answer the call of anyone, whether he or she is on his list or not. Even the sanctity of our homes can be invaded at will at any hour of the day or night. No barriers exist to protect him from the insatiable appetite of the public for medical attention. No prayers have been heard on his behalf in Parliament, or from any political party, to nullify their terms of service.

If such a state of affairs is allowed to go unremedied I would advise my brethren in his branch of the profession to accept as a battle-cry the words of Byron in "The Gladiator," "Arise, ye Goths, and glut your ire."

Some may say that the answer lies in a full-time salaried service. With this view I am in entire disagreement, although I know that it appeals to many of my colleagues who may be attracted to it, by the shelter that it offers.

Others favour the establishment of health centres; here again I fail to agree. I can foresee such a project bristling with difficulties, both financial and administrative, perhaps it was for these very reasons that Mr. Bevan unostentatiously allowed this particular pipe-dream to vanish into thin air. My experience of visiting last year, in the company of Dr. Bleakley, a prototype health centre on the outskirts of London, allowed me no doubt that Mr. Bevan's ability

to implement this favourite feature of his "brave new world," left as much to be desired as did his efforts to supply the nation with houses.

Group-practice offers another alternative. Here I believe lies the answer to this vexed question. It is one worthy of thorough exploration and examination in all its aspects. It is one which seems to me to offer a fair chance of success, if we, as general practitioners, can get together and formulate a scheme, agreeable to ourselves, favourable to our patients, and having official sanction.

### On the Administrative Side.

I hesitate to attack or suggest remedies. A reorientation of the official outlook towards us, and our contribution to the National Health Service is of the first order. We must be trusted, and allowed a more liberal interpretation of our terms of service. We must be freed from much of the form filling certifications and unnecessary clerical work that attend our daily tasks. Unless it is intended to produce a new branch of medicine in the profession: The Medico-Clerical Branch, which I pray may never come to pass.

### OUR PATIENTS.

As to the remedy for the canker that has crept into the lives of the community, I leave it to others, with more time at their disposal, and better qualified than the general practitioner to evolve a more equitable outlook and sense of responsibility towards the new social services, and a prayer that a resurgence of that correctitude so characteristic of the Ulster man and woman in the past may take place in the near future. The general practitioner must never be inveigled into or allow himself to become "The scavenger of the profession." This job must be left to those responsible for creating the condition.

To the consultant and specialist, teacher and administrator I would say, It is as much your concern as ours, how we live, and move, and have our being. The general practitioner alone is the first to meet disease at its earliest appearance, and the responsibility is his to evaluate the early signs and symptoms of disease. He must ever remain an efficient filter of clinical material referred to your hospitals. Each in his own particular field must take his stand with those of us who are conscious of our heritage as general practitioners and of our desire for an efficient general practitioner service collateral with your desire for an efficient hospital service.

At his best, the general practitioner is, in his own field, in no way inferior to the leaders of any other branch of medicine; At his worst—he does no more harm.

"Of all the manifold problems besetting the medical profession to-day in its desire to provide the public with the best medical care, that of restoring the family physician to a position commensurate with his contribution to society, by improving standards of general practice, appears to be one of the most pressing." This extract from a leader in the "New England Medical Journal," is worthy of serious consideration.

And in "The Times" we read:-"There can be no substitute for the able family

doctor. He still holds in his hands the lives of his patients. No hospital or specialist service, however elaborate, can offset defective treatment in the home or surgery. If general practice is not raised to a new level of competence, some would say restored to its rightful place, the whole of British medicine will suffer.

In conclusion, let me say that I have sufficient faith left in me to hope that the general practitioner will rise again to his place and prominence in the profession and in society.

No one, and least of all the general practitioner, wishes his branch of medicine to remain static, and given the tools he will continue to do the job, and do it well.

To my colleagues in our branch of medicine, we might take to heart the lines of Miss Rosamund Praegar, in "Mr. Magee Hits Out":—

"Sure you're all as tied as you can be,
Afraid to stand on your own two feet.
Ready to follow the flock and bleat,
Taking a path when you know you'll rue it,
Seeing the right but afraid to do it.
You yourselves are fast in a yoke,
So allow no freedom to other folk.
Slaves to a house or slaves to a habit,
You scoop out a burrow just like a rabbit.
And in the burrow you sit and blink.
Which of you now can be said to think?
Slaves to your species! I tell you what.
I'll give you a bone to put in your pot.

When you've tucked your vanity up on the shelves And can swallow a joke at your own wee selves, When you've mastered Fear and Hate and Greed, Then you may claim to be free indeed. But till that Day, as far as I see, You're talking Blethers! said Mr. Magee."

Perhaps some Mr. Magee may say to me, just after tea: "You're talking blethers, my friend, J. B.

# The Value and Limitation of Electroencephalography

By J. H. D. MILLAR, M.D., M.R.C.P.(LOND.)

# Department of Neurology, Royal Victoria Hospital, Belfast

ELECTROENCEPHALOGRAPHY, as an aid to diagnosis in clinical neurology, has been in operation for the past twelve years or more. Hans Berger's classical researches in the electrical potentials of the human brain recorded from the scalp were published between 1929-1937. This work received little attention, probably for the reason that it was published in psychiatric journals which are not as a rule read by physiologists. Since Adrian and Matthews in Cambridge studied the normal rhythms in 1934-35, a vast amount of physiological and clinical research has been done. In 1935 Gibbs, Davis and Lennox described the electrical changes associated with epilepsy. Another important landmark was Walter's description of a method for the localisation of brain tumours.

Electroencephalography has been in use in the Royal Victoria Hospital since April, 1949, and more than a thousand records have been made on our machine, which is a four-channel machine manufactured by the Marconi Instruments Company. The number of channels or pairs of leads varies, with each type of machine from three to eight or more. The ideal number is probably about six, as any more than this number places too great a strain on the technician controlling the machine at the time of recording.

The head is covered at equal spaces with a number of electrodes, usually four longitudinal antero-posterior lines and three transverse lines of electrodes. Good contact is made by cleansing the skin with spirit and covering the electrodes with saline pads. The electrodes are held in position by a rubber harness similar to that used in ladies' hairdressing establishments. It may be remarked at this junction that electroencephalography is an entirely painless procedure and there is no risk to life. Recording is made by pens writing directly onto moving paper. Each pen is fed by one channel, and the record resulting is a measure of the changing potential differences between two electrodes amplified approximately two million times. This is called bipolar recording. In unipolar recording a common electrode is used, usually attached to the ear. In our machine, then, four channels simultaneously record from usually five electrodes arranged serially in a straight line. The connections from the electrodes are so arranged that in a series of three electrodes connected to two channels, should a discharge occur, near to the central or common electrode, then a "phase reversal" in the records of the two pens will be apparent. When this phase reversal is persistent in more than one plane, then a focus is present. A persistent focus in the E.E.G. is of great clinical importance. The usual routine record requires about half an hour to forty minutes to complete and a difficult record may require as long to interpret.

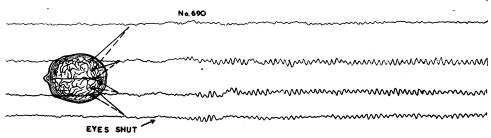


Fig. 1.—The record of a normal uniovular twin; their records are identical and show the abrupt appearance of the alpha rhythm with eye closure.

Some of you may have seen a record being made and have seen the normal rhythms. The normal record is difficult to define, but its chief characteristic is the alpha rhythm (fig. 1). This consists of sine waves of a frequency between 8-13 c/s., best seen in the parieto-occipital regions, and according to Adrian and Matthews is supposed to arise in the higher visual centres. Classically, it disappears when the subject attends, that is, on concentration—mental or visual. The routine stimulus is eye opening when the alpha rhythm disappears, to return dramatically with eye closure. The frequency, persistence, amplitude and spread of the alpha rhythm varies considerably and it more often than not can be analysed into more than one component, although the resulting form may appear to have but one frequency. Some people may have only a very occasional burst and in others it persists even with eyes open. It is least apparent in "visual thinkers" and in persons who have difficulty in relaxing and are easily distracted. A certain amount of other frequencies may appear in a normal record. At this point it might be as well to define some common terms used in electroencephalography. Waves with frequencies from 0.5 to 3.5 c/s. are called delta waves, from 4 to 7 c/s, theta, 8-13 c/s. alpha activity, and waves of faster frequencies are called beta or fast activity. It would be more correct to call each rhythm by its numerical frequency, but the above names are useful for more than one reason. The normal adult record may contain, in addition to the alpha rhythm, frontal fast activity at 22 c/s. of low amplitude compared to the alpha rhythm. Waves in the theta range at 6-7 c/s. are also commonly seen and regarded as normal, provided their amplitude is also small. The record in children is very different and alters with age; there is a slow maturation from birth to the middle "teens." The infant's record is dominated by slow delta waves of high amplitude, which tend to become more rhythmic. Between the ages of 2-7 theta activity becomes dominant, although about this time the alpha rhythm makes its appearance it is not usually dominant until the age of eight. After eight the amount of theta lessens until the adult record emerges in the early 'teens, although the record is more unstable than the adult record, reacting more readily to changes in the internal environment, such as hypoglycæmia and alkalosis. The E.E.G. maturity correlates more with emotional maturity than with physical or mental age. In fact, psychopaths in adult life continue to show an excess of theta activity, especially

the aggressive psychopath. Clarke and Taylor (1949) have shown that 70 to 80 per cent. of murderers have abnormal E.E.Gs. when the crime was not motivated. In interpreting a record, the various frequencies and their position of greatest amplitude are noted and the symmetry of the rhythms on the two sides are compared with regard to amplitude, persistence and spread. Focal abnormalities are sought and also specific epileptic wave forms. All this would be simple were it not for the inevitable presence of artefact. These artefacts are seen in even the most careful recordings, and may be due to factors in the environment such as electrical mains and passing trams, which may be reduced by careful screening, factors in the patient, such as muscle contraction potentials which simulate fast activity, pulse tracings simulating slow activity, sweating, coughing and movements of the head which change the electrode contacts; the eye is a dipole, and every eye movement causes a change in electrical field, which is readily picked up by the frontally placed electrodes. Other artefacts may be caused by the machine. In fact, the first thing to learn in E.E.G. interpretation is the characteristics of artefacts, and even the most experienced electroencephalographer is at the mercy of his recordist. It should be stated here that the E.E.G. has only a limited number of ways of presenting pathological states. These may be excess of slow or fast rhythms, the presence of focal activity, or the presence of poly-rhythmic wave forms called complexes such as are characteristically seen in epileptics.

### EPILEPSY.

Records taken during clinical attacks, especially major attacks, are such rare events that they are not of great importance; as a general rule petit mal attacks are accompanied by a bilateral outburst of wave-and-spike complexes and grand mal by high amplitude fast activity, preceded and followed by high amplitude slow activity. However, it is the recognition of the interseizure patterns which is the most important task in routine records, Classically, the hallmark of idiopathic epilepsy is the 3½ c/s. wave-and-spike complexes occurring synchronously in all channels (fig. 2). These complexes can occur without clinical manifestations when they are called larval seizures. They are not confined to the petit mal type of clinical seizure, but can occur in the interseizure records of patients suffering from major epilepsy without apparently petit mal attacks. The complexes are made up of many frequencies harmonically related, and the slow component frequency may vary from 2-4 c/s. The slower this component the worse the prognosis and the epilepsy is usually associated with mental backwardness; the faster the component the better the prognosis, and this type also responds better to therapy (fig. 3 b). When the complexes are clearer frontally the more likely are major fits to accompany the petit mal. When the complexes are however occipital, the form of epilepsy approaches that of a pyknolepsy. Dawson and Walter (1944), using an automatic analyser, found that the resting records of epileptics contained many frequencies which were harmonically related, and they showed that the complex wave-and-spike pattern could be synthesised when these frequencies were locked together and certain phase relationships obtained; namely, when the fundamental or slow component increases in amplitude and is out of phase with the other harmonics; the lock is very critical and exact. It is of interest to note that factors which can increase the amplitude of the slow frequencies also increase the occurrence of wave-and-spike outbursts, namely, hyperpnæa, sleep and hypoglycæmia. These workers too have been able to manufacture their complexes using an electrolytic model of the human head and various generators placed internally emitting the harmonically related frequencies.

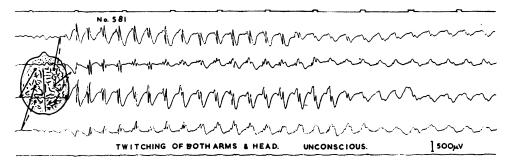


Fig. 2.—Wave-and-spike complexes occurring synchronously in all channels.

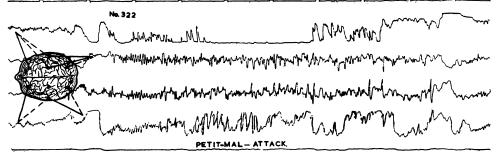
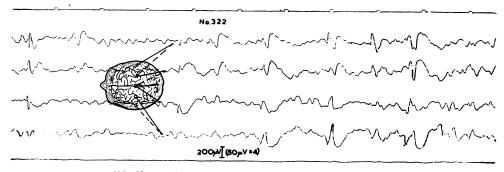


Fig. 3.—(a) "Petit mal" seizure in a deteriorated child. A more correct description would be a brief major attack.



(b) Slow spike-and-wave complexes in the same child.

The origin of the wave-and-spike complex has long been postulated to be diencephalic and it came as no great surprise when similar complexes were produced in the cat by electrical stimulation of the midline nuclei of the thalamus by Hunter and Jasper (1949); Williams (1949), using long electrodes, has shown that in man the wave-and-spike complexes appear in the thalamus before the cortex. In fact, many workers suggest that idiopathic epilepsy is due to a state of low threshold existing in the synapses of the reticular nuclei of the thalamus, and that the complexes spread to the cortex along the newly-discovered reticular projection system.

TABLE I.

The distribution of first 100 idiopathic epileptics.

| Age Group   |  | Numbe | ers | Norm | al | Abnor | mal | Abnor | mal | Suggest    | tive | Diagnostic |
|-------------|--|-------|-----|------|----|-------|-----|-------|-----|------------|------|------------|
| 0 9         |  | 27    |     | 6    |    | 1     |     | 2     |     | 12         |      | . 6        |
| 1039        |  | 66    |     | 12   |    | 16    |     | 10    | ••• | 18         |      | 10         |
| 40 and over |  | 7     |     | 3    |    | 3     |     | 1     |     | _          |      |            |
|             |  |       |     |      |    |       |     |       |     |            |      |            |
| Total       |  | 100   |     | 21   |    | 20    |     | 13    |     | <b>3</b> 0 |      | 16         |

The first hundred idiopathic epileptic records made in the Royal Victoria Hospital were studied. This group consisted of patients in whom it was possible to establish that there was no symptomatic cause for their complaint and who had been followed up clinically for at least one year. The results are shown in Table I, and are very similar to those obtained in other centres. The group suggestive of idiopathic epilepsy contains records where there were paroxysmal outbursts of activity of higher amplitude than normally seen or where complexes suggestive of, but not identical to the wave-and-spike pattern were present. The "abnormal" group records showed excess of slow and/or fast activity which was not paroxysmal in nature. The "mildly abnormal" group mainly includes records where it was difficult to decide whether they belonged to the "normal" or "abnormal" groups. It should be stated here that 10-15 per cent. of the "normal" population have mildly abnormal or abnormal E.E.Gs. In view of the large number of epileptics who have normal or mildly abnormal E.E.Gs. various methods of activation are used.

- 1. Hyperpnæa: Three minutes hyperpnæa is used as a routine procedure.
- 2. Photic stimulation: It is possible to evoke cortical responses by a bright light flashed into the eyes, and in this way to supply the necessary harmonic in the manner described above. The most successful results are obtained with flashes between 10 and 20 per second. Walter, using a special circuit, has been able to produce diagnostic features in 30 per cent. of epileptics (fig. 4).

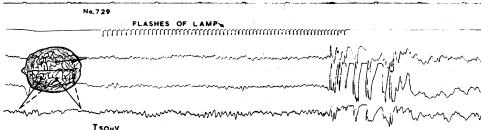


Fig. 4.—Outburst of epileptic complexes with photic stimulation.

- 3. Cardiazol activation: Cardiazol intravenously in subconvulsant doses is also used, and Mr. Luke and myself have tried this method in a number of cases which presented diagnostic difficulties. When we had studied 11 normal control and 39 neuropsychiatric patients, we came to the conclusion that considerable caution was required in interpreting the results, in view of the responses in our series of normal controls. Cardiazol was of great value, however, if it activated a focus and the focal clinical signs.
- 4. Sleep: Sleep has been used since Hippocratic days as an aid in the diagnosis of epilepsy. Narcotic drugs are given in association with the E.E.G.

All methods of activation especially cardiazol are particularly useful in showing focal lesions causing epilepsy.

When epilepsy has a focal origin it is sometimes possible to localise the focus in the E.E.G. on the principle of phase reversal, described earlier. Epilepsy can, of course, be caused by gross lesions, such as tumours, but I wish to discuss foci where the lesions are small, such as cortical scars and atrophic gyri, following birth injuries and other accidents. Here the E.E.G. picture is one of focal complexes, sharp waves or spikes arising from the damaged tissue near the lesion (fig. 5). It must always be remembered that scars, abscesses tumours, etc., are electrically inert, and that it is the partially damaged cells nearby which give rise to the abnormal waves. It is of interest that the whole cortex may take on epileptic features secondary to a focal epileptic lesion. This may explain why fits may continue after a focus has been surgically excised, although the frequency of the fits may be reduced. When a focus is excised it is not sufficient to remove the scar only, but also the partially damaged cells surrounding it which, in fact, cause the epilepsy. Also the sooner the focus is excised the less chance is there for the epileptic tendency to spread over the cortex. Recently, the whole cortex of one hemisphere has been excised where the E.E.G. has shown that it is much more abnormal than its fellow. Following the operation, the fits have been reduced in number and severity, and the E.E.G. of the remaining hemisphere shows a return to normal. This severe operation is undertaken rarely and only when the epilepsy is severe and uncontrolled by other methods. When the hemisphere is severely damaged, as in cases of porencephaly following severe hæmorrhage or birth trauma, there is little further neurological deficit. In fact Krynauw (1951), in a series of twelve cases of infantile hemiplegia, actually obtained improvement in motor and

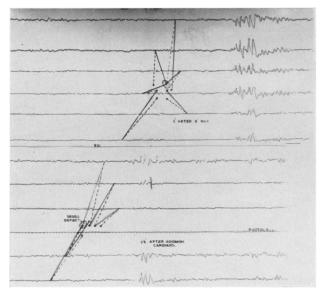


Fig. 5

Sharp waves in a man aged 23, focal in the right frontal region. There was a five year history of epilepsy and a bone defect in the right frontal region present from birth.

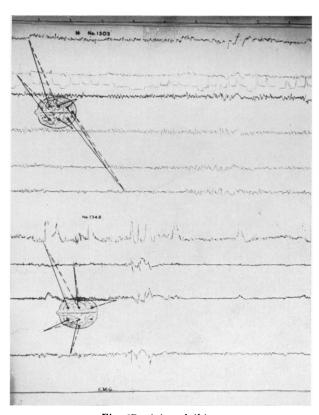


Fig. 17—(a) and (b)
The records of two sisters suffering from Familial Myoclonic Epilepsy.

lessening of spasticity and clumsiness as well as marked improvement in mentality, character and behaviour. Ten of the twelve cases also suffered from epilepsy and have had no further attacks since the operation.

### PATHOLOGICAL LESIONS (see Table II).

The E.E.G. changes in organic lesions of the cerebrum have in general certain features in common. The more rapid the progression of the lesion the more abnormal the record, the nearer the lesion is to the cortex, the slower is the dominant frequency, and lesions near the third ventricle show an excess of theta activity. Subtentorial lesions cause no change unless there is an increase in the intracranial pressure, when diffuse slow activity appears but without a persistent focus.

### INTRACRANIAL SPACE-OCCUPYING LESIONS.

Of thirty-two supra-tentorial tumours, twenty-six were correctly localised as proven by surgical or pathological confirmation (fig. 6). In two cases the E.E.G. was normal; one was a small and slowly growing astrocytoma, the other was a parasaggital meningioma, which are notoriously difficult to localise. I had previously examined this patient, and the negative E.E.G. was for me confirmatory evidence that there was a parasaggital meningioma. In another the first record was incorrectly interpreted, as the anterior slow activity was considered to be due to eye movements. Another tumour was correctly lateralised, but the wrong lobe indicated. In three other cases the tumour was incorrectly localised, in two it was considered deep-seated and central when it was lateralised, and the third vice versa. In these cases associated cerebral cedema extended far beyond the confines of the tumours and led to the misinterpretation. Williams has shown that dehydration by hypertonic intravenous solutions has uncovered a focus where only generalised abnormality existed previously. The E.E.G., therefore, can be a valuable aid in the localisation of brain tumours, especially where the record is read against the background of good clinical notes. A normal record does not however eliminate the possibility of a slow-growing tumour which causes little distortion of the surrounding normal cells. The E.E.G. not infrequently gave the

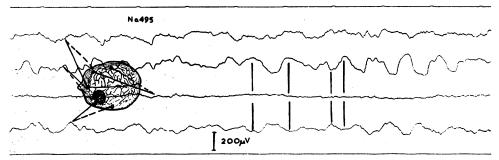


Fig. 6.—A case of cerebral tumour in the left frontal region showing the presence of slow waves over the tumour and the phase reversal about a common electrode.

first positive evidence of a tumour. False localisation arises mainly as a result of cerebral ædema and excess of artefact. Attempts have been made in the U.S.A. to diagnose the type of tumour from the E.E.G. I cannot agree that this is possible, and feel satisfied if I have localised a lesion correctly. No evidence of a supratentorial lesion was reported in ten subtentorial tumours; these as a rule show no abnormality except when the intracranial pressure is increased.

Four out of six supratentorial acute abscesses were localised correctly. One was missed on account of artefact. The other case was interesting, a diffuse suppurative encephalitis without a definite encapsulated abscess. Here, although pus was found surgically, the slow activity from the abnormal hemisphere swamped any localised abnormality.

Eleven cases of possible supratentorial tumours and five cases of possible abscesses, including two cases of otitic hydrocephalus were referred and correctly reported to have no focal lesion.

Records of two cases of tuberculoma showed correct localisation.

### VASCULAR LESIONS.

Localised vascular lesions, such as thrombosis and hæmorrhages, behave as space occupying lesions, if however the lesion resolves, the focus also disappears; hence the importance in doubtful cases of doing serial records.

Benign hypertension causes little change, but rapid changes may occur in malignant hypertension, when cerebral cedema probably accounts for the E.E.G. abnormality.

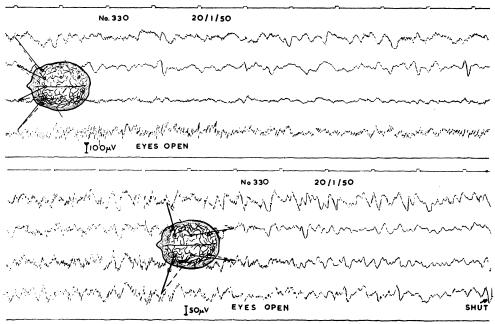
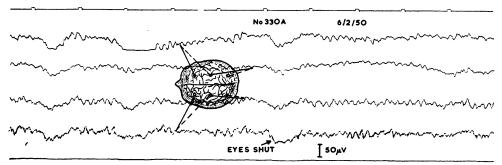


Fig. 7.—A case of subarachnoid hæmorrhage.
(a) and (b)—Showing the diffuse slow activity on 20.1.50.



(c.)—A further record 6.2.50 showing the return to normal with clinical recovery.

Subarachnoid hæmorrhage in the acute states gives rise, in my opinion, to a characteristic picture not unlike that of an acute meningitis (fig. 7). In addition, it is possible to detect a phase reversal in frontal region, and where confirmed in one case by angiography, this focus was on the side of the aneurysm. The E.E.G. abnormality again resolves as the clinical state improves. The E.E.G. is useful, however, if a persistent abnormality remains, as this would indicate local activity such as a hæmatoma. A large berry aneurysm was undetected by the E.E.G. this was not unexpected, as its expansion was very slow and intermittent. Two subdural hæmatomas were not localised by the E.E.G., although both records were abnormal. The E.E.G. in subdural hæmatoma presents many difficulties. If the hæmatoma is small and localised it may present the same E.E.G. picture as that of a space occupying lesion, but if it is extensive it may insulate the abnormal brain waves and the more normal hemisphere appears to be the abnormal side. However, when considered against the dramatic clinical findings, the diappointing record may suggest the correct diagnosis. The same remarks hold good for extradural lesions, vascular and infective.

### PRESENILE DEMENTIA.

The usual record is one of diffuse abnormality, the more rapid the progression the more marked the abnormality. Localised slow activity may occur in the early stages. In the slowly progressive or static cases a "slowed alpha" or dominant rhythm about 7-8 c/s. is a frequent finding. I have gained the impression that

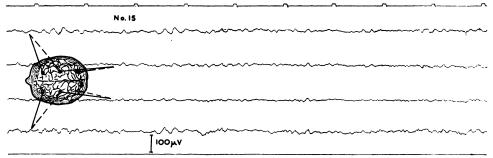


Fig. 8.—A case of presentle dementia showing diffuse theta activity.

excess fast activity in an elderly person may be an early sign of presenile changes (fig. 8).

### DISSEMINATED SCLEROSIS.

It is rare to find any marked abnormality, but it has been suggested that an abnormal E.E.G. might be used as evidence in a doubtful case for multiplicity of lesions.

No particular features are noted in cases of Parkinsonism or motor neurone disease. Cases of G.P.I. and meningiovascular syphilis have similar records to other cases of presenile dementia.

### ENCEPHALOPATHY.

In one case of post T.A.B. encephalopathy, clinically presenting with pyrexia, drowsiness, vomiting and papilledema, the E.E.G. showed diffuse delta and theta

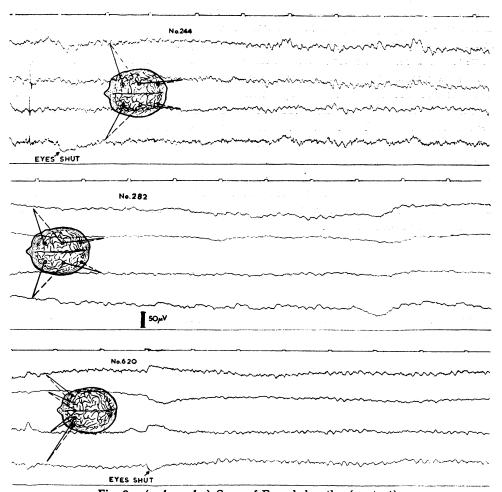


Fig. 9.—(a, b, and c) Cases of Encephalopathy (see text).

at the outset (fig 9a). The E.E.G. improved, but was still mildly abnormal two months after the onset and the patient still showed slight abnormalities on examination.

Two cases of acute encephalopathy presented very similar physical signs; in one, the condition had come on spontaneously in a man of 26 (fig. 9b), in the other,

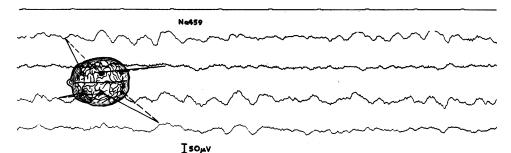


Fig. 10.—The record of a man aged 33 suffering from tuberculosis meningitis.

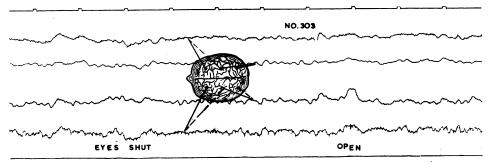


Fig. 11.—Record of a man aged 41 suffering from congenital cystic kidneys.

B.P. 190/130. Blood urea 400 mgms. per cent. Semi-comatose and left-sided hemiparesis.

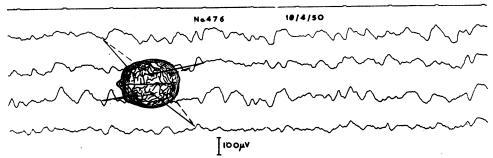


Fig. 12.—A man aged 39 admitted to hospital in coma with a history of head injury ten days previously. No subdural clots found at operation.

Later, it transpired that he had been vomiting for some time. (E.E.G. done before operation).

(a) Alkali reserve 67.2 cc. per cent.; Blood chlorides 158 mgms. per cent.; Blood urea 148 mgms. per cent.

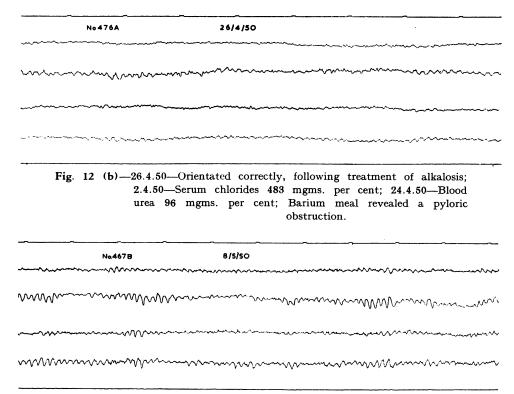


Fig. 12 (c)—Normal record and complete clinical recovery.

a man of 36 (fig. 9c), the onset followed two days after an injection of protein. Both had marked temperatures, were emotionally labile and dysarthric, and showed marked nystagmus, cerebellar signs in the upper limbs and minimal pyramidal signs. Both E.E.Gs. returned to normal in about six months, and were accompanied by nearly complete clinical recovery. The original E.E.Gs. presented very similar features, being dominated by theta activity which was mainly frontal.

Six cases of acute encephalitis showed varying degrees of abnormality. In four cases of tuberculous meningitis the E.E.G. was very abnormal, much more so than in the cases of encephalitis (fig. 10). In the four cases of uræmia the abnormality was not proportional to the height of the blood urea, but varied rather with rate of change of the toxic state and level of consciousness (fig. 11). For instance, in a case of chronic nephritis with a high blood urea the abnormality was only slight. In a coma state, due to alkalosis and dehydration following persistent vomiting, the E.E.G. was very abnormal, but rapidly returned to normal with the blood chemistry (fig. 12).

### HEAD INJURIES.

The abnormality is usually proportional to the severity of the injury and is localised when the lesion is circumscribed (fig. 13). Again the abnormality

resolves as the patient recovers; if however, the patient has residual signs and symptoms when the E.E.G. has returned to normal, it is unlikely that further organic recovery will take place; serial records are thus useful from a prognostic point of view. It may also be possible to predict the onset of post-traumatic epilepsy. These epileptic changes may be generalised or of a focal nature. In the latter type it may be possible to eradicate the focus surgically (fig. 5).

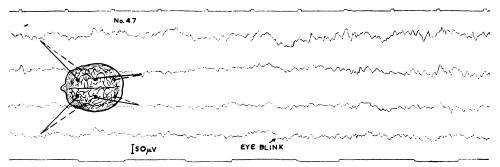


Fig. 13.—The record of a woman aged 47 suffering from a severe head injury who died five days later, and the post-mortem showed diffuse softening of the brain.

### MISCELLANEOUS CASES.

I should now like to mention a few cases of particular interest. The E.E.G. in patients suffering from Huntington's chorea presents a striking absence of the spontaneous rhythms (fig. 14). This was first observed by Hill, and it has received no satisfactory explanation. The pathological lesion is diffuse and it is impossible to incriminate any particular cortical area or cerebral pathway.

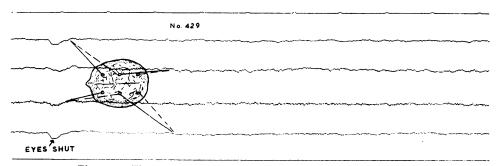


Fig. 14.—The record of a case of Huntington's chorea showing the absence of rhythmic activity.

We were fortunate to obtain the E.E.G. of a patient in whom cardiac arrest of forty minutes' duration had occurred during thoracotomy. He was treated in a respirator after the operation and spontaneous respiration commenced ten hours after the operation although consciousness was never regained, and he died thirty

hours later. An E.E.G. was done twenty hours after the operation, at the request of the anæsthetist. The record was grossly abnormal, showing diffuse slow waves at 2-4 c/s. (fig. 15).

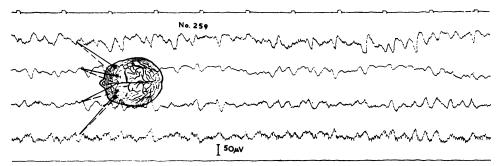


Fig. 15.—Record of a man suffering from anoxia following cardiac arrest (see text).

Another case of particular interest was a boy, aged 11, suffering from subacute encephalitis, in whom the E.E.G. presented the features which Hill and Cobb consider diagnostic of Van Bogaert's encephalitis or Dawson's "inclusion cell" encephalitis, on the basis of five cases (fig. 16). The clinical history is one of progressive dementia leading to mutism, deafness, blindness and loss of consciousness and death in usually six months. Characteristically, there are brief episodic involuntary movements lasting one second at intervals of eight to twelve seconds, accompanied by high amplitude slow discharges in the E.E.G. This boy had a similar history. The involuntary movements were lightning-like twitches or myoclonic flexor jerks involving head, trunk and limbs, occurring about every twenty seconds. The C.S.F. showed a marked paretic curve as described in the cases reported. When I first saw him there was little on neurological examination except for mild Parkinsonian features, but slowly he deteriorated and passed through a dystonic phase when his arms took up attitudes of "frozen athetosis." There was a marked "striatal" quality to his clinical picture. The post-mortem findings were those of a subacute encephalitis, but there was no evidence of the two pathological conditions which Hill and Cobb found in their cases.

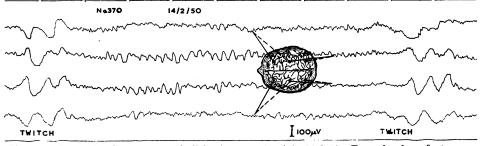


Fig. 16—Case of subacute encephalitis (see text). (a) 14.2.50—Record when first seen.

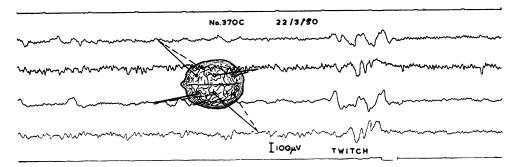
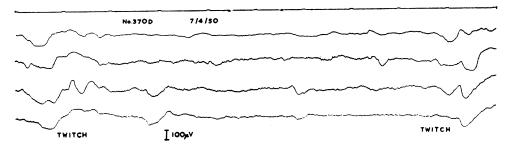


Fig. 16 (b)—22.3.50.



(c) 7.4.50—A few days before the patient died.

Another patient of considerable interest was a woman, aged 34, suffering from Unverricht's Familial Myoclonic Epilepsy. She gave a history of jerking movements for seven years, two major epileptic fits associated with pregnancy and increasing difficulty in speaking. On examination, there were frequent episodes of myoclonic jerks involving mainly the arms, and the musculature of articulation; the arm movements bore a close resemblance to chorea, and her speech was markedly dysarthric. Of her five sisters and one brother, two sisters were similarly affected and examined by me, one being bed-ridden. E.E.G. records were done in the patient and one sister and showed similar changes to those described by Grinker et al. (1938). These consist of bilateral short outbursts of epileptic complexes associated with the myoclonic jerks (fig. 17). The patient had previously been regarded as neurotic but since she has been treated as an epileptic there has been considerable improvement; the myoclonic jerks in her limbs have largely disappeared, although her speech is still slightly affected. Eventually mental deterioration will set in and progress. One sister is mentally deteriorated.

Finally, of fourteen patients who were diagnosed as suffering from functional headaches, the E.E.G. was mainly normal, at the most mildly abnormal. A high proportion of these cases showed an excess of muscle artefact as a result of contractions in the temporal muscles.

### SUMMARY.

The E.E.G. is of value in about 50 per cent. of epileptics, and gives on occasion invaluable information not obtainable by other methods. It is useful in brain tumour localization when the lesion is supra-tentorial and progressive. It should localise all supra-tentorial acute abscesses, and is useful in the differential diagnosis between supra-tentorial and subtentorial space occupying lesions. In cases who have been treated for abscesses it is useful in the follow-up period in the detection of residual abscesses and the prediction of secondary epilepsy. Diffuse or localised changes occur in head injuries, toxic states and diffuse progressive "encephalopathies."

I would like to express my thanks to Dr. R. S. Allison for his advice and encouragement, and to Mr. R. J. Luke and Mr. T. R. Hewish for their helpful assistance.

### TABLE II.

| Diagnosis      | N    | umber |           | Normal |   | Mildly<br>onormal | A     | bnormal      | Very<br>Abnorm | Focus  |
|----------------|------|-------|-----------|--------|---|-------------------|-------|--------------|----------------|--------|
| Cerebral       |      |       |           |        |   |                   |       |              |                |        |
| Thrombosis     | ٠    | 6     |           |        |   | 1                 |       | 5            | <br>           | <br>4  |
| Hypertension   |      | 8     |           | 4      |   | 3                 |       | 1            | <br>           | <br>   |
| Malignant      |      |       |           |        |   |                   |       |              |                |        |
| Hypertension   |      | 2     |           |        |   |                   |       | 2            | <br>           | <br>   |
| Subarachnoid   |      |       |           |        |   |                   |       |              |                |        |
| Hæmorrhage     |      | 6     | . <b></b> |        |   | <del></del>       |       | 3            | <br>3          | <br>4  |
| Presenile      |      |       |           |        |   |                   |       |              |                |        |
| Dementia       |      | 9     |           |        |   | 2                 | • • • | 7            | <br>           | <br>   |
| Disseminated   |      |       |           |        |   |                   |       |              |                |        |
| Sclerosis      |      | 8     |           | 2      |   | 5                 |       | 1            | <br>           | <br>   |
| Encephalopathy | •••• | 3     |           |        |   |                   |       | 3            |                | <br>   |
| Encephalitis   | •••  | 6     |           |        |   | 2                 |       | 4            | <br>-          | <br>   |
| Tuberculous    |      |       |           |        |   |                   |       |              |                |        |
| Meningitis     |      | 4     | •         |        | • |                   |       | . <b>2</b> . | <br>2          | <br>?1 |
| Toxæmias       |      | 5     |           | _      |   | 2                 |       | 3            | <br>           | <br>   |
| Functional     |      |       | -         |        |   |                   |       |              |                |        |
| Headaches      |      | 14    |           | 9      |   | 5                 |       |              | <br>           | <br>   |

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# **REVIEW**

# A HISTORY OF ENGLISH PUBLIC HEALTH. By W. M. Frazer. Baillière, Tindall and Cox. Pp. 498. Price 35s.

This is the first large history of the development of the Public Health Services in England in the nineteenth century. There have in the past been numerous small studies, mostly prepared for lectures and several lighter general surveys of the subject, but until now no one has attempted to present a proper co-ordinated study. The work involved must have been immense, and it is fortunate indeed that Professor Frazer, whose experience and gifts render him so suitable a person, has found time to make such a thorough study. It will be of immense value, not only to students of public health, but to all social historians of the period.

Perhaps the only criticism which occurs to the reviewer is that the material selected for study and the deductions drawn from it are so eminently conventional. Perhaps in an endeavour to be fair in all respects the author has not allowed his own opinions to appear sufficiently often. The reader will, for example, search in vain for clues as to the reasons for the failure of the existing public health services to survive unscathed the impact of the changes of the last twenty years. Perhaps one clue can be read between the lines; how few names of medical officers of health mentioned as contributors to new knowledge appear in the latter half of the book.

Such criticism, which is, after all, but opinion, cannot detract from the great merits of this book—its accuracy, its collection of information never before assembled, and its pleasant, clear presentation.

A. C. S.

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# Hyperchloræmic Acidosis with Nephrocalcinosis

By Joan B. T. Logan, m.d., m.r.c.p.

Registrar in Pædiatrics, Royal Belfast Hospital for Sick Children

THE syndrome of hyperchloræmic acidosis with nephrocalcinosis was first described by Dr. Reginald Lightwood at a meeting of the British Pædiatric Association at Newcastle, Co. Down, in May, 1935. He described the finding at post-mortem in six infants of heavy deposits of insoluble calcium salts in the renal tubules. The six cases occurred in a consecutive series of 850 autopsies. This may indicate a higher incidence than is correct. These children have a prolonged illness and there is a tendency for cases where the diagnosis remains obscure to gravitate to Great Ormond Street. Renal acidosis of this type is relatively rare.

Working backwards from these autopsy findings of renal calcification, Dr. Lightwood made a study of the clinical records and found certain features common to all cases. Anorexia, constipation and failure to thrive were present in all and obstinate vomiting in three. On examination, the principal findings were wasting and muscular hypotonia. Sometimes the urine contained a little albumen, a few leucocytes and bacteria but there was no general improvement after treatment of this mild and inconstant infection. The immediate cause of death was infection. There was no evidence of other disease, the feeding had been correct and vitamin D dosage not excessive.

In September, 1936, Dr. Lightwood took the subject further by describing a similar case diagnosed during life with a reduced alkali reserve persisting over six months and a persistently alkline urine containing a trace of albumen, occasional white cells and streptococci on culture.

Since then numerous case reports have been published often of single cases with a few small series from the larger medical centres. Albright and his fellowworkers in the United States have studied extensively the bone changes in adolescent and middle-aged persons showing the same biochemical changes. From these reports the following picture emerges.

In children the onset may be in the first few weeks after birth or more frequently at any time during the first year. Less often it has been reported during childhood, at puberty or at any age in adult life. In adults the prominent feature is usually the bony changes secondary to the effects on calcium metabolism. The development of these bony changes can often be traced back many years—even up to twenty—suggesting that the onset is most often if not always in early life.

The symptoms in infants are anorexia, vomiting which is variable in persistence and severity, but often sufficiently severe to produce recurrent episodes of dehydration, constipation, hypotonia, failure to thrive and polyuria. Added to this are renal rickets and dwarfism in older children, while asthenia and osteomalacia

with its bone pains and deformity are the presenting features in adults. Any of these symptoms may be absent or any combination present. This presents a diagnostic problem, for there are obviously few of the maladies of infancy that may not be simulated.

The urine is persistently alkaline or at best weakly acid. It is usually of low specific gravity with a trace of albumen and a few white cells. Mild and transient urinary infections are common. They may clear up without treatment and their presence seems to be unconnected with the main picture.

Examination of the blood biochemistry shows a persistently low CO<sub>2</sub> combining power, raised serum chlorides, a normal blood urea when the infant is not dehydrated, a normal or low serum calcium, a low phosphorus and in some cases a raised alkaline phosphatase.

At post-mortem small bristle like deposits of calcium can be seen naked eye as radial white lines in the renal medulla most evident in the pyramids with relatively normal cortices, calyces and ureters. Histologically, the calcium is seen to be between the basement membrane of the tubule and the lining epithelium. The collecting tubules are most often involved. Changes are also described in the proximal convoluted tubules. Here there are swollen epithelial cells containing vacuoles of various shapes and occasional deposits of calcium are seen in proximal and distal convoluted tubules. There are no signs of pyelonephritis or chronic nephritis, no metastatic calcification and no other significant abnormalities.

X-rays of infants during life may show this diffuse calcification. If not, X-ray of the kidneys at post-mortem may show it. In the older child and the adult with this syndrome the presence of calcium deposits or multiple renal stones is almost invariable.

It is easy to understand that this condition results from failure of the tubular cells to carry out the selective reabsorption of water and other substances from the glomerular filtrate, on which the renal control of the pH of the blood depends. That the glomeruli are normal or only minimally affected is supported by the usually minor degree of albuminuria, the normal blood urea in the absence of dehydration and the usual only slight impairment of clearance tests.

It is more difficult to determine the site and nature of the tubular defect. It has been widely accepted, possibly incorrectly, that the fault lies in the distal tubule, because it is the site of the production of ammonia and the acidification of the urine. Failure of these two processes could explain the retention of the acid radicles in the blood and the simultaneous excretion of an alkaline urine with the loss of fixed base including calcium occurring as a compensatory measure in the removal of the excess acid radicles. Greenspan has suggested that the basic fault is the inhibition of carbonic anhydrase, an enzyme which controls the exchange of H-ions for base across the tubular cell. Possibly a toxic reaction to suphonamides may be one of the factors causing this inhibition. Certainly these cases do not tolerate sulphonamides well. Recent work by Latner and Burnard hinges on the high urinary bicarbonate which suggested to them a failure to absorb bicarbonate

in the proximal tubule; which, passing on down to the distal tubule, increases the absorption of chlorides and upsets the process of acidification. By perfusing the kidneys of children showing the accepted clinical and biochemical findings of this syndrome with phosphate solutions, they showed that the tubular cells could produce normal amounts of free acid and ammonia once the excess of bicarbonate over phosphate was reversed.

Just as the pathology of the condition is not fully understood the natural history of the disease is not fully known. Some cases die during an episode of dehydration, some of secondary infection to which they are specially prone if kept in hospital over a long period. Some cases die of a severe toxic reaction to one of the suphonamides usually suphathiazole. Some cases recover after an illness of many months. Some evidently recover only partially and after months or years show the effects of this continued expenditure of fixed base to cover the excretion of acid radicles.

In this condition of tubular insufficiency without glomerular insufficiency the urinary calcium and potassium are both raised. The calcium precipitation in the tubule is believed to be a consequence of the unusually high concentration in a tabular fluid of higher pH than normal and the cellular damage caused by the deposited calcium is believed to explain the polyuria and low specific gravity.

Loss of calcium in the urine lowers the serum calcium level which stimulates the parathyroid gland to overaction. Hyperparathyroidism results in increased excretion of phosphorus which lowers the serum phosphorus level. This is the factor which causes the rickets of the growing child and the osteomalacia of the adult for there is a reciprocal relationship between serum phosphorus and serum calcium and the low serum phosphorus causes an elevation of the serum calcium to normal at the expense of the body's reserves of calcium in the bones. Increased activity of the osteoblasts causes an increase in the serum alkaline phosphatase before changes can be seen on X-ray. Osteomalacia develops insidiously with its attendant train of bone pains and deformities with pseudofractures and generalised decalcification on X-ray. Another factor of importance here is that gastric acidity is decreased in acidotic states, which would tend to decrease the calcium absorbed from the gastro-intestinal tract.

Loss of potassium in the urine gives rise to clinical signs less often than loss of calcium, but Albright reports several cases who showed paralysis of the limbs which lasted for several days at a time and usually cleared spontaneously though at least one death may have been attributable to potassium lack.

The aims in treatment are to replace the base lost in the urine and to combat the acidosis. This can best be done by giving the salt of a mineral base and an organic acid, e.g., sodium citrate or if hypopotassiumæmia is a factor, a combination of sodium and potassium citrate should be used. An organic acid, e.g., citric acid can be given also to increase gastric acidity and therefore the amount of calcium absorbed. The organic acid will largely be burned after absorption. The usual mixture given consists of 140 grams of citric acid and 98 grams of sodium citrate

in a litre of water, given in doses of 60—120 c.c. per day. This will cure the acidosis, restore the blood chlorides to near normal and raise the CO<sub>2</sub> combining power, but large doses of vitamin D and adequate amounts of dietary calcium must be ensured to replace the expended stores of calcium in the cases of osteomalacia. Once the osteomalacia is cured, the vitamin D dosage should be reduced, provided alkali therapy is preventing further wastage in the urine. Normal growth will be restored in those children whose epiphyses are not yet united.

Though this type of acidosis is rare, it should be borne in mind as a possible diagnosis for the infant who vomits persistently or simply fails to thrive, for the ailing child with recurrent mild urinary infections or for the case which simulates atypical and unresponsive Pink Disease. In all such cases the blood biochemistry and the urine should be examined to exclude hyperchloræmic acidosis.

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### **REVIEW**

ILLUSTRATIONS OF BANDAGING AND FIRST-AID. By Lois Oakes, S.R.N., D.N. Fourth Edition. Pp. 308. Edinburgh: E. & S. Livingstone Ltd. 8s. 6d.

This should prove a particularly useful book for the student who requires to master the art of bandaging. The instructions are clearly and concisely given, and on the opposite page the illustrations show each step of the application. The lay-out is excellent. The First-Aid Section only covers, unfortunately, Shock, Hæmorrhage, and Fractures. One would have liked further chapters on Principles of First-Aid, Burns, etc. The new chapter on Application of Elastoplast is also exceptionally well illustrated and should be a valuable addition.

E. M.

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# Evaluation of the Treatment of Disseminated Sclerosis with Arsenicals

By J. H. D. MILLAR, M.D., M.R.C.P.(LONDON). Department of Neurology, Royal Victoria Hospital, Belfast

THE evaluation of treatment of disseminated sclerosis where long, natural remissions occur is difficult. Allison (1950) found that in a series of 40 cases first seen in 1929, 12 had survived when revisited in 1949. The longest duration of the disease amongst the survivors was 39 years, and 5 had had the complaint for over 30 years, the average being 27-28 years. These cases had had no arsenical treatment. This emphasizes the extreme variability of the course of the disease, and shows that the prognosis is often better than is usually realized. During a recent investigation into the status of 91 patients who had been diagnosed for

|           |          |       |          |       | Cas       |         | ABLI<br>Freat |         |      |       |              |       |               |       |                         |     |              |
|-----------|----------|-------|----------|-------|-----------|---------|---------------|---------|------|-------|--------------|-------|---------------|-------|-------------------------|-----|--------------|
|           | Case No. |       | Sex      |       | Onset Age |         | N.A.B.        |         | Time |       | 1. Full Work |       | 2. Light Work |       | 3. Not Yet<br>Bedridden |     | 4. Bedridden |
|           | 3        |       | F        |       | 23        |         | 48            |         | 1    |       | 0            |       | 5             |       | 0                       |     | 10           |
|           | 9        |       | F        |       | 23        |         | 128           |         | 0    |       | 12           |       | 0             |       | 0                       |     | 0            |
|           | 20       |       | F        |       | 20        |         | $\bf 24$      |         | 1    |       | 17           |       | 0             |       | 0                       |     | 0            |
|           | 23       |       | F        |       | 36        |         | 48            |         | 1    |       | 0            |       | 16            |       | 0                       |     | 0            |
|           | 27*      |       | F        |       | <b>20</b> |         | 24            |         | 18   |       | 18           |       | 0             |       | 1                       |     | 11           |
|           | 25       |       | F        |       | <b>29</b> |         | 32            |         | 2    |       | 2            |       | 12            |       | <b>2</b>                |     | 7            |
|           | 29       |       | M        |       | 28        | • • •   | 74            | • • • • | 1    |       | 1            |       | 0             |       | 14                      |     | 0            |
|           | 35       |       | M        |       | <b>27</b> |         | 60            |         | 17   |       | 22           |       | 0             | • • • | 5                       |     | 0            |
|           | 39       |       | M        |       | 32        |         | 24            |         | 4    |       | 10           |       | 10            |       | 0                       |     | 0            |
|           | 47       | • • • | F        | • • • | 26        | • • • • | <b>2</b> 8    |         | 2    | • • • | 1            | • • • | 0             | • • • | 18                      | ••• | 0            |
| Totals    | 10       | •••   | F7<br>M3 | •••   | 264       |         | 490           |         | 47   |       | 83           |       | 43            |       | 40                      | ••• | 28           |
| Averages  |          |       |          | 5     | 26.4      |         | 49            |         | 4.7  |       | 8.3          |       | 4.3           |       | 4.0                     |     | 2.8          |
| Average T | `ОТАІ    | Dı    | IRATI    | ION   |           |         |               |         |      | 19.   | 4            |       |               |       |                         |     |              |

N.A.B. column: Total number of injections.

Time Column: Interval to nearest year between onset and beginning of treatment.

at least ten years, the opportunity was taken to compare cases which had been treated with intravenous arsenicals, Neoarsphenamine and Silver Salvarsan, with a series of control cases (Millar, 1949).

Only those patients who had received more than three courses of eight injections in the early stages of the disease were considered to have had the minimum treatment likely to affect the course of such a disease process. Ten cases only fulfilled these requirements. The ten control cases were chosen as those with as nearly as possible in this series, a similar type and age of onset. These ten control cases had received only a few injections.

The course of the disease in each patient was divided, to the nearest year, into four stages:—

- 1. Able to carry on gainful employment.
- 2. Able to do light work.
- 3. Unable to work, but able to walk a little and look after themselves with assistance.
- 4. Bedridden.

|           |           |         |          |     |           | Co    | NTRO   | LS  |      |              |       |               |         |                         |         |             |
|-----------|-----------|---------|----------|-----|-----------|-------|--------|-----|------|--------------|-------|---------------|---------|-------------------------|---------|-------------|
|           | Case No.  |         | Sex      |     | Onset Age |       | N.A.B. |     | Time | 1. Full Work |       | 2. Light Work |         | 3. Not Yet<br>Bedridden |         | A Rodriddon |
|           | 40        |         | M        |     | 12        |       | 0      |     | 0    | 32           |       | 0             |         | 0                       |         |             |
|           | 1         |         | M        | ••• | 18        |       | 6      |     | 6    | 0            |       | 0             |         | 17                      |         |             |
|           | 10        |         | M        |     | 30        |       | 4      |     | 10   | 10           |       | 0             | • • • • | 12                      |         |             |
|           | <b>32</b> |         | F        |     | 37        |       | 6      |     | 6    | 6            |       | 5             |         | 0                       | • • • • |             |
|           | <b>26</b> |         | M        |     | 18        |       | 8      |     | 7    | 8            |       | 0             |         | 0                       |         | 1           |
|           | 36*       | • • • • | M        |     | 17        |       | 0      |     | 0    | 9            |       | 9             |         | 0                       |         |             |
|           | <b>2</b>  |         | F        |     | 41        |       | 6      |     | 3    | 3            |       | 0             | •••     | 0                       |         | 1           |
|           | 8         |         | F        |     | 15        |       | 6      |     | 16   | 9            |       | 0             |         | 16                      |         |             |
|           | 21        | • • •   | F        |     | 29        |       | 0      |     | 0    | 3            |       | 7             | •••     | 5                       |         |             |
|           | 45        | • • •   | M        | ••• | 28        | • • • | 15     |     | 13   | 10           | •••   | 0             |         | 1                       | •••     | 1           |
| Totals    | 10        |         | F4<br>M6 |     | 245       | •••   | 51     | ••• | 61   | 90           | • • • | 21            | •••     | 51                      | •••     | 5           |
| Averages  |           |         |          | 5   | 24.5      |       | 5.1    |     | 6.1  | 9.0          |       | 2.1           |         | 5.1                     |         | 5.          |
| Average 7 | Готаі     | . Dī    | URATI    | ON  |           |       |        |     |      | 21.9         |       |               |         |                         |         |             |

<sup>\*</sup>Cases 27 and 36. The long interval between onset and beginning of treatment is explained by a long initial symptomless remission.

Controls: Chosen as those cases with little treatment and as similar a type and age of onset as possible.

This sub-division made comparison easier, but it must be remembered that different occupations would influence the first two stages.

The two groups, treated and controls, are compared in the table. This method of comparison, unfortunately, does not fulfil the requirements of a statistical approach and would probably not be considered valid by a purist. However, I plead my case on several points. Firstly, the long period of observation; and secondly, the similarity of the treated and control groups clinically. Little more can be expected from a clinical evaluation. The figures suggest that treatment with arsenicals has little effect on the course of the disease. This confirms the present-day opinion of most neurologists.

#### DISCUSSION.

There is no known treatment of disseminated sclerosis which is generally accepted to influence favourably the course of the disease. Fashions in treatment come and go, and arsenicals have enjoyed a long régime and are still in favour in some clinics. In such a long-standing condition, it is only possible to evaluate treatment after many years. The literature contains only accounts of short-term therapy, general impressions, statements of faith and accounts of the treatment of one or two cases. The basis for the use of arsenicals in disseminated sclerosis was founded on the erroneous idea that it was spirochætal in ætiology (Siemerling, 1918). Many authors have supported the use of intravenous arsenicals (Adams et al., 1924; Kalberlah, 1919; Osnato, 1928; Perrin, 1920; Prados and Such, 1922; Sauer, 1926; Schafsen, 1924; Stern-Piper, 1920). Others have either found inconclusive results or decided against the use of arsenicals. (Fleck, 1921; Simmonds, 1920; Speer, 1920; Veraguth, 1924; Wichura, 1920).

However, no attempt, as far as the author is aware, has been made to analyse the end-results after a number of years or compare them against a control series. It was for this reason considered that present investigations were worth while despite the fact that arsenical treatment is not accepted at present in this department.

### CONCLUSIONS.

An attempt has been made to compare the course of the disease in ten patients suffering from disseminated sclerosis treated with intravenous arsenicals, with that of ten similar controls. No significant difference in clinical status could be seen about ten years later.

If the results of this investigation are generally true, it does not seem justifiable to submit patients to the real risk of toxic complications and the possibility of syringe jaundice.

I wish to thank the physicians of the Royal Victoria Hospital, Belfast, for permission to use their cases, and Dr. R. S. Allison for his help and encouragement.

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### **REVIEW**

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# Post-Partum Eclampsia

By William S. Campbell, B.SC., M.B., B.CH., F.R.C.S.(ENG.)., M.R.C.O.G., L.M.(ROTUNDA).

Obstetric Surgeon, Belfast City Hospital; Gynzcologist, Samaritan Hospital, Belfast

#### Introduction

THE subject of post-partum eclampsia is of importance in (i) consideration of the theories of origin of eclampsia in general, and of the so-called placental origin theories in particular; and (ii) the management and methods of delivery of cases of pre-eclamptic toxæmia. This communication is a study of fifty-three cases of post-partum eclampsia in which the maternal and fœtal mortality, the time interval between delivery and the occurrence of fits, and the nature and duration of the previous labour were investigated in detail.

### GENERAL CONSIDERATIONS.

Eclampsia is a disease occurring only in pregnant or recently pregnant women, and consequently the products of conception must play some part in its causation. Older workers considered that the disease was a toxæmia, the toxin of which originated in the fœtus and placenta. Eclampsia, however, can occur with hydatidiform mole where the fœtus is absent, and this clinical fact has been taken to indicate that the toxin arises in the placenta and not the fœtus. The subject of the relation between eclampsia and hydatidiform mole has recently been reviewed by Chesley et al., who found in the literature thirty-five cases of mole in which fits occurred, apart from many cases showing pre-eclamptic toxæmia. Toxæmia appeared to occur in cases with more than four months amenorrhœa, or where the uterus reached above the level of the umbilicus.

It appears to be generally accepted that viable placental tissue must be present for pre-eclamptic toxæmia to develop, but, while death of the fœtus in utero usually results in improvement of the mother in pre-eclampsia and eclampsia, this is not invariably so. Hirsch has recently described a case of eclampsia at the twenty-sixth week four days after death of the fœtus in utero. Zondek, however, has shown that if the fœtus dies in utero the placenta may sometimes continue to function normally, and remain in communication with the maternal circulation for as long as four weeks.

Most modern theories of the origin of eclampsia come in the end to involve the placenta. Many workers believe that circulatory disturbances of the uterus and placenta are an important ætiological factor, and cite as evidence the fact that eclampsia is commoner in those women in whom the uterus is tightly stretched about its contents—twins, hydramnios and primigravidæ—or where the patient is in labour or has essential hypertension. Page considers that as a working

hypothesis the placenta may be regarded as behaving like the kidney, i.e., uterine ischæmia may cause the liberation of a pressor substance from the placenta. Actual placental lesions peculiar to pre-eclampsia and eclampsia have been described by Bartholomew and Colvin in America, Young in England and Falkiner and Apthorp in Ireland, but there is as yet no general agreement on these.

One of the main arguments used against the placental origin theories is the occurrence of post-partum eclampsia, particularly the so-called late cases. Nevertheless, generally speaking, rapid improvement follows delivery in cases of ante-partum and intra-partum type. Further, it should be remembered that the placenta has both a maternal and foetal origin, that the maternal decidua remains after birth, that the Zondek-Aschheim reaction normally remains positive for eight days after expulsion of the placenta, and that it may remain positive longer if chorionic fragments remain in the uterus in communication with the maternal circulation.

#### MATERIAL.

The records of two hundred and fifty-three cases of eclampsia were examined. These cases constituted those admitted to the Royal Maternity Hospital, Belfast, during the period 1932-1950, and to the Jubilee Maternity Hospital, Belfast, during 1939-1950. Fifty-three cases of post-partum eclampsia (i.e., where fits occurred for the first time in the puerperium) were found, and seventeen cases which had post-partum fits, in addition to fits before delivery.

### MATERNAL AND FŒTAL MORTALITY.

The maternal mortality in post-partum eclampsia varies considerably in different published series of cases. Most writers consider the post-partum type, properly treated, as being the least serious. Eden, however, in 1922, found, in investigating two thousand and five cases of eclampsia from various parts of the British Isles, that the mortality in post-partum cases was 27.6 per cent. compared with 20.5 per cent. in ante-partum cases and 16.6 per cent. in intra-partum cases. Earlier mortality figures published for post-partum eclampsia are 33 per cent. by Johnstone and 87 per cent. by Fletcher Shaw.

|                   |                |   | IABLE   | L.          |                        |          |                             |             |
|-------------------|----------------|---|---------|-------------|------------------------|----------|-----------------------------|-------------|
| Туре              | Numbe          | r | Per Cen | Mat<br>Numb | Mortality<br>Per Cent. |          | etal Mortality<br>Per Cent. |             |
| Post-Partum       | <br>53         |   | 21      |             | 5                      | <br>9.43 |                             | 4.92        |
| Ante-Partum or    |                |   |         |             |                        |          |                             |             |
| Intra-Partum plus | <br>17         |   | 8.5     |             | 5                      | <br>29.4 | • • •                       | 29.4        |
| Post-Partum       |                |   |         |             |                        |          |                             |             |
| Ante-Partum and   |                |   |         |             |                        |          |                             |             |
| Intra-Partum      | <br>200        |   | 79      |             | 19                     | <br>9.50 |                             | 44.5        |
|                   |                |   |         |             |                        |          |                             | <del></del> |
| Total             | <br><b>253</b> |   |         |             | 24                     | <br>9.48 |                             | 35.4        |

Table I shows the maternal and fœtal mortality in the writer's series. The

uncorrected fœtal mortality includes both still-births and neo-natal deaths. Five maternal deaths occurred among the post-partum cases, i.e., a maternal mortality of 9.43 per cent., compared with 9.50 per cent. for cases occurring before delivery, and 9.48 per cent. for the series as a whole. In intra-partum cases of eclampsia where fits persisted after delivery, Eden found that the maternal mortality was little if at all affected. In the writer's series, however, it was greatly increased (29.4 per cent.) and this is what one would expect, as persistence of fits is probably one of the most gloomy prognostic signs.

It is possible that in the earlier series mentioned cases other than true post-partum eclampsia have been included (the differential diagnosis is considered later), and this possible error applies particularly to cases of post-partum eclampsia occurring after forty-eight hours. In this connection, it is interesting to note that Olshausen (quoted by Johnstone) at the beginning of this century believed that in post-partum eclampsia, if the fits occurred within a few hours of delivery, they were few in number and the prognosis was good, but if they occurred at a later period in the puerperium, the fits were more numerous and the prognosis bad. From a study of the writer's series it appears that although clinically many cases of post-partum eclampsia are assessed as mild, a few severe ones (with anuria, cerebral hæmorrhage, etc.) occur, resulting in the maternal mortality being about the same as the overall mortality for all types of eclampsia.

The fœtal mortality in the writer's series of post-partum eclampsia cases was 4.92 per cent., compared with a fœtal mortality of 44.5 per cent. in cases of eclampsia occurring before delivery. This illustrates the lethal effect of fits on the fœtus, but it must be pointed out, in addition, that the post-partum cases of eclampsia in this series, as will be seen later, occurred mostly in women in whom the pregnancy had advanced beyond thirty-six weeks. It can be concluded that the fœtal mortality in post-partum eclampsia is probably of the same order as that in severe pre-eclamptic toxæmia. The above considerations illustrate forcibly how in pre-eclamptic toxæmia the prognosis for both mother and child becomes infinitely worse if fits supervene, and how all our resources should be directed towards preventing their occurrence.

### AGE, PARITY AND DURATION OF PREGNANCY.

#### TABLE II.

|                  |           |       |       |          | Average |          |          |            |
|------------------|-----------|-------|-------|----------|---------|----------|----------|------------|
|                  |           |       |       | Α        | ge      | under    | over     | number     |
|                  | No.       | Prim. | Mult. | under 30 | over 30 | 36 weeks | 36 weeks | of fits    |
| Post-Partum      | <b>53</b> | 38    | 15    | 41       | 12      | 5        | 48       | 3.0        |
| Ante-Partum      |           |       |       |          |         |          |          |            |
| or Intra-Partum  |           |       |       |          |         |          |          |            |
| plus Post-Partum | 17        | 12    | 5     | 13       | 4       | 0        | 17       | <b>2.6</b> |

Table II shows further details of the series. Post-partum eclampsia most commonly occurs in young primigravidæ, and, in this series, the pregnancy had

usually advanced beyond thirty-six weeks. Lennon believes that the younger the patient and the less parous, the later in pregnancy is the onset of eclampsia and the milder its severity.

#### TIME BETWEEN DELIVERY AND THE OCCURRENCE OF FITS.

### TABLE III.

| Hours after delivery |  | Number | Per cent. of Total | Sum of per cent. |
|----------------------|--|--------|--------------------|------------------|
| 0 6                  |  | 36     | <br>67.9           | <br>67.9         |
| 612                  |  | 15     | <br>28.3           | <br>96.2         |
| 12—24                |  | 1      | <br>1.9            | <br>98.1         |
| 24-30                |  | 1      | <br>1.9            | <br>100.0        |

Table III shows the time after delivery when the first fit occurred, and it will be seen that 98 per cent. of cases of post-partum eclampsia occurred in the first twenty-four hours of the puerperium, and by far the largest number of these in the first twelve. Hence it appears that the eclamptic process as a general rule comes to an end within twenty-four hours of delivery of the placenta. Clinical verification of this comes from the fact that diuresis in cases of eclampsia does not usually become established until twenty-four hours after delivery.

### DIFFERENTIAL DIAGNOSIS.

Fits occurring for the first time over forty-eight hours from delivery are usually regarded as late cases of eclampsia. The criteria on which a diagnosis of eclampsia is based must be carefully scrutinized in all cases of post-partum fits, and especially the so-called late eclampsia cases. Apart from post-mortem evidence in fatal cases, diagnosis is usually made by excluding other causes of convulsions and coma, and by relying on certain clinical signs and symptomshistory of pre-eclamptic toxemia in the preceding pregnancy or labour, and the presence of hypertension, cedema, albuminuria, oliguria and retinal changes with complete return to normal at a later date apart from hypertension. Stander, in addition, emphasises certain biochemical changes-elevated blood uric acid, carbon dioxide combining power and decreased clearance with complete return to normal. Many of the so-called late cases of eclampsia will be found to be due to hysteria, epilepsy, meningitis, uræmia and malignant hypertension. Purdon Martin and Sheehan (1941) have described the syndrome of primary thrombosis of the cerebral veins following childbirth, and some previously unexplained cases may fall into this category. Sheehan regards the existence of late post-partum eclampsia as doubtful, as most of the signs and symptoms on which it is diagnosed are seen in cerebral venous thrombosis.

As described by Purdon Martin and Sheehan the salient features of the condition are as follow. It occurs usually between four and twenty days after confinement. Usually the delivery has been normal and the puerperium uneventful. The first symptom is usually fits, which may be preceded by headache or intermittent cramp and weakness of a limb. The fits may continue over several

hours or days, and following them there is usually some involvement of the motor cortex—paralysis of one limb and weakness of the rest of that side (the limb is usually the arm). The cerebro-spinal fluid is normal, there may be slight pyrexia, slight albuminuria and mild hypertension which usually clears up in a few days. Hence it will be seen that the condition should usually be differentiated from true eclampsia without difficulty. In this series in no case was any evidence found, such as paralysis, which would suggest a primary cerebral thrombosis, but two probable cases have occurred recently at the Jubilee Maternity Hospital, Belfast. I would stress the diagnostic significance of marked albuminuria following a true eclamptic fit and usually preceding it. If this is not present the diagnosis should be very carefully reviewed. In this connection what is called eclampsia reflectorica should be mentioned, i.e., where after a severe labour, or during the last part of the second stage, the patient has a single convulsion or a second milder one, with or without very slight albuminuria. This condition has been said to be due to heightened irritability of the nerve centres, or to excessively strong stimuli from the uterus. De Lee has likened it to the discharge of an electric accumulator, toxins being massed during labour in a specially predisposed person. It is doubtful if these are cases of true eclampsia. In our series there were four cases that could be placed in this category.

# PERSISTENCE OF FITS AFTER DELIVERY. TABLE IV.

| Hours after delivery |  | Number | P   | er cent. of Total | Sum of per cent. |
|----------------------|--|--------|-----|-------------------|------------------|
| 0— 6                 |  | 13     |     | 76.4              | <br>76.4         |
| 6—12                 |  | 1      | ••• | 5.9               | <br>82.3         |
| 12—24                |  | 1      |     | 5.9               | <br>88.2         |
| 24-48                |  | 1      |     | 5.9               | <br>94.1         |
| 48 plus              |  | 1      |     | 5.9               | <br>100.0        |

Table IV shows a similar analysis of the seventeen cases in which post-partum fits occurred following ante or intra-partum eclampsia. Again it will be seen that the great majority occurred in the first twenty-four hours. One case, however, had what appeared to be a second attack of eclampsia one hundred and nine hours following delivery. I believe this to be a true case of late post-partum eclampsia, and, in view of its interest, will report it in more detail.

Mrs. M. McW., aged 27, primigravida, was admitted to the Jubilee Maternity Hospital, Belfast, on 5th June, 1948, as an emergency. The last menstrual period was on 2nd September, 1947, and the expected date of delivery 12th June, 1948. Apart from scarlet fever, measles and appendicitis, there were no previous illnesses and no family history of epilepsy or hypertension. Her father died of pleurisy aged 38. Her mother and four sisters were alive and well. She was married two years, and her menstrual cycle was regular, four weekly, with a normal loss and no dysmenorrhæa. The menarche was at 16 years of age.

On admission, she complained of swelling of the ankles for one month and occasional headaches. On examination, there was gross ædema of the face, hands, abdominal wall and legs, the blood pressure was 135/90 and the urine loaded with albumen. At twelve midnight on the day of admission rupture of the forewaters was performed under anæsthesia (C.E. mixture), and she was put on a modified Stroganoff's régime. The cervix admitted one finger, and the head was engaged in the R.O.A. position. Labour began fifteen hours later, and the first stage lasted seventeen hours. Ten minutes after the start of the second stage the patient has an eclamptic fit, and she was delivered forthwith under anæsthesia (C.E. mixture) by forceps of a living male child. The third stage was normal.

|         |     |     |                   |    |                 |         | TABLE V |             |                                     |
|---------|-----|-----|-------------------|----|-----------------|---------|---------|-------------|-------------------------------------|
|         |     |     | Intake            |    | Outpu           | t       |         | Highest     | •                                   |
| Date    |     |     | ozs.              |    | ozs.            |         | Albumen | B.P.        | Fits                                |
| 6/6/48  |     |     | 17 .              |    | $14\frac{1}{2}$ | • • • • | +++     | <br>165/100 |                                     |
| 7/6/48  |     |     | <b>17</b> .       |    | $18\frac{3}{4}$ |         | +++     | <br>158/95  | One at 08.10 hours                  |
| 8/6/48  |     |     | $52\frac{1}{2}$ . |    | 148             |         | ++      | <br>155/100 |                                     |
| 9/6/48  |     |     | <b>56</b> .       |    | 114             |         | ++      | <br>155/100 |                                     |
| 10/6/48 |     |     | <b>47</b> .       |    | 105             |         | +       | <br>190/100 |                                     |
| 11/6/48 |     |     | <b>2</b> 8 .      |    | 17              |         | +++     | <br>210/110 | One at 22.30 hours                  |
| 12/6/48 | ••• | ••• | $24\frac{1}{4}$ . | •• | $36\frac{1}{2}$ | •••     | 1.25    | <br>155/110 | One at 00.15, 00.45 and 06.35 hours |
| 13/6/48 |     |     | $33\frac{1}{2}$ . |    | 37              |         | 0.75    | <br>150/90  |                                     |
| 14/6/48 |     |     | <b>47</b> .       |    | 47              |         | Trace   | <br>148/90  |                                     |
| 15/6/48 |     |     | <b>48</b> .       |    | <b>7</b> 9      |         | Trace   | <br>155/100 |                                     |
| 16/6/48 |     |     | <b>54</b> .       |    | 70              |         | Trace   | <br>136/90  |                                     |

Her subsequent progress is shown in Table V.

A diuresis occurred and the ædema and albuminuria decreased. On 12th June, 1948, at 20.00 hours, she complained of severe occipital headache and dimness of vision for the past two hours. On examination, ædema was marked, all the reflexes were present, and the fundi showed papillædema. Stroganoff's treatment (morphia and chloral hydrate) was instituted at 20.15 hours, but at 22.30 hours, i.e., one hundred and nine hours after delivery, she had an eclamptic fit. The morphia was repeated and ten c.cs. of 10 per cent. magnesium sulphate given intravenously. After three further fits the condition started to improve, and she progressed uneventfully till discharged. The following investigations were carried out—blood urea, lumbar puncture, urea concentration, urea clearance and Wassermann reaction—and all results were within normal limits. There was at no time any paralysis or paresis. Six weeks later the blood pressure was 125/70 and the urine free from albumen.

This appears to be a genuine case of late post-partum eclampsia, fulfilling the postulates of diagnosis given above, and its remarkable feature was that after

apparent recovery from the intra-partum eclampsia, the condition should recur as an apparently new attack. The placenta and membranes were complete at delivery, and there was no post-partum hæmorrhage at any time. Paramore has described a somewhat similar case except that the first attack was post-partum. Stander et al, have recently described three apparently true cases of late post-partum eclampsia occurring on the fourth, sixth and eighth days following confinement, and among recent literature other late cases of more doubtful authenticity have been described by Whapham and Hogg (sixteenth day) and by Winterton (twenty-sixth day). Hence it may be concluded that late post-partum eclampsia probably does occur although very rarely, and that future research may further reduce the number of cases. It is interesting to note that late post-partum eclampsia has been a subject of discussion for many years. Dienst (1902) regarded late post-partum cases as due to the products of involution of the uterus and decidua, while in cases occurring during the first twenty-four hours the cause, as in pregnancy, was from the placenta.

### RELATION TO PREVIOUS TOXÆMIA.

Next to be considered is the relation of post-partum eclampsia to previous toxemia during pregnancy and labour. In Stander's series of twenty-four cases of post-partum eclampsia, "one had ante-partum eclampsia, five had severe pre-eclamptic toxemia, five had ante-partum 'toxemia," while thirteen had non-toxemic pregnancies and labours," i.e., in more than half the cases post-partum eclampsia made its appearance without signs and symptoms indicative of toxemia during the ante- and intra-partum period. In our series, however, investigation showed the following figures.

TABLE VI.

|          |        |        | Нуре | ertensio | n A | lbumin | uria |       |   |      |     |       |
|----------|--------|--------|------|----------|-----|--------|------|-------|---|------|-----|-------|
| Place    | Total  | P.E.T. |      | only     |     | only   | C    | edema | n | None | Not | Known |
| Hospital | <br>34 | <br>25 |      | 2        |     | 3      |      | 0     |   | 4    |     | 0     |
| Home     | <br>19 | <br>7  |      | 1        |     | 0      |      | 1     |   | 0    |     | 10    |

It will be seen that in cases delivered in hospital all but four showed some evidence of toxæmia before the fits occurred. Of the twenty-five cases of pre-eclamptic toxæmia (i.e., a combination of hypertension, ædema and albuminuria), ten had labour induced surgically, and in three cases Cæsarean section was performed, the indication being the severity of the pre-eclamptic toxæmia. The four cases showing no evidence of toxæmia during pregnancy or labour are those previously referred to as being probably examples of eclampsia reflectorica, and hence possibly not true cases. In the series where delivery had occurred before admission to hospital, the case notes were very scanty and signs of toxæmia may have been overlooked, but in about half the cases some sign of toxæmia was mentioned. Of the eleven cases showing only mild toxæmic signs or none at all six were twin pregnancies.

### RELATION TO MODE OF DELIVERY AND DURATION OF LABOUR.

The relation of the occurrence of post-partum eclampsia to the type of delivery and the duration of labour is shown below:

|             |   |                                   |       |          | T | ABLE    | VII  |          |      |      |  |          |               |
|-------------|---|-----------------------------------|-------|----------|---|---------|------|----------|------|------|--|----------|---------------|
| Method of   |   |                                   |       |          | D | uration | of I | Labour   | in H | ours |  |          | Not           |
| Delivery    |   | Number 0-6 6-12 12-18 18-24 24-48 |       |          |   |         |      |          |      |      |  |          | Known         |
| Cæsarean    |   |                                   |       |          |   |         |      |          |      |      |  |          |               |
| Section     |   | 3                                 |       | <b>2</b> |   | 0       |      | 0        |      | 0    |  | 1        | <br>0         |
| Forceps     | ٠ | 7                                 |       | 1        |   | 0       |      | 4        |      | 0    |  | 0        | <br><b>2</b>  |
| Twins       |   | 8                                 |       | 1        |   | 2       |      | 0        |      | 3    |  | . 0      | <br><b>2</b>  |
| Breech      |   | <b>2</b>                          |       | 0        |   | 0       |      | 0        |      | 0.   |  | 0        | <br>2         |
| Face        |   | 1                                 |       | 0        |   | . 0     |      | 0        |      | 0    |  | 0        | <br>1         |
| Spontaneous | 5 |                                   |       |          |   |         |      |          |      |      |  |          |               |
| Vertex      |   | 25                                | • • • | 3        |   | 4       |      | <b>2</b> |      | 1    |  | <b>2</b> | <br>13        |
| Not Known   |   | 7                                 |       | 0        |   | 0       |      | 0        |      | 0    |  | 0        | <br>7         |
|             |   |                                   |       |          |   |         |      |          |      |      |  |          |               |
| Totals      |   | <b>53</b>                         |       | 7        |   | 6       |      | 6        |      | 4    |  | 3        | <br><b>27</b> |

The large number of Cæsarean sections and forceps deliveries is due to their having been indicated by the pre-existing toxæmia. The large number of twin pregnancies is noteworthy. Among the two hundred and fifty-three cases of eclampsia investigated ten cases (3.95 per cent.) of twin pregnancy were found. Of these, eight were cases of post-partum eclampsia, i.e., 15 per cent. of post-partum eclamptic cases occurred in twin pregnancies, compared with 1.0 per cent. in ante-partum cases.

The figures for the duration of labour do not yield information of much value. It is generally agreed that labour and eclampsia are closely linked. Eclampsia often precipitates labour and labour often precipitates eclampsia. Hence a long labour is undesirable in pre-eclamptic toxemia as intra-partum fits may be initiated, or in an established case of eclampsia be increased in number. On the other hand, if labour is short the acme of the eclamptic process may occur following delivery, (i.e. in the first few hours) before the expulsion of the placenta and resultant break in the ætiological chain has had time to become effective.

### CONCLUSIONS.

- 1. About 20 per cent. of cases of eclampsia originate in the puerperium.
- 2. Although clinically many cases appear mild, the maternal mortality is probably little if any less than for eclampsia occurring before delivery. The feetal mortality is however much less, and is probably of the same order as that for severe pre-eclamptic toxemia.
- 3. About 98 per cent. of cases occur in the first twenty-four hours, and the great majority in the first twelve. Many cases previously described as late post-partum eclampsia may have been examples of primary cerebral venous

- thrombosis, malignant hypertension, epilepsy, etc., True late post-partum cases do, however, occasionally occur.
- 4. Cases of eclampsia where the fits do not cease after delivery of the placenta carry an increased risk to the mother.
- 5. Probably all or nearly all true cases of post-partum eclampsia show evidence of toxæmia in the preceding pregnancy or labour.
- 6. Post-partum eclampsia appears to be a common form of eclampsia in twin pregnancies. The pre-eclamptic symptoms may be mild.
- 7. It is probable that the eclamptic process comes to an end as a general rule within twenty-four hours of delivery of the placenta.

My thanks are due to Mr. H. L. Hardy Greer, Professor C. H. G. Macafee and Mr. J. A. Price for access to records of many of the cases included in the series, and to Professor Macafee for his very helpful suggestions and constructive criticism during the preparation of this paper.

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# Leucotomy: A Follow-up of Sixty Cases

By C. B. ROBINSON, M.D., F.R.C.P.(I.), D.P.M.
Purdysburn Hospital, Belfast

A Neurosurgical Unit was opened at Purdysburn Hospital in October, 1948, to carry out pre-frontal leucotomy on suitable cases. By the end of June, 1950, 60 cases had been treated of whom 31 were from Purdysburn and 29 from County Mental Hospitals. While it is recognised that the time since operation is much too short to attempt to draw dogmatic conclusions, nevertheless a preliminary report on the results obtained might be of help in the selection of patients in the future and in the choice of technique to be used. In all sixty cases the blind lateral approach, as standardized by Freeman and Watts, was employed. This is carried out by means of trephine holes marked out by taking a point on the skin three centimetres behind the outer angle of the eye and six centimetres vertically above the zygoma. The maximum cut possible was made in each case at right angles to the parasagittal plane and as near to the tip of the anterior horn of the ventricle as possible. Bleeding was encountered in a number of cases but was eventually controlled.

The following table gives a summary of the types of patients dealt with and the effect of the operation on their mental condition:—

### TABLE I.

|                          |   |    | Total |     | Much   |              | 1   | ehavio<br>mprov<br>Mental | ed | Jnchan | nged |      |
|--------------------------|---|----|-------|-----|--------|--------------|-----|---------------------------|----|--------|------|------|
| Туре                     |   |    | No.   |     | Improv | Improv       |     | nchan                     | •  | or Wo  | -    | Died |
| Schizophrenia<br>Simple- |   |    |       |     | ,      | •            |     |                           | J  |        |      |      |
| Hebephrenia              | - | ٠. | 8     |     | 3      | <br>2        |     | 1                         |    | 2      |      | 0    |
| Schizophrenia            |   |    |       |     |        |              |     |                           |    |        |      |      |
| Katatonia                | - | -  | 18    |     | 4      | <br>3        |     | 5                         |    | 6      |      | 0    |
| Schizophrenia            |   |    |       |     | •      |              |     |                           |    |        |      |      |
| Paranoid                 | - | -  | .15   |     | 6      | <br>1        |     | 3                         |    | 5      |      | 0    |
| Paraphrenia              | - | -  | 7     |     | 0      | <br>3        |     | 2                         |    | 0      |      | 2    |
| Affective                |   |    |       |     |        |              |     |                           |    |        |      |      |
| Psychosis                | - | -  | 8     |     | 3      | <br><b>2</b> |     | 2                         |    | 0      |      | 1    |
| Paranoia                 | - | -  | 2     |     | 0      | <br>2        |     | 0                         |    | 0      |      | 0    |
| Obsessive-               |   |    |       |     |        |              |     |                           |    |        |      |      |
| Compulsive               | - | -  | 1     |     | 1      | <br>0        |     | 0                         |    | 0      |      | 0    |
| Others                   | - | -  | 1     |     | 0      | <br>0        |     | 1                         |    | 0      |      | 0    |
|                          |   |    |       |     |        |              |     | _                         |    |        |      |      |
| Totals                   | - | -  | 60    | ••• | 17     | <br>13       | ••• | 14                        |    | 13     |      | 3    |

75

Of the 60 patients treated 30 have mentally improved, 13 others show improved behaviour with unchanged mental state, and 13 are unchanged or worse mentally.

TABLE II
Shows the number discharged of the various types treated:—

| Type          |      | N | o. Treat | ed | Died | D   | ischarge | ed | Working | 1 | Re-admitted |
|---------------|------|---|----------|----|------|-----|----------|----|---------|---|-------------|
| Schizophrenia | ı    |   |          |    |      |     |          |    |         |   |             |
| Dementia Pra  | æcox | - | 41       |    | U    |     | 15       |    | 11      |   | 1           |
| Paraphrenia   | -    | - | 7        |    | 2    |     | O        |    | O       |   | 0 .         |
| Affective     |      |   |          |    |      |     |          |    |         |   |             |
| Psychosis     | -    | - | 8        |    | 1    |     | 3        |    | 2       |   | -1          |
| Obsessional   | -    | - | 1        |    | 0    |     | 1        |    | 0       |   | 0 -         |
| Paranoia      |      | - | 2        |    | 0    |     | 0        |    | 0       |   | 0           |
| Others        | -    | - | 1        |    | 0    | ••• | 0.       |    | 0       |   | 0           |
|               |      |   |          |    |      |     |          |    |         |   |             |
| Totals        | -    | - | 60       |    | 3    |     | 19       |    | 13      |   | 2           |

Nineteen patients have been discharged, of whom two required to be re-admitted but thirteen of the remainder are working. Their employment varies from a coppersmith in the shipyards to full household duties. One male patient has been accepted for the Army and has already served six months!

The vast majority of these cases were chronic mental hospital patients whose prognosis was poor and possibility of discharge nil. There were three deaths in the series giving a 5 per cent. mortality rate. Two patients died from post-operative pneumonia and one from blood clot in the aqueduct.

The patients were selected for operation by the recognised standard of the degree of tension, apprehension, fear and worry present irrespective of diagnosis.

A one-stage operation was carried out at the beginning of the series but with more experience it was found that such a radical procedure was much too severe for the older patients, and a two-stage operation is now performed where there is any arterial degeneration or where the physical condition is below standard. The oldest patient treated was a female of 68 years and profiting by previous experience, one side only was cut in the first instance giving rise to a transient hemiparesis. Her recovery was stormy and her eventual physical state so frail that completion of the operation was not attempted. Following operation, there was no change in her mental condition, although her behaviour improved, and she died eleven months after operation from myocardial degeneration due to arteriosclerosis.

A male of 62 years and a female of 58 years had both frontal lobes divided at the one operation, and both died from pneumonia following operation.

A female of 60 years had a one-stage operation, with a resultant transient hemiplegia. She required brain needling on two occasions, and eventually recovered after a very severe illness. In view of the extensive lesion produced by this technique, a two-stage operation appears to be essential for patients in the late fifties and over.

TABLE III

Sets out the cases according to the duration of illness showing the number improved and discharged for each group:—

| Duration   |   |    |          |      |            |              |            |     |            |
|------------|---|----|----------|------|------------|--------------|------------|-----|------------|
| of Illness |   | Un | der 2 Ye | ears | 2-5 Yrs.   | 6-10 Yrs.    | 11-14 Yrs. | Ove | er 15 Yrs. |
| Number     | - | -  | 5        |      | ${\bf 24}$ | <br>14       | <br>6      |     | 8          |
| Improved   | - | -  | 2        |      | 16         | <br>7        | <br>3      |     | 2          |
| Percentage | - | -  | 40%      |      | 66%        | <br>50%      | <br>50%    |     | 25%        |
| Discharged | - | -  | <b>2</b> |      | 13         | <br><b>2</b> | <br>1      |     | 1          |

It will be observed from this table that although approximately the same improvement resulted in all categories except that of over 15 years duration, yet 15 of the 19 discharges were those with a history under 5 years.

Investigation of family history and adequate exciting cause gave little information of value. However, in this series there were two females of approximately the same age who broke down while serving in the Forces with a diagnosis of paranoid type of schizophrenia. Both had a family history of the male parent being of unsound mind, both had similar treatment prior to operation and had a leucotomy carried out within two weeks of each other. One girl is clear mentally, actively employed in the sewing room, makes her own dresses and is a most useful worker. The other is leading a vegetative existence and has defective habits. It is suggested that the difference in the result may be due to a difference in the extent or the situation of the cut. By the employment of a direct vision technique it should be possible to correlate the extent and situation of the lesion with the eventual mental condition of the patient.

Without detailed testing there is no doubt that there is a deterioration in personality in the majority of the cases which is probably largely due to the amount of brain tissue sacrificed at operation. It is realised, however, that the outlook for these patients was very gloomy with little hope of discharge, yet seventeen have returned to the enjoyment of their homes and further twenty-six have found life more tolerable and have found pleasure in their existence where previously tension had ruled their lives. All chronic mental hospital patients exhibiting tension should be reviewed in the light of these results as the alternative to operation is to keep them under restraint.

Of sixty cases treated by prefrontal leucotomy, thirty have shown mental improvement, of whom nineteen were discharged and two subsequently re-admitted. Of those discharged, thirteen are fully employed and find pleasure in their home life.

Thirteen other patients have shown improvement in behaviour without mental change.

It is suggested that an open operation would enable closer correlation to be made between the extent of the lesion and the eventual mental state of the patient.

A further review of chronic mental hospital patients exhibiting tension should be carried out with a view to leucotomy.

# Recent Reverses in Immunisation

By W. J. McLeod, M.D., D.P.H. Municipal Health Department, Belfast

DIPHTHERIA immunisation has been criticised recently on two counts. First, as one type of trauma to muscle which may influence the site of paralysis in persons incubating poliomyelitis; and second, on its failure to restrict epidemic spread of the gravis strain of diphtheria bacillus.

Evidence of an association between the site of a previous inoculation and the site of paralysis in poliomyelitis is contained in reports from Melbourne (McCloskey), and London (Geffen), and in the statistical survey of certain areas in England by Hill and Knowelden. These reports state that children inoculated within four weeks prior to the onset of poliomyelitis have developed paralysis of the arms more often than non-inoculated children. This finding has not been confirmed in Cardiff or in Belfast. A report of the 1950 epidemic in Belfast, in which the non-inoculated children show an unusually high proportion of arm paralysis, has been published in the British Medical Journal. Geffen, in London, calculates the risk of association, site of paralysis with site of inoculation, using the combined diphtheria-pertussis antigen to be 1 in 1,800 inoculations, while, in Belfast, using a pure diphtheria toxoid (P.T.A.P.), we failed to find any such association in 6,250 inoculations. This difference may be due to the use of different antigens.

The responsibility of advising the cessation of immunisation during an epidemic of poliomyelitis is a heavy one, and should rest on the Medical Officer of Health of the affected area. This was pointed out by the Ministry of Health in England during the 1950 epidemic. Certainly the Medical Officer of Health should be in a position to assess the relative risk of diphtheria in the non-immunised and arm paralysis in the recently immunised. There is no shadow of doubt that the former is by far the greater risk in Belfast.

A recent M.R.C. report describes outbreaks of diphtheria, mainly of the gravis type, in Dundee and on Tyneside, in which immunisation failed to prevent epidemic spread. It is emphasised that deaths did not occur in immunised children. A similar report comes from Amsterdam (Ruys and Noordam). The M.R.C. report states neither the number or percentage of children immunised nor the relative incidence of diphtheria in immunised and non-immunised children, but the point is made that large numbers of immunised children contracted diphtheria. Most people will not be surprised at this result, when they note that in many of the cases in which the dosage of antigen is stated, the doses given were 0.1 c.c. and 0.3 c.c., of A.P.T. This minute dosage was accepted as entitling children to be described as "fully inoculated."

These adverse reports describe events occurring before 1942. This date is important as in that year a standard was set, under the Therapeutic Substances

Act, for diphtheria antigens. This required them to contain at least fifty Lf. units per c.c.; a high level of potency which compelled more than half of the makers in England to withdraw their products, temporarily, from the market (Holt, 1950). It was about this time, too, that Bousfield pointed out the necessity of an adequate first dose, at least 0.5 c.c. A.P.T., to give high and prolonged immunity, and also the enhancing effect on immunity of lengthening the time interval between doses of A.P.T. to at least four weeks. Since then diphtheria in England has been decreasing with increasing acceleration.

In Belfast, immunisation started in 1936. An extensive epidemic of diphtheria occurred in 1940 when only 15 per cent. of the child population had been immunisation. Dr. H. A. Warnock, then in charge of the immunisation scheme, was checking the effect of immunisation by Schick testing and by the incidence and severity of diphtheria in immunised children. By the end of 1940 he was convinced that the optimum first dose of A.P.T. was 0.5 c.c. From 1942, with increasing numbers of children immunised (now over 60 per cent.), the incidence of diphtheria in Belfast has continuously declined. In the period 1942 to 1950 there has been sixty-one deaths from diphtheria, all in non-immunised children. In fact, since 1945 there has been an increasing case mortality rate in non-immunised children. Many of the fatal cases were infected with the gravis strain of diphtheria bacillus. The presence of this strain in Belfast has not resulted in any increase in the incidence of diphtheria in the immunised.

While the advent of the gravis strain is usually an importation Professor J. W. McLeod of Leeds has postulated that it may arise by mutation from other strains of the diphtheria bacillus.

The striving after even better immunity has resulted in the production of a new antigen by Holt (Holt and Bousfield, 1949) called Purified Toxoid, Aluminium Phosphate precipitated (P.T.A.P.). This is a highly purified product in that practically all bacterial protein has been removed with the result that it very seldom causes local reactions. It has given even better Schick conversion rates than A.P.T., but it has been suggested that while it produces high antitoxic immunity it may not stimulate antibacterial immunity. This appears to be rather a fine point as the stimulation of bacterial antibodies can hardly be claimed for any diphtheria antigen all of which are essentially toxoids. P.T.A.P. has been used in the Belfast clinics for three years now, and children receiving it have not suffered any undue incidence of diphtheria when compared with their fellows who received A.P.T. over the same period.

Tetanus toxoid is in many respects a comparable antigen to diphtheria toxoid. During the last war it not only practically eliminated deaths from tetanus but also the symptoms of tetanus in the actively immunised wounded. Here, apparently, the need for antibacterial immunity does not arise. The improved diphtheria toxoids should reach the same high efficiency by means of proper dosage and spacing of doses.

The present system of immunisation is to give the first two doses at 9 and 10

months of age followed by a booster dose at 5 years. This leaves a possibility of some waning of immunity at 3 or 4 years of age. This might be overcome by delaying the second dose until six or nine months after the first. This long interval would, however, result in many second doses being overlooked.

### SUMMARY.

An association between inoculations and paralysis in poliomyelitis was not found in the 1950 epidemic in Belfast. The unusually high incidence of arm paralysis found elsewhere in inoculated children occurred in Belfast in non-inoculated children.

The use of a highly potent diphtheria antigen, such as P.T.A.P., properly administered, provides substantial protection against diphtheria even in the presence of the gravis type.

I am greatly indebted to Dr. F. F. Kane, Northern Ireland Fever Hospital for his very complete clinical information; to Dr. S. Barron, M.O.H. Belfast, for helpful criticism and permission to publish; to Dr. H. A. Warnock, for his excellent records of the earlier years of immunisation; and to Dr. D. B. Keith, who continues the immunisation work in the Belfast clinics.

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# Congenital Atresia of the Œsophagus

By A. A. H. GAILEY, M.D., D.C.H., M.R.C.P.E., and W. A. B. CAMPBELL, M.D., D.C.H., M.R.C.P.E.

# The Royal Maternity Hospital and The Royal Belfast Hospital for Sick Children

Although congenital atresia of the œsophagus is comparatively rarely diagnosed, it is, as will be shown, a not uncommon congenital anomaly, and since early treatment now offers a chance of complete cure we have felt it worth while to make the following short summary of the literature on the subject, with notes on the clinical appearances of typical cases.

The condition has been reported in the literature for at least 250 years, Mackenzie (1884) quotes a case recorded by Durston in 1670. However, the earlier reports were largely of academic interest, and cases with successful operative repair were not recorded in any number until 1944. In that year, Haight (1944) reported 32 cases, 24 of which were explored surgically; in 16 the œsophagus was reconstructed, and 6 of these infants survived seven months or longer. Daniel (1944) reported 7 cases; all were explored—in 2 the segments were united, 1 child survived.

Lam (1945) recorded 25 successful results of either single or multiple stage operation, and added 3 more cases of his own.

Since then numerous series, large or small, have been reported with progressively better operative results. In all these, stress has been laid upon the need for early diagnosis, since delay in operation beyond the first twenty-four hours of life has been accompanied by a marked rise in mortality (Weiner and Richmond, 1950) (Ladd and Swenson, 1947), although successful results have been obtained even when operation has been delayed until the fourth day of life (White and Birdsong, 1946).

### CLINICAL DIAGNOSIS.

The condition may be suspected within a very short time after delivery, even before any feeds have been given, in an infant who shows cyanosis, with copious frothy saliva, which continues or increases.

Belsley and Donnison (1950) advise that such infants should be given nothing by mouth until this anomaly has been excluded by passing a catheter into the stomach. If feeds are offered to these infants inadvertently they will return the feed as soon as a few mouthfuls have been taken, often becoming cyanosed at the same time. Where this occurs, it is essential that no more should be given until the child has been carefully examined to confirm or exclude the presence of æsophageal atresia. Clinical signs of atelectasis, especially of the right upper lobe may be present, and this is not necessarily due to aspiration of feeds, since it may be present before any food had been given. Distension of the abdomen is frequently

marked since air can get into the stomach through the tracheo-œsophageal fistula which is almost always present.

The diagnosis can be confirmed by passing a rubber catheter into the esophagus when an obstruction will be found about 10-12 c.ms. from the lips. When this has been done, the infant should be removed as soon as possible to a centre where surgical treatment can be carried out, before radiological diagnosis is attempted.

### RADIOLOGICAL DIAGNOSIS AND CLASSIFICATION.

Although a clinical diagnosis of œsophageal atresia can be made with a fair degree of certainty, more exact evidence can be obtained radiologically, not only of the presence of the condition, but of the type and, to some extent, of the distance between the segments.

From the time when the earliest suspicion arises, the infant should be kept in a head-down position to facilitate drainage of saliva, and during the journey to hospital the child should be accompanied by a trained nurse who can aspirate the blind upper pouch every few minutes by catheter and syringe.

In the X-ray Department, a catheter is passed until the obstruction is reached; A-P and lateral films of the chest and an A-P film of the abdomen are taken. 0.5 ml. of lipiodol are then instilled and the chest films repeated; the lipiodol is then re-aspirated by the catheter. (Barium or bismuth must never be used because of the danger of their passing into the lungs).

The most generally used classification is that of Ladd (Ladd and Swenson, 1947) (Ladd, 1944);

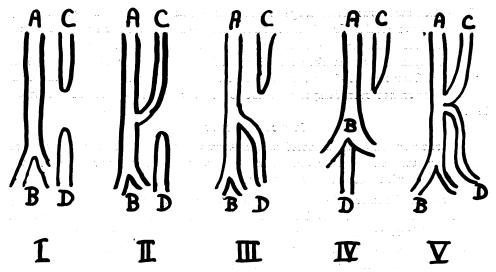


Diagram showing the arrangement of the trachea and esophagus in the various types of esophageal atresia and tracheo-esophageal fistula. The letters refer to the following structures: A—Trachea; B—Bifurcation of trachea; C—Upper segment of esophagus; D—Lower segment of esophagus.

(From Ladd, W. E., New Engl. J. Med. 230, 625-637, May 25, 1944).

Types I and II will show no air in the stomach or intestine, lipiodol will enter the lungs in Type II. Types III and IV will show air in the stomach and intestine, with no entry of lipiodol into the lungs. Type V shows air in the stomach and also lipiodol into the lungs.

Holt, Haight and Hodges (1946) describe a similar radiological technique and adopt the classification of Vogt (1929), which is essentially similar to that of Ladd, but includes the rare group where there is complete absence of the esophagus.

By far the most common group is that consisting of a blind upper pouch and a fistula between the lower pouch and trachea-Vogt group IIIb, Ladd types III and IV.

Associated Anomalies are common but rarely serious according to Ladd and Swenson (1947). They found associated anomalies in 91 out of 114 cases, but in the last 82 cases only 18 had anomalies sufficiently serious to modify the treatment of prognosis, e.g., atresia of the intestine, congenital heart disease, etc.

Operation should not be delayed when the diagnosis has been established. Pre-operatively, continuous oxygen should be given, with continuous or repeated pharyngeal suction. Penicillin injections may well be started even before the radiological investigations have been carried out, and in view of the increasing number of penicillin resistant organisms among the flora of hospitals, streptomycin might well be combined. Except in cases where treatment has been delayed, it will not usually be necessary to give parenteral fluid before operation, but a blood transfusion is started at the time of operation.

The actual operative technique is a matter of choice for the surgeon concerned. Ladd and Swenson generally adopted a retropleural approach, as did Lam (1945), Belsley and Donnison (1950), on the other hand, use a transpleural approach.

When the œsophagus has been exposed and the tracheal fistula ligatured, the choice lies between direct end-to-end anastomosis, which is preferable whenever possible and a multiple stage operation. In the latter case, the upper pouch is brought to the exterior in the neck, removing the danger of aspiration, and a gastrostomy performed, an artificial anterior œsophagus being constructed at a later date. Operative details are given in the articles quoted above.

The post-operative course in cases of primary anastomosis differs; Ladd and Swenson perform a gastrostomy as a routine twenty-four hours after operation, through which the infant is fed for ten days, after which ordinary feeds are gradually started.

Belsley and Donnison do not perform gastrostomy; they follow up the initial blood transfusion of 200 c.cs. with infusion of saline and start oral feeds of sterile water twenty-four hours after operation. They publish a series of ten cases, of which 5 died; three of the deaths were due to leakage at the anastomosis, this they attribute to failure to trim back the cyanotic tissue at the upper end of the lower segment, which is liable to slough owing to interference with the blood supply on ligature of the tracheo-æsophageal fistula; in their last 7 cases, after this danger had been appreciated, only 2 deaths occurred.

Ladd and Swenson, in a larger series, quote 16 survivors out of 43 primary

operations, and 14 out of 32 multiple stage operations; only 1 out of the last 14 primary repairs had died.

Incidence: Grey Turner (1949) estimates the incidence at 1 in 2,500 births; Belsley and Donnison, however, in a two-year experience found 5 cases in 4,000 odd births, and suggest that the true incidence is even higher than the figure of 1 in 800 which this would suggest, since infants may die of broncho-pneumonia or atelectasis without suspicion of atresia if autopsy is not carried out.

A search of the records of the Royal Belfast Hospital for Sick Children and of the Royal Maternity Hospital during the years 1940-1949 brought to light 11 proven cases in all of which a post-mortem examination was done. Nine of these were discovered in the Nurseries of the Royal Maternity Hospital from 15,943 infants live born in that hospital during that period. Since post-mortem examinations were not performed in all premature or weakly infants in the earlier years of the period under review, it is not suggested that this figure in any way represents the incidence of the condition in this hospital. The two cases seen at the Royal Belfast Hospital for Sick Children were admitted from Banbridge Hospital.

Consideration was given especially to the condition on delivery of these infants and to the earliest symptom or sign noted in each case, and the findings are briefly summarised.

| Condition on delivery -         | - | Mucus++                     |       | 4 cases |
|---------------------------------|---|-----------------------------|-------|---------|
|                                 | - | Cyanosis                    | • • • | 3 cases |
|                                 | - | Normal                      |       | 2 cases |
|                                 | - | No note                     | • • • | 3 cases |
| Symptoms in order of appearance | - | Mucus++                     |       | 4 cases |
|                                 | - | Cyanosis                    |       | 6 cases |
|                                 | - | Regurgitation of first feed |       | 6 cases |
|                                 | - | Respiratory difficulty      |       | 3 cases |

The case histories of two infants are described, since they indicate the typical course of the condition.

Case 5—1946. Male infant, 7 lbs. 4 ozs. (Kg. 3.4) at birth. Maturity thirty-six weeks. Immediately after birth an excessive outpouring of mucus from the mouth was seen. Respirations were shallow and poor air entry into all lung areas was noted. Cyanotic attacks were frequent. Excessive amounts of mucus continued to pour from his mouth and feeds were regurgitated down his nose. The abdomen became distended and tympanitic.

Operation was not considered, and death occurred on the fifth day. Necropsy showed that there was esophageal atresia, and though the type was not stated, the description suggested that this case should be considered Type V (Ladd).

Case 10—1949. Female born 21st February, 1949, weight not noted. Admitted to Banbridge Hospital on 27th February, 1949.

There was a history of cyanotic attacks with regurgitation of all feeds offered.

A diagnosis of œsophageal atresia was made on X-ray screening after the passage of an œsophageal tube and injection of lipiodol. A film taken at this time showed no evidence of lung consolidation. The infant was transferred to The Royal Belfast Hospital for Sick Children on the same evening for surgery.

On admission to Royal Belfast Hospital for Sick Children, the infant's condition was fair and her colour was good. On examination, coarse crepitations were heard at both lung bases, and an X-ray taken at this time showed consolidation of the left lower and right middle lung lobes. There was no sign of lipiodol injected at Banbridge remaining.

Before operation, an intravenous drip infusion of 5 per cent. glucose in one-fifth normal saline was given, the trachea was aspirated and oxygen administered. A very light general anæsthesia was started, and during the operation plasma and blood were given intravenously by the drip apparatus. The operation lasted for three hours and, though on several occasions breathing stopped, was concluded successfully.

The immediate post-operative condition was fairly good, but respiratory difficulty continued in spite of oxygen and penicillin therapy. Nothing was given by mouth and the intravenous drip was continued till death occurred approximately twenty-four hours after the commencement of the operation.

Necropsy showed that the atresia had been Type III (Ladd); that the anastomatic line in the œsophagus at the site of operation was satisfactory and no leak had occurred; that no other congenital abnormality existed, but that there was extensive pneumonia in both lungs.

From even such a small number of cases the excessive outpouring of mucus from the mouth, with or without cyanotic attacks, is confirmed as an important early sign and one which presents itself before the need to offer a feed occurs. When such a sign appears, the suspicion that an atresia of the œsophagus is present should be proved or disproved by radiography before feeding by mouth is commenced.

It is further emphasised that when, in a suspected or proven case, it is necessary for the infant to be transferred some distance to the central hospital for surgery, aspiration of mucus from the upper œsophagus should be done at intervals to prevent the dangerous complication of aspiration pneumonia. The description of Case 10 suggests that the areas of consolidation in the lungs noted after transfer from Banbridge Hospital might have been due to material aspirated during the journey by ambulance.

The two cases admitted to the Royal Belfast Hospital for Sick Children were operated on by Professor H. W. Rodgers. In each case, though the anastomosis was later shown to be functionally sound, death occurred due to aspiration pneumonia following a delay in diagnosis of five days.

### SUMMARY AND CONCLUSIONS.

A review of the condition of congenital œsophageal atresia is given and illustrative cases are quoted.

It is felt that the incidence of the condition is greater than suspected and that, since even nine cases out of 15,900 live births is believed to be an under estimate, this would suggest that at least fifteen cases should be expected yearly in Northern Ireland out of a birth rate of 30,000. The condition ranks as a surgical emergency and, since the chances of saving life are good when operation can be performed in the first twenty-four hours after birth the importance of early diagnosis is stressed.

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# ROYAL MEDICAL BENEVOLENT SOCIETY OF IRELAND

DR. Harrington has sent us the following report for inclusion in the Journal. Fellows and Members of the Society will agree that the medical community as a whole are indebted to Dr. Harrington for the outstanding success of his campaign to increase the membership of the Society. The cases that come before the Fund's committee for consideration are not numerous, but each is pitiful, and, were the circumstances widely known, would arouse instant sympathy. In recent years the local funds have risen gradually above the point where we got more out of the Irish fund as a whole than we put into it, but the present position, for which once more, we must congratulate Dr. Harrington, is at last becoming more in keeping with the size and means of this city and medical school. With the ever decreasing value of the pound it is important that this should be maintained, and we would take this opportunity of bringing the Royal Medical Benevolent Fund Society of Ireland to the attention of those who are still not subscribers.

The Annual Meeting of the Belfast Branch of the Royal Medical Benevolent Fund Society of Ireland was held in the Whitla Medical Institute on the 6th April, 1951, with Dr. Robert Marshall in the chair, and a full attendance of members.

The Honorary Secretaries were happy to report a big increase in membership—the total being doubled within two years.

In 1949, there were 182 members; in 1950, 213; and in 1951, the total, including four new Life members, reached 369.

The gain has been consolidated by the unanimous support of old subscribers. On the financial side the funds for the year have passed the £500 mark.

# **REVIEWS**

HANDBOOK OF CHILD HEALTH. By Austin Furniss, L.R.C.P., L.R.C.S. (Edin.), L.D.S., D.P.H. Pp. 280. Sylviro Publications Ltd. 25s.

This little book is described in the author's preface as a review of the main features of child health, specially intended for students preparing for the diploma of Child Health. It is made up principally of descriptions of the services of the several public health departments concerned with infants and children, and may prove useful in this respect. Unfortunately, the matter is not clearly set out, and the author's style of writing makes it difficult to understand exactly what is meant in some places.

He is, in general, better when dealing with the administrative aspects of the servcies than when describing clinical conditions. Clinicians will find it difficult to agree with the bald statement that children (at nursery schools) who have enlarged tonsils and adenoids "invariably do well after operation." It is difficult to understand the principle of selection of clinical descriptions of diseases, e.g., the chapter on "more common bone and joint and neurological conditions" devotes three pages to achondoplasia, but makes no mention of talipes, and includes such rare conditions as Schilder's disease,

It is, perhaps, a sign of the times that, although there are recommendations for referring children to Orthopædic Clinics, Speech Clinics, Child Guidance Clinics, and to Pædiatricians, there is no mention of the general practitioner or of co-operation with the family doctor.

The book would be much improved if Dr. Furniss confined himself to describing the organisation of the health services and the work done at the various clinics, and omitted descriptions of diseases which are better described in standard text-books. Bibliographies at the end of the chapters to enable the numerous references to be traced, and facsimilies of the forms quoted would also be helpful. The price (25s.) seems excessive for 270-odd pages without illustrations.

W. A. B. C.

MEDICAL RESEARCH COUNCIL—SPECIAL REPORT SERIES—No. 276: Occupational Factors in the Ætiology of Gastrie and Duodenal Ulcers, with an Estimate of their Incidence in the General Population. By Richard Doll and F. Avery Jones, with the assistance of M. J. Buckatzsch. H.M. Stationery Office, 1951. 2s. 6d.

This is an account of a survey to determine the incidence of peptic ulcer and to investigate the rôle of occupation in etiology. In London the incidence is calculated as being 5.8 per cent. for men aged between 15-64, and 1.9 per cent. for women in the same age group. The authors estimate that the total number of persons now living in England and Wales with peptic ulcer, past or present, is nearly one and a half million. Over half a million men annually suffer from symptoms. Occupations having a high incidence for duodenal ulcer are doctors, foremen, business executives, and workers holding responsible positions in industry. In contrast, significantly low rates of incidence were found among agricultural workers and in the large group of sedentary workers, composing clerks, administrative and executive civil servants, research workers. Though adequate provision was made to include a representative number of drivers of motor tehicles and bus conductors, there was no direct evidence that a higher rate of incidence occurred in these groups than in other groups. Investigation of the possible bad effects of shift work or of irregularity in the taking of food also failed to show any association between them and the existence of an ulcer.

The report concludes, therefore, that actual conditions of work play no important rôle in the genesis of gastric-duodenal dyspepsia. As one would expect, the authors noted a strong correlation between anxiety and duodenal ulcer, the patients being commonly of the hard-working, ambitious over-conscientious type. This aspect of the problem, however, was not investigated specially.

R. S. A.

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R. S. A.

### REVIEW

INCONTINENCE IN OLD PEOPLE. By John C. Brocklehurst, M.D. Pp. 187. E. & S. Livingstone Ltd.

In this monograph Dr. Brocklehurst outlines an active approach to a problem which too often is passively accepted as a necessary evil. The system of "laisser-faire," commonly adopted towards incontinence, entails endless use of skilled nursing care on frequent bed changes, and needless expense on laundering and replacement of bed linen. Dr. Brocklehurst has investigated this problem in the adult population in general wards and mental observation wards of five Glasgow hospitals, and made a study of cystometric and rectal pressure findings under certain conditions in a group of incontinent patients.

The book is written in three parts, describing first, a review of the literature on this subject with special reference to the anatomy and physiology of bladder and rectum, and the theories of disordered mechanism leading to incontinence. In the second part the actiology of incontinence is discussed in the light to Dr. Brocklehurst's experience. His investigations suggest that the function of the sphincters is of less importance in the onset of incontinence than the tonus and movements of bladder and large bowel. The main predisposing factor is a lesion in the central nervous system, and obstetrical trauma or prostatic enlargement are of negligible importance as actriological factors. Precipitating factors are a cerebro-vascular accident, the patients' becoming bedfast, and mental confusion. He confirms the belief that the status of the nurses and nursing methods employed clearly affect the incidence of incontinence in hospital wards.

The third part of the book is a discussion on treatment, and the bulk of this section is devoted to the design and construction of a special bed for bedfast patients with intractable incontinence. Like earlier workers in geriatrics, Dr. Brocklehurst has found that when an incontinent patient is back on his feet incontinence lessens or even clears up completely. If this fails, there is no simple remedy and one must still resort to the portable urinal or special bed. The possibility of commercial production of Dr. Brocklehurst's bed is being considered by the Dunlop Rubber Company. It is a pity, however, that the value of Wilson's cystometric treatment could not have been assessed in this series, for it is undoubtedly effective in certain cases although a time-consuming procedure.

This book should focus attention on the possibility of alleviating one of the most distressing afflictions of old age, and to the need for further research into other neglected disabilities associated with degenerative disease. The book is attractively produced and illustrated, but although the subject is one of general interest the book is expensive, and most likely to be borrowed for reference, except by those with a particular interest in the management of these patients.

G. F. A.